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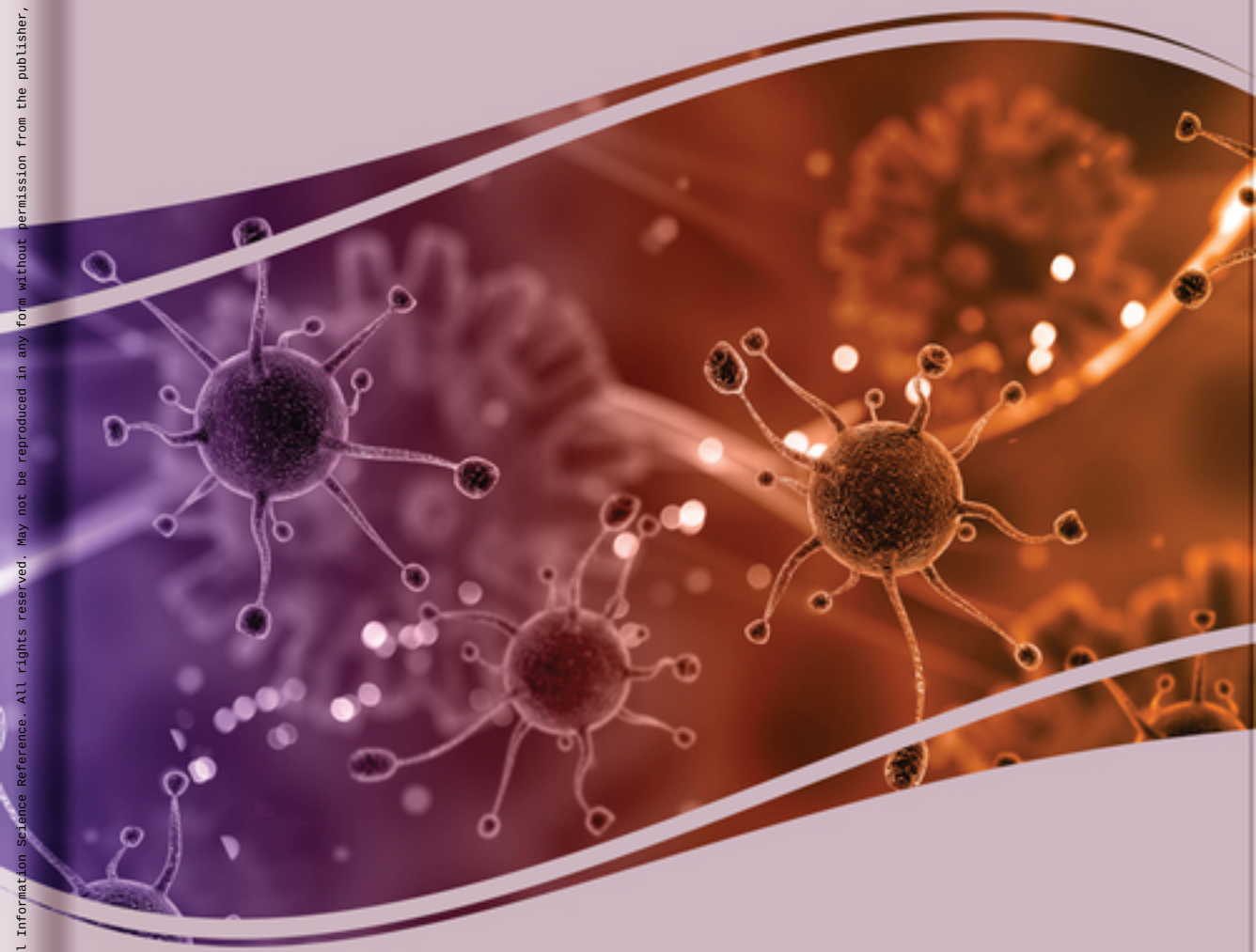
Mathematical Models of Infectious Diseases and Social Issues



Nita H. Shah and Mandeep Mittal

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Mathematical Models of Infectious Diseases and Social Issues

Nita H. Shah
Department of Mathematics, Gujarat University, Ahmedabad, India

Mandeep Mittal
Department of Mathematics, Amity Institute of Applied Sciences, Amity University, Noida, India

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Detailed Table of Contents

We all are living in such a world where the pollution and global warming are threats. Every year in India, at the time of festival seasons of Dussehra and Deewali, the smog and pollution are so much that millions of people suffer from different health issues. Also, the farmers of Punjab and Hariyana burn the Parali of their crops due to less awareness, and it becomes a challenge in the national capital, Delhi, to breathe. The government invests resources and the vehicles are allowed as per their even odd numbers. The authors team, including government officials, educationists, academicians, and students, along with IT experts, performed significant experiments on the ancient Indian Vedic science of Yajna and Mantra, and they found surprising results in the reduction of pollution on respective days. The chapter is an effort to present that scientific study conducted in 2018 and 2019 in random days after doing Yajna, and it was found that the pollution level was drastically decreased.

Smoking tobacco has some hazardous implications on an individual's physical, physiological, and psychological health; health of the passive smokers near him or her; and on the surrounding environment. From carcinomas to auto-immune disorders, smoking has a role to play. Therefore, there arises a need to frame a systemic pathway to decipher relationship between smoking and a perilous disease such as tuberculosis. This research work focuses on how drugs or medications can affect individuals who are susceptible to tuberculosis because of smoking habits and also on individuals who have already developed symptoms of tuberculosis due to their smoking addiction. The mathematical model is formulated using non-

linear ordinary differential equations, and then threshold is calculated for different equilibrium points using next generation matrix method. Stability analysis along with numerical simulations are carried out to validate the data.

In this chapter, the authors considered a smoking cessation model formulated with a non-linear system of differential equations and obtained the continuous fractional order model and through discretization its discrete form to study the effectiveness of quitting smoking applications in giving up smoking. The existence of smoking free equilibria and smoking present equilibria are discussed, and the dynamical analysis of these two equilibria is put forward with the assistance of the smoking generation number. The numerical simulations aided by time series, phase portraits, and bifurcation diagrams confirm the results that are obtained analytically.

This chapter considers the dynamical behavior of a new form of fractional order three-dimensional continuous time prey-predator system and its discretized counterpart. Existence and uniqueness of solutions is obtained. The dynamic nature of the model is discussed through local stability analysis of the steady states. Qualitative behavior of the model reveals rich and complex dynamics as exhibited by the discrete-time fractional order model. Moreover, the bifurcation theory is applied to investigate the presence of Neimark-Sacker and period-doubling bifurcations at the coexistence steady state taking h as a bifurcation parameter for the discrete fractional order system. Also, the trajectories, phase diagrams, limit cycles, bifurcation diagrams, and chaotic attractors are obtained for biologically meaningful sets of parameter values for the discretized system. Finally, the analytical results are strengthened with appropriate numerical examples and they demonstrate the chaotic behavior over a range of parameters. Chaos control is achieved by the hybrid control method.

Demonetization is a fundamental regulatory act of stripping in which a currency unit's status as an exchange is professed worthless. Generally, it is done whenever there is a change of national currency, often to be replaced of the old notes or coins with a new one. Sometimes, a country totally replaces the old currency with new currency. For example, in India recently the government demonetized RS. 500 and 1000 notes. So, one has to deposit their cash within limited time in the banks. The demonetization affects individuals mildly or potentially, which in turn affects banking sector. So, SMPB-model is proposed and analyzed for demonetization. The SMP-model is formulated with the system of nonlinear differential equations. The effect of demonetization is studied by calculating threshold using next generation matrix. The local and global stability for demonetization free equilibrium and demonetization equilibrium is worked out. The existence of the equilibrium is investigated. The model is validated with numerical simulation.

Preface

DYNAMICS OF INFECTIOUS DISEASES AND SOCIAL ISSUES

Understanding the transmission dynamics of disease spread will not only help us to control the disease, but will also make us prepared for the future as it will make the scenario and intensity of spread clear.

Biological mechanism and behaviour of a parasite or virus involved in disease spread for their intensity as well as their ability to survive in host and make an individual infected is too complex to understand. The mathematical modelling is a tool to measure or understand such mechanism and the relevance of any public health care strategy. With certain constitutive assumptions, notations and variable definitions, a mathematical model transforms a biological problem in to mathematical equations which describes the actual situation. Using mathematics as language to interpret the assumptions concerning the biological and population mechanics, one can make predictions by comparing the actual epidemiological data and verify validity of same using mathematical tests and results. Age plays an important role in the dynamics of disease transmission. In particular, mathematical models can be used to visualize the transmission dynamics and spread of these diseases. These models can help us to understand the right disease status and predict if the disease is going to be genetic or controlled. Such applications are highly helpful to the society if utilized wisely. Models under consideration are deterministic models, also known as compartmental models. Stochastic models depend on chance and provide deeper insight as it is individual-level modelling but they deal with small population and very complex in nature. Deterministic models that are described in this study can deal with the case of large population by dividing and subdividing the total population into

various compartments. In this Edited Book, our focus is to understand the disease dynamics in population. The disease can be interpreted as social issue transmission, environmental issue and many more.

CONTROLLING ASTHMA DUE TO AIR POLLUTION

Health of our respiratory system is directly affected by the atmosphere. Nowadays, eruption of respiratory disease and malfunctioning of lung due to presence of harmful particles in the air is one of the most sever challenge. In Chapter 1, association between air pollution related respiratory diseases; namely, dyspnea, cough and asthma is analysed by constructing a mathematical model. Local and global stability of the equilibrium points is proved. Optimal control theory is applied in the model to optimize stability of the model. Applied optimal control theory contain four control variables, among which first control helps to reduce number of individuals who are exposed to air pollutants and remaining three controls helps to reduce spread and exacerbation of asthma. Positive impact of controls on the model and intensity of asthma under the influence of dyspnea and cough is observed graphically by simulating the model.

ANALYTICAL STUDY OF LARGE-SCALE HOUSEHOLD YAGYA EFFECTS ON AMBIENT AIR POLLUTION

We all are living in such a world where the pollution and global warming are threats. Every Year in India, at the time of festival seasons of Dussehra and Deepali, the smog and pollution are so much terrific that million people suffer from different health issues. Also, the Farmers of Punjab and Hariyana burn the Parali of their crops due to less awareness and it becomes challenge in National Capital Delhi to breath. The government invest high resources and the vehicle are allowed as per their even odd numbers. The authors team including government. officials, Educationists, Academicians and students along with IT experts performed significant experiments on Ancient Indian Vedic Science of Yajna and Mantra and they found surprising results in the reduction of pollution on respective days. The Chapter 2 is an effort to present that scientific study conducted in year 2018 and 2019 in random days after doing Yajna and it was found that pollution level was drastically decreased.

SPREAD OF TUBERCULOSIS AMONG SMOKERS: A MATHEMATICAL MODEL

Smoking tobacco has some hazardous implications, on an individual's physical, physiological and psychological health, health of the passive smokers near him or her and on the surrounding environment. From carcinomas to auto-immune disorders, smoking has a role to play. Therefore, there arises a need to frame a

systemic pathway to decipher relationship between smoking and a perilous disease such as tuberculosis. Chapter 3 focuses on how drugs or medications can affect individuals who are susceptible to tuberculosis because of smoking habits and also on individuals who have already developed symptoms of tuberculosis due to their smoking addiction. The mathematical model is formulated using non-linear ordinary differential equations and then threshold is calculated for different equilibrium points using next generation matrix method. Stability analysis along with numerical simulations are carried out to validate the data.

DYNAMICAL ANALYSIS OF THE EFFECT OF QUIT SMOKING APPLICATIONS ON SMOKING CESSATION

In Chapter 4, we consider a smoking cessation model formulated with a non-linear system of differential equations and obtained the continuous fractional order model and through discretization its discrete form and intend to study the effectiveness of quit smoking applications in giving up smoking. The existence of smoking free equilibrium and smoking present equilibrium are discussed and the dynamical analysis of the two equilibriums is put forward with the assistance of the Smoking Generation Number. The Smoking Generation Number plays a crucial role in the determination of stability of equilibriums. Conditions are determined for the stability. Efficiency of quit smoking applications is investigated for effectiveness of quit smoking mobile apps and fractional order on the system using numerical simulation. The numerical simulations aided by time trajectory, phase line diagrams and bifurcation diagrams confirm the results that are obtained analytically. It is shown that effective campaign with apps is an effective to curb smoking habit.

BIFURCATION AND CHAOS IN A DISCRETE FRACTIONAL ORDER PREY-PREDATOR SYSTEM INVOLVING INFECTION IN PREY

Chapter 5 considers the dynamical behaviour of a new form of fractional order three-dimensional continuous time prey predator system and its discretized counterpart. Existence and uniqueness of solutions is addressed. Dynamical nature of the model is discussed through local stability analysis of the steady states. Qualitative behaviour of the model reveals rich and complex dynamics exhibited by the discrete-time fractional order model. Moreover, the bifurcation theory is applied to investigate the presence of Neimark – Sacker and Period-doubling bifurcations at the coexistence steady state taking as a bifurcation parameter for the discrete fractional order system. Also the

trajectories, phase diagrams, limit cycles, bifurcation diagrams and a chaotic attractor are obtained for biologically meaningful sets of parameter values in the discretized system. Finally, the analytical results are strengthened with appropriate numerical examples and they demonstrate chaotic behaviour over a range of parameters. Chaos control is achieved by the hybrid control method.

ANALYSIS OF DISCRETE SYSTEM MODELLING FOLLOWED BY SPREAD OF INFECTIOUS DISEASES MODELLING IN FUZZY ENVIRONMENT

In Chapter 6, authors discuss the solution of spread of infectious diseases in terms of SI model in fuzzy environment which is modelled in a typical discrete system. As the system is discrete in nature, the concept of difference equation has been embarked. In order to understand the underlying uncertainty perspective, we explored the fuzzy difference equations to study the problem. In this chapter we discuss the solution of spread of infectious diseases in terms of SI model in fuzzy environment which is modelled in a typical discrete system. As the system is discrete in nature, the concept of difference equation has been embarked. In order to understand the underlying uncertainty perspective, we explored the fuzzy difference equations to study the problem.

STABILITY ANALYSIS OF CO-INFECTION OF MALARIA-DENGUE

Dengue and Malaria most commonly occur in tropical and sub-tropical areas. Dengue is a viral infection in a human being caused by a bite of a female Aedes Mosquito whereas malaria is caused by plasmodium parasite transmitted by a bite of infected mosquito. In Chapter 7, a mathematical model of co-infection of malaria and dengue is described by deterministic system of non-linear ordinary differential equations. This system considers the force of infection which is applied to dengue susceptible individuals. Moreover, two sub-models namely Malaria-only and Dengue-only are also constructed to study the transmission dynamics. Basic reproduction number is calculated for these models to investigate the existence of the models. The system is proved to be locally and globally stable at its equilibrium points. Stability of these models is also shown through numerical simulation.

FRACTIONAL-ORDER MODEL TO VISUALIZE THE EFFECT OF PLASTIC POLLUTION ON RAIN

In this era, one of the biggest issues faced by humans is due to plastic pollution as it dwells in environment and depletes the ecosystem. This affects the climate and disturbs the chain of rain which is the common source of obtaining water body. Also, this resulting pollution causes the toxicity in rain. Accordingly, the mathematical model is framed in Chapter 8 by considering fractional order derivative. Pollution free and endemic equilibrium points are worked out for integer order system of non-linear differential equations. Local stability of equilibrium points brings attention on dynamical behaviour of model with sufficient condition. With the help of basic reproduction number, bifurcation is analysed which shows the chaotic nature of this model. Providing Caputo derivative of fractional order, numerical simulation has been done by taking different values of order for the system.

CONTROL OF PEST POPULATION BY STERILE INSECT TECHNIQUE CONSIDERING LOGISTIC GROWTH WITH SPATIAL SPREAD INVASION AND OPTIMAL PRODUCTION POLICIES

In Chapter 9, authors have proposed an SIT model to eradicate the pest population. It has been assumed that the females after mating with wild males grow logistically. Pest population is being controlled with the release of sterile insects in their habitat. The model is formulated with the system of differential equations and we have discussed the local stability analysis of deterministic logistic growth rate model. Further, we have also obtained a potential function by incorporating one-dimensional insect release with an invasion on patch size L which has a toxic exterior as its surrounding. It has been obtained that in the presence of spatial spread over a finite patch size, the sterile release of the insects produces a sudden declination of the pest population. Finally, authors have obtained the optimal production of sterile male population using Pontryagin's maximum principle. The applicability of our proposed model is finally illustrated through numerical solution.

TRANSMISSION OF WATER AND FOOD WASTE IN AQUAPONIC SYSTEM

In order to conserve natural resources, the quest for recycling water and food waste culture is ongoing nowadays. One of the possible and good ways to reuse these wastes

is hydroponic culture. It is an advanced technology that cultivates plants without soil. Instead of using root system, it needs nutrient-rich water. Most of the nutrients used in hydroponic culture come from aqua culture, the branch for propagation, emergence and maintenance of aquatic (water) organisms. Humans convolve aqua culture with hydroponic culture that has come up as an aquaponic system. It has been universally adopted for indoor food production. The solution arising out of this system has eliminated the lack of vegetable and fish. The continuous nature of these cultures gives rise to the system of non-linear ordinary differential equations. This system is investigated through logistic growth rate in Chapter 10. Logistic growth rate offers an oscillating threshold. The simulative results analyse the periodicity of the system solutions which will help to survive the ecosystem.

VERTICAL TRANSMISSION OF SYPHILIS WITH CONTROL TREATMENT

Syphilis is sexually transmitted disease having different signs and symptoms with four main multiple stages namely primary, secondary, latent, and tertiary. Congenital (vertical) transmission of syphilis from infected mother to foetus or neonatal is still a cause of high perinatal morbidity and mortality. A model of transmission of syphilis with three different ways of transmission namely vertical, heterosexual and homosexual is formulated in Chapter 11 as a system of nonlinear ordinary differential equations. Treatment is also incorporated at various stages of infection. Total male and female population is divided in various classes viz were susceptible, exposed, primary and secondary infected, early and late latent, tertiary, infected treated, latent treated, infected child (new born), and treated infected child (at birth time). Stability of disease free equilibrium and endemic equilibrium is established. Control treatment is applied. It is observed that safe sexual habits, controlled treatment in each stage including pregnancy are effective parameters to curb disease spread.

MATHEMATICAL MODEL TO ANALYZE EFFECT OF DEMONETIZATION

Demonetization is a fundamental regulatory act of stripping in which a currency unit's status as an exchange is professed worthless. Generally, it is done whenever there is a change of national currency, often to be replaced of the old notes or coins with a new one. Sometimes, a country totally replaces the old currency with new currency. For example, in India recently the government demonetized RS. 500 and 1000 notes. So, one has to deposited their cash within limited time in the banks.

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Nita H. Shah
Department of Mathematics, Gujarat University, Ahmedabad, India

Mandeep Mittal
Department of Mathematics, Amity Institute of Applied Sciences, Amity University, Noida, India

Chapter 1
Controlling Asthma Due to Air Pollution

Ankush H. Suthar
Department of Mathematics, Gujarat University, Ahmedabad, India

Purvi M. Pandya
Department of Mathematics, Gujarat University, Ahmedabad, India

ABSTRACT

The health of our respiratory systems is directly affected by the atmosphere. Nowadays, eruption of respiratory disease and malfunctioning of lung due to the presence of harmful particles in the air is one of the most sever challenge. In this chapter, association between air pollution-related respiratory diseases, namely dyspnea, cough, and asthma, is analysed by constructing a mathematical model. Local and global stability of the equilibrium points is proved. Optimal control theory is applied in the model to optimize stability of the model. Applied optimal control theory contains four control variables, among which first control helps to reduce number of individuals who are exposed to air pollutants and the remaining three controls help to reduce the spread and exacerbation of asthma. The positive impact of controls on the model and intensity of asthma under the influence of dyspnea and cough is observed graphically by simulating the model.

INTRODUCTION

Rapid growth in population and industrialization have resulted in increasing demand for energy which effects the levels of atmospheric particulate matter. Epidemiological studies have proved that exposure to air pollutants lead to respiratory symptoms

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and decrease in lung functionality (Gehring U. *et al.*, 2013; Usemann J. *et. al.*, 2019). Toxic and hazardous pollutants from the air penetrates deep into the lungs in inhalation, that can increase one's risk of cardiovascular and respiratory diseases including cardiac arrests, lung cancer, chronic obstructive pulmonary diseases and respiratory infections (Ciencewicki J., & Jaspers I., 2007, Ko F. W., & Hui D. S., 2012). Air pollution is more significant environmental health risk factor in Asia, as its mortality rate is higher (Abas N. *et. al.*, 2019, Wang Q. *et. al.*, 2019). As a result of increased pollutants in the environment, World Health Organization has estimated that globally more than seven million premature death are occurring mainly due to cardiac arrest, respiratory infections, stroke, lung cancer, chronic obstructive pulmonary disease, chronic bronchitis and asthma (WHO, 2019).

Respiratory defence mechanisms are altered by specific allergens present in polluted environment which trigger asthma exacerbation and enhance rate of hospitalization for asthma (Tatum A. J., & Shapiro G. G., 2005). A recent estimations states that, globally 339 million people are suffering from asthma (Global Asthma Network, 2018). Moreover, its prevalence continues to rise in children as pollutants have damaging effect on their lung function (Brokamp C. *et. al.*, 2019; Fielding S. *et. al.*, 2019; Soto-Martínez *et. al.*, 2019). There is a widespread concern that prevalence of asthma is still rising in developed countries, but the economic and humanitarian effects of asthma are on rise in developing countries, where the prevalence is also rising. Therefore the initial prevention strategies to tackle the asthma epidemic are sought after. Asthma is characterized by production of excess amount of gluey secretions inside the airways which fallouts in inflammation of the respiratory tubes (Groneberg D. A. *et. al.*, 2002). As a result of it, early threatening common sigh seen in asthma patients are tightness in chest, coughing, dyspnea and wheezing. Also, presence of dyspnea and cough enduringly can accelerate asthma exacerbation or generate more harmful diseases.

Dyspnea is normally correlated with the situations in which the neural central control system, the sensory input systems, the muscular effect systems or respiratory system are stop functioning as it should be. In medical term, these situations are characterized by shortness of breath or air hunger (Simon P. M. *et. al.*, 1990; Wasserman K., & Casaburi R., 1988). It is a serious health concern, as interval of shortness of air can differ from minor and momentary to serious and long lasting. Causes of dyspnea depends upon several factors including low blood pressure, asthma, cardiac arrests, stress, anxiety and exposure to air pollutants etc. Dyspnea can reduce life span for patient by inviting other chronic obstructive pulmonary diseases, like cardiac arrest, asthma, etc.

Coughing is a common reflex action that clears your throat of mucus or foreign irritants whenever it requires. Infections and noxious waste present in the atmosphere can cause more frequent coughing (Chung K. F. *et. al.*, 2013). Eosinophilic airway

inflammation is associated to large amount of chronic cough patients including cough variant asthma or non-asthmatic eosinophilic bronchitis (Matsumoto H. *et. al.*, 2007; Niimi A. *et. al.*, 2009). However, with pollution levels rising in developing countries, experts warn that a long-standing chronic cough could be a sign of more serious respiratory conditions. Various study showing that exposure to toxic particulate matter, irritant gases, mixed pollutants, etc. is associated with an increase in cough and asthma exacerbation (Chen L. C., & Thurston G., 2002; Delfino R. J. *et. al.*, 2002).

As epidemiology has turned out to be as a serious problem for the modern world, study of theories based on correlation between epidemiology, mathematical modeling and computational tools has been increasing. For the researchers, a mathematical model offers a significant research tool to study spread of infectious diseases (Cruz-Aponte M., 2014; Keeling M. J., & Rohani P., 2011). Additional to mathematical modeling, the optimal control theory is used to analyse the optimal strategies for the system using control variables which are associated with diminution of the disease during an outbreak (Rodrigues H. S., 2014). Many researchers have proposed various mathematical models and analyzed the stability of the models to study the spread of asthma exacerbation due to tobacco smoke and pollutants exhausted from industries (Ghosh M., 2000; Ram N., & Tripathi, A., 2009).

Aim of this study was to analyze transmission of dyspnea, cough and asthma as a result of polluted environment using mathematical modeling. In addition to this, the model is designed by avoiding the case where asthma causes dyspnea and cough, this construction helps to understand the role of dyspnea and cough in asthma exacerbation. Construction of the mathematical model is explained in section 2. In third section, equilibrium points of the dynamical system are founded. In section 5, local and global stability is proved using Routh-Hurwitz criteria and constructing Lyapunov functions respectively. In section, optimal control theory is applied in the model that helps to optimize stability of the dynamical system. In next section, graphical presentation of flow of model and effect of controls on model is analysed by numerical simulation.

FORMULATION OF A MATHEMATICAL MODEL

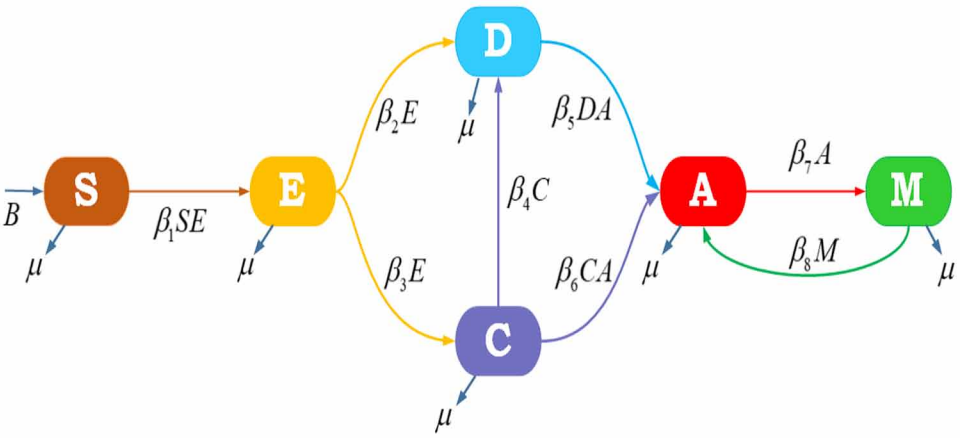
As we know air pollution is a major factor for respiratory diseases (dyspnea, cough and asthma). In this section, a mathematical model is constructed to describe effect of dyspnea and cough on asthma exacerbation. Moreover, effect of cough on intensity of dyspnea can be described by analysing the model. Basic idea and structure of the mathematical model is based on following hypothesis:

- The total human population size (N) is divided into six subpopulation compartments: class of susceptible people (S), humans exposed (but still not infected) (E), population suffer from dyspnea (D), population suffer from cough (C), class of population suffer from asthma (A) and class of population who opt medication (M).
- The parameter B is the birth rate of humans. μ is the natural death rate which is assumed equal for all the compartment.
- All the Parameters which defines rates of transformation from one compartment to other compartment of the model are described in the following table 1. Since it is not possible to cure asthma completely all the time, in present model the case have been taken where individual who get medication comes again in the class of asthma individuals.

Table 1. The parameters for the mathematical model

Parameters	Description
β_1	Rate at which susceptible population moves to exposed class
β_2	Rate at which exposed individuals consuming dyspnea
β_3	Rate at which exposed individuals infected by cough
β_4	Rate at which individuals infected by cough having dyspnea
β_5	Rate by asthma exacerbation is accelerated by individuals having dyspnea
β_6	Rate by which individual infected by cough trigger asthma exacerbation
β_7	Rate at which asthma patients go for medication
β_8	Rate at of medicated individual who suffers from asthma

Figure 1. Flow diagram of model



These groundings delivers the dynamical system of non-linear differential equations as follows:

$$\begin{aligned}
\frac{dS}{dt} &= B - \beta_1 SE - \mu S \\
\frac{dE}{dt} &= \beta_1 SE - \beta_2 E - \beta_3 E - \mu E \\
\frac{dD}{dt} &= \beta_2 E + \beta_4 C - \beta_5 DA - \mu D \\
\frac{dC}{dt} &= \beta_3 E - \beta_4 C - \beta_6 CA - \mu C \\
\frac{dA}{dt} &= \beta_5 DA + \beta_6 CA + \beta_8 M - \beta_7 A - \mu A \\
\frac{dM}{dt} &= \beta_7 A - \beta_8 M - \mu M
\end{aligned} \tag{1}$$

FEASIBLE REGION AND EQUILIBRIUM POINTS

Invariant region for the model is by Λ and all the solutions of the model are positive for $t \geq 0$. The total population size N can be defined by $N=S+E+D+C+A+M$.

Also, $\frac{dN}{dt} = B - \mu N$ and hence the feasible region is given by:

$$\Lambda = \left\{ (S,E,D,C,A,M) / S+E+D+C+A+M \leq \frac{B}{\mu}; S \geq 0, E \geq 0, D \geq 0, C \geq 0, A \geq 0, M \geq 0 \right\} \tag{2}$$

The present model have three equilibrium point:

I. Disease free equilibrium point (E_0): $E_0(S_0,0,0,0,0,0)$, where, $S_0 = \frac{B}{\mu}$.

II. II. Asthma free equilibrium point $\left(E_{P_1}\right)$: $E_{P_1}(S_1,E_1,D_1,C_1,0,0)$, where,

$$S_1 = \frac{\beta_2 + \beta_3 + \mu}{\beta_1}, E_1 = \frac{B\beta_1 - (\beta_2 + \beta_3 + \mu)}{(\beta_2 + \beta_3 + \mu)\beta_1},$$

$$D_1 = \frac{B\beta_1(\beta_2(\beta_4+\mu)+\beta_3\beta_4)-\beta_2\mu(\beta_4+\mu)(\beta_2+\beta_3+\mu)-\beta_3\beta_4\mu((\beta_3+\mu)-\beta_2)}{\beta_1\mu(\beta_4+\mu)(\beta_2+\beta_3+\mu)},$$

$$C_1 = \frac{\beta_3(B\beta_1-\mu(\beta_2+\beta_3+\mu))}{\beta_1(\beta_4+\mu)(\beta_2+\beta_3+\mu)}.$$

III. III. Endemic equilibrium point $\left(E_p^*\right): E_p^*(S^*,E^*,D^*,C^*,A^*,M^*)$. Where,

$$S^* = \frac{\beta_2+\beta_3+\mu}{\beta_1}, E^* = \frac{B\beta_1-\mu(\beta_2+\beta_3+\mu)}{(\beta_2+\beta_3+\mu)\beta_1}, D^* = -\frac{\beta_6 r(\beta_8+\mu)-\mu(\beta_7+\beta_8+\mu)}{\beta_5(\beta_8+\mu)},$$

$$C^*=r,\ A^* = \frac{-\beta_1 r(\beta_4+\mu)(\beta_2+\beta_3+\mu)+\beta_3(B\beta_1-\beta_2\mu)-\beta_3\mu(\beta_3+\mu)}{(\beta_2+\beta_3+\mu)\beta_6\beta_1 r},$$

$$M^* = \frac{\beta_7\left(-\beta_1 r(\beta_4+\mu)(\beta_2+\beta_3+\mu)+\beta_3(B\beta_1-\beta_2\mu)-\beta_3\mu(\beta_3+\mu)\right)}{(\beta_8+\mu)(\beta_2+\beta_3+\mu)\beta_6\beta_1 r}$$

Here, $r = root\ of\ (a_0x^2+a_1x+a_2=0)$ where,

$$a_0 = (\beta_8+\mu)\left(\beta_1\beta_5\beta_6\mu(\beta_2+\beta_3)-\beta_1\beta_6^2\mu(\beta_3+\mu)+\beta_1\mu(\beta_5\beta_8\mu-\beta_2\beta_6^2)\right)$$

$$a_1 = \left((\beta_8+\mu)\beta_2\beta_4\beta_6(\beta_2+2\beta_3\mu+\mu^2)+\beta_3\beta_5\beta_6(\beta_3+\mu^2)-B\beta_1\beta_5\beta_6(\beta_2+\beta_3)\right.$$
$$\left.-\beta_1\beta_2\beta_5\mu(\beta_4+\mu)\right)+\beta_1\mu(\beta_7+\beta_8+\mu)\left(\beta_6\mu(\beta_2+\beta_3+\mu)-\beta_5(\beta_3+\mu)(\beta_4+\mu)\right.$$
$$\left.-\beta_2\beta_5\beta_7(\beta_4+\mu)\right)$$

$$a_2 = \beta_3\beta_5\mu(\beta_7+\beta_8+\mu)\left(B\beta_1-\mu(\beta_2+\beta_3+\mu)\right)$$

STABILITY

In this section, local and global stability of all three equilibrium points are proved using standard mathematical theories.

Local Stability

Theorem 4.1.1: The disease free equilibrium point (E_0) is locally asymptotically stable if three conditions, $\frac{B\beta_1}{\mu} < \beta_2 - \beta_3 - \mu$, $\beta_5 D < \beta_7 + \beta_8 + 2\mu$ and $\beta_5 D(\beta_8+\mu) < \mu(\beta_7+\beta_8+\mu)$ are satisfied.

Proof: The Jacobian matrix $J(E_0)$ for system (1) associated with point E_0 is given by:

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{\beta_1 B}{\mu} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_1 B}{\mu} - \beta_2 - \beta_3 - \mu & 0 & 0 & 0 & 0 \\ -\mu & \beta_2 & -\mu & \beta_4 & -\beta_5 D & 0 \\ 0 & \beta_3 & 0 & -\beta_4 - \mu & 0 & 0 \\ 0 & 0 & 0 & 0 & D\beta_5 - \beta_7 - \mu & \beta_8 \\ 0 & 0 & 0 & 0 & \beta_7 & -\beta_8 - \mu \end{bmatrix}$$

The eigenvalues of matrix $J(E_0)$ are:

$$\lambda_1^0 = -\mu, \lambda_2^0 = -\mu, \lambda_3^0 = -\beta_4 - \mu, \lambda_4^0 = \frac{B\beta_1 - \beta_2\mu - \beta_3\mu - \mu^2}{\mu}, \lambda_5^0 = \frac{1}{2}\left(x_1 + \sqrt{y_1}\right),$$

and $\lambda_6^0 = \frac{1}{2}\left(x_1 - \sqrt{y_1}\right)$ where,

$$x_1 = \beta_5 D - \beta_7 - \beta_8 - 2\mu, y_1 = D^2\beta_5^2 - 2D\beta_5\beta_7 + 2D\beta_5\beta_8 + \beta_7^2 + 2\beta_7\beta_8 + \beta_8^2.$$

To prove equilibrium point E_0 is locally asymptotically stable, it is enough to show all the eigenvalues of Jacobian matrix $J(E_0)$ have negative real part. Clearly, λ_1^0, λ_2^0, and λ_3^0 are negative. Note that $\lambda_4^0 < 0$ if and only if $\frac{B\beta_1}{\mu} < \beta_2 - \beta_3 - \mu$.

Let $x_1<0$. When $y_1<0$, real part of eigenvalues λ_5^0 and λ_6^0 are negative and whenever $y_1>0$, real part of eigenvalues λ_5^0 and λ_6^0 are negative only when $x_1^2 - y_1 > 0$.

Clearly, $x_1<0$ and $x_1^2 - y_1 > 0$ implies $\beta_5 D<\beta_7+\beta_8+2\mu$ and $\beta_5 D(\beta_8+\mu)<\mu(\beta_7+\beta_8+\mu)$ respectively.

Theorem 4.1.2: The disease free equilibrium point $\left(E_{p_1}\right)$ is locally asymptotically stable if $B\beta_1>\mu(\beta_1+\beta_3+\mu)$ and

$$\min\left\{\frac{\mu(\beta_7+\beta_8+\mu)}{(\beta_8+\mu)}, \beta_7+\beta_8+2\mu\right\} > (\beta_5 D + n_2).$$

Proof: The Jacobian matrix $J\left(E_{p_1}\right)$ for system (1) associated with point $\left(E_{p_1}\right)$ is given by:

$$J\left(E_{p_1}\right) = \begin{bmatrix} -n_1-\mu & -\beta_2-\beta_3-\mu & 0 & 0 & 0 & 0 \\ -n_1 & 0 & 0 & 0 & 0 & 0 \\ -\mu & \beta_2 & -\mu & \beta_4 & -\beta_5 D & 0 \\ 0 & \beta_3 & 0 & -\beta_4-\mu & -n_2 & 0 \\ 0 & 0 & 0 & 0 & D\beta_5-\beta_7-\mu+n_2 & \beta_8 \\ 0 & 0 & 0 & 0 & \beta_7 & -\beta_8-\mu \end{bmatrix}$$

where,

$$n_1 = \frac{B\beta_1 - (\beta_2+\beta_3+\mu)\mu}{\beta_2+\beta_3+\mu}, n_2 = \frac{\beta_6\beta_3\left(B\beta_1 - (\beta_2+\beta_3+\mu)\mu\right)}{\beta_1\left((\beta_2+\beta_3+\mu)(\beta_4+\mu)\right)}. \quad \text{Let,}$$

$B\beta_1<\mu(\beta_1+\beta_3+\mu)$. (3)

Eigenvalues of $J\left(E_{p_1}\right)$ are:

$$\lambda_1^1 = -\mu, \lambda_2^1 = -\beta_4 - \mu, \lambda_3^1 = \frac{1}{2}\left(x_2 + \sqrt{y_2}\right), \lambda_4^1 = \frac{1}{2}\left(x_2 - \sqrt{y_2}\right), \lambda_5^1 = \frac{1}{2}\left(x_3 + \sqrt{y_3}\right), \lambda_6^1 = \frac{1}{2}\left(x_3 - \sqrt{y_3}\right).$$

Here,

$$x_2 = -n_1 - \mu, y_2 = -2n_1(2\beta_2 + 2\beta_3 + \mu) + \mu^2 + n_1^2, x_3 = \beta_5 D - \beta_7 - \beta_8 - 2\mu + n_2,$$

$$y_3 = (\beta_8 + n_2)(2D\beta_5 + \beta_8 + n_2) + 2\beta_7(\beta_8 - n_2) + D\beta_5(D\beta_5 - 2\beta_7) + \beta_7^2.$$

Note that two eigenvalues λ_1^1 and λ_2^1 are negative, let assume x_3<0, i.e.

$$\beta_7 + \beta_8 + 2\mu > \beta_5 D + n_2 \tag{4}$$

Moreover, $x_2^2 - y_2 = 4n_1(\mu + \beta_2 + \beta_3) > 0$ and $x_3^2 - y_3 > 0$ if

$$\mu(\beta_7 + \beta_8 + \mu) > (\beta_5 D + n_2)(\beta_8 + \mu) \tag{5}$$

Using similar logic and arguments used in previous theorem, we can say that all the eigenvalues of the matrix $J\left(E_{P_1}\right)$ have negative real part whenever our model satisfies all the three conditions (3), (4) and (5).

Theorem 4.1.3: The disease free equilibrium point $\left(E_p^*\right)$ is locally asymptotically stable if $\beta_7+\mu > \beta_5 D^*+\beta_6 C^*$,

$$\min\left\{A^*C^*\beta_6^2, A^*D^*\beta_5^2, E^*S^*\beta_1^2, n_6n_7, n_5n_8, n_7n_8\right\} > \beta_7\beta_8,$$

$\beta_6<\beta_8+\mu$ and $\beta_2+\beta_3+\mu>\beta_1 S^*$.

Proof: The Jacobian matrix $J\left(E_p^*\right)$ for system (1) associated with point $\left(E_p^*\right)$ is given by:

$$J\left(E_p^*\right)=\begin{bmatrix} n_3 & -\beta_1 S^* & 0 & 0 & 0 & 0 \\ \beta_1 E^* & n_4 & 0 & 0 & 0 & 0 \\ n_9 & \beta_2 & n_5 & \beta_4 & -\beta_5 D^* & 0 \\ 0 & \beta_3 & 0 & n_6 & -\beta_6 C^* & 0 \\ 0 & 0 & \beta_5 A^* & \beta_6 A^* & n_7 & \beta_8 \\ 0 & 0 & 0 & 0 & \beta_7 & n_8 \end{bmatrix}$$

where,

$$n_3=-(\beta_1 E^*+\mu), n_4=\beta_1 S^*-\beta_2-\beta_3-\mu, n_5=-\beta_5 A^*-\mu, n_6=-\beta_4-\beta_6 A^*-\mu,$$

$$n_7=\beta_5 D^*+\beta_6 C^*-\beta_7-\mu, n_8=-\beta_8-\mu, n_9=-\beta_5 A^*-\mu.$$

Clearly n_i are negative for all i=3,4,5,6,7,8,9 when $\beta_2+\beta_3+\mu>\beta_1 S^*$ and $\beta_7+\mu>\beta_5 D^*+\beta_6 C^*$.

Characteristic polynomial of $J\left(E_p^*\right)$ is:

$$\lambda^6+p_1\lambda^5+p_2\lambda^4+p_3\lambda^3+p_4\lambda^2+p_5\lambda^1+p_6=0.$$

where, $p_1=-(n_8+n_7+n_6+n_5+n_4+n_3)>0$,

$$\begin{aligned} p_2 &= A^*(C^*\beta_6^2+D^*\beta_5^2)+E^*S^*\beta_1^2-\beta_7\beta_8+n_3(n_4+n_5+n_6+n_7+n_8)+n_4(n_5+n_6+n_7+n_8) \\ &\quad +n_5(n_6+n_7+n_8)+n_6(n_7+n_8)+n_7n_8>0 \\ p_3 &= A^*C^*\beta_6(\beta_4\beta_5-\beta_6 n_8)-(n_3+n_4+n_5)(A^*C^*\beta_6^2-\beta_7\beta_8)-n_6(A^*D^*\beta_5^2-\beta_7\beta_8) \\ &\quad -A^*D^*\beta_5^2(n_2+n_4+n_8)-(n_5+n_6+n_7+n_8)(E^*S^*\beta_1^2+n_3n_4)-n_7n_8(n_3+n_4+n_5+n_6) \\ &\quad -(n_6+n_7+n_8)n_5(n_4+n_4)-(n_7+n_8)n_6(n_3+n_4+n_5) \end{aligned}$$

$$\begin{aligned} p_4 &= (A^*C^*\beta_6^2-\beta_7\beta_8)(n_3n_4+n_3n_5+n_4n_5)+n_6(A^*D^*\beta_5^2-\beta_7\beta_8)(n_3+n_4)+n_5n_6(E^*S^*\beta_1^2-\beta_7\beta_8) \\ &\quad +A^*E^*S^*\beta_1^2(C^*\beta_6^2+D^*\beta_5^2)-A^*C^*\beta_4\beta_5\beta_6(n_3+n_4+n_8)+A^*C^*\beta_6^2n_8(n_3+n_4+n_8) \\ &\quad +A^*D^*\beta_5^2(n_3(n_4+n_8)+n_8(n_4+n_6))+E^*S^*\beta_1^2((n_5+n_6)(n_7+n_8)-\beta_7\beta_8+n_7n_8) \\ &\quad +n_3n_4n_5(n_6+n_7+n_8)+n_6(n_3(n_4+n_5)+n_4n_5)(n_7+n_8)+n_3n_7n_8(n_4+n_5+n_6) \\ &\quad +n_7n_8(n_4(n_5+n_6)+n_5n_6) \end{aligned}$$

$$p_5 = -A^*D^*E^*S^*\beta_1^2\beta_5^2(\beta_6+n_8) + A^*C^*E^*S^*\beta_1^2\beta_4\beta_5\beta_6 - A^*C^*E^*S^*\beta_1^2\beta_6^2(n_5+n_8)$$
$$+ A^*C^*\beta_4\beta_5\beta_6(n_3n_4+n_3n_8+n_4n_8) - A^*C^*\beta_6^2(n_3n_4(n_5+n_8)+n_5n_8(n_3+n_4))$$
$$- A^*D^*\beta_5^2(n_3n_4(n_6+n_8)+n_6n_8(n_3+n_4)) + (\beta_7\beta_8 - n_6n_7)(E^*S^*\beta_1^2n_5 + n_3n_4n_5)$$
$$+ (\beta_7\beta_8 - n_5n_8)(E^*S^*\beta_1^2n_6 + n_3n_4n_6) + (\beta_7\beta_8 - n_7n_8)(n_5n_6(n_3+n_4))$$
$$- n_7n_8(n_5+n_6)(E^*S^*\beta_1^2 + n_3n_4)$$

$$p_6 = n_5n_6(n_7n_8 - \beta_7\beta_8)(n_3n_4 + E^*S^*\beta_1^2) + A^*C^*\beta_6n_8(E^*S^*\beta_1^2 + n_3n_4)(\beta_6n_5 - \beta_4\beta_5)$$
$$+ A^*D^*\beta_5^2n_6n_8(E^*S^*\beta_1^2 + n_3n_4)$$

$p_i>0$, i=3,4,5,6 if and only if

$$\min\left\{A^*C^*\beta_6^2, A^*D^*\beta_5^2, E^*S^*\beta_1^2, n_6n_7, n_5n_8, n_7n_8\right\} > \beta_7\beta_8$$

and $\beta_6<\beta_8+\mu$. Hence, the Characteristic polynomial of matrix $J\left(E_p^*\right)$ come to be Hurwitz polynomial under these conditions.

Global Stability

Theorem 4.2.1: The disease free equilibrium point (E_0) is global asymptotically stable in Λ when $\beta_1S<\beta_2+\mu$.

Proof: Consider the Lyapunov function: $L_1(t)=E+C$.

Hence,

$$L_1' = (\beta_1S - \beta_2 - \mu)E - (\beta_4 + \beta_6A + \mu)C\ .$$

$L_1' \le 0$ when $\beta_1S<\beta_2+\mu$. Moreover $L_1' = 0$ when $E=C=0$. The equilibrium point E_0 is the invariant set of the system (1) containing entirely in Λ; hence by the asymptotic stability theorem (Barbashin E. A., 1970; LaSalle J. P., 1976; LaSalle J., 1961) the equilibrium point E_0 is globally asymptotically stable.

Theorem 4.2.2: The asthma free equilibrium point $\left(E_{p_1}\right)$ is globally asymptotically stable in Λ if $\beta_1S<\mu$.

Proof: Let consider the Lyapunov function: $L_2=E+D+C$.

$$L_2' = (\beta_1 S - \mu)E - (\beta_5 A + \mu)D - (\beta_6 A + \mu)C ,$$

$L_2' \le 0$ when $\beta_1 S < \mu$ and $L_2' = 0$ only when all the three compartments, E, D and C are zero. Using LaSalle's Invariance principle, it is clear that every solution of the system (1), with initial conditions in Λ approaches to E_1 as $t \to \infty$. Hence, E_1 is globally asymptotically stable.

Theorem 4.2.3: The endemic equilibrium point $\left(E_p^*\right)$ is globally asymptotically stable.

Proof: Consider the Lyapunov function:

$$L_3(t) = \frac{1}{2}\left[\left(S(t) - S^*\right) + \left(E(t) - E^*\right) + \left(D(t) - D^*\right) + \left(C(t) - C^*\right) + \left(A(t) - A^*\right) + \left(M(t) - M^*\right)\right]^2$$

$$\begin{aligned} L_3' &= \left[\left(S(t) - S^*\right) + \left(E(t) - E^*\right) + \left(D(t) - D^*\right) + \left(C(t) - C^*\right) + \left(A(t) - A^*\right) + \left(M(t) - M^*\right)\right] \\ &\quad \left[S' + E' + D' + C' + A' + M'\right] \\ &= \left[\left(S(t) - S^*\right) + \left(E(t) - E^*\right) + \left(D(t) - D^*\right) + \left(C(t) - C^*\right) + \left(A(t) - A^*\right) + \left(M(t) - M^*\right)\right] \\ &\quad \left[B - \mu(S(t) + E(t) + D(t) + C(t) + A(t) + M(t))\right] \end{aligned}$$

By putting $B = \mu(S^* + E^* + D^* + C^* + A^* + M^*)$, we get

$$L_3' = -\mu\left[\left(S(t) - S^*\right) + \left(E(t) - E^*\right) + \left(D(t) - D^*\right) + \left(C(t) - C^*\right) + \left(A(t) - A^*\right) + \left(M(t) - M^*\right)\right]^2$$

Clearly, $L_3' \le 0$ and $L_3' = 0$ only when $S(t) = S^*$, $E(t) = E^*$, $D(t) = D^*$, $C(t) = C^*$, $A(t) = A^*$ and $M(t) = M^*$. Hence, point $\left(E_p^*\right)$ is globally asymptotically stable.

OPTIMAL CONTROL THEORY

Globally, many researchers have been working on controlling and developing effective vaccination to regulate different respiratory diseases. During the present study, optimal control theory is applied to develop an effective strategy which helps to curtail the spread and intensity of dyspnea, cough and asthma. In this model, four control variables have been used as described in Table 2.

Table 2. Control Variables

Control Variables	Description
μ_1	This function is applied to control the class of susceptible individuals who gradually turn into class of exposed individuals. This can be controlled by avoiding the susceptible individuals to loiter in polluted environment or by using mask kit to breathe pollutant-free air.
μ_2	This control function is applied to reduce the possibility of individuals who are suffering from dyspnea and are vulnerable to get affected by asthma. This can be controlled by proper medications and by improving their lifestyle at the initial stages of dyspnea.
μ_3	This function is applied to reduce the possibility of individuals who are suffering from cough and are prone to get affected by asthma. This can be controlled by proper medications and by improving their lifestyle at the initial stages of cough.
μ_4	This control function is applied on class of asthma infected individuals, which can be controlled by medication to avoid further exacerbation.

The system (1) is modified as below:

$$\begin{aligned}
\frac{dS}{dt} &= B - \beta_1 SE - u_1 S - \mu S \\
\frac{dE}{dt} &= \beta_1 SE - \beta_2 E - \beta_3 E + u_1 S - \mu E \\
\frac{dD}{dt} &= \beta_2 E + \beta_4 C - \beta_5 DA - u_2 D - \mu D \\
\frac{dC}{dt} &= \beta_3 E - \beta_4 C - \beta_6 CA - u_3 C - \mu C \\
\frac{dA}{dt} &= \beta_5 DA + \beta_6 CA + \beta_8 M - \beta_7 A + u_2 D + u_3 C - u_4 A - \mu A \\
\frac{dM}{dt} &= \beta_7 A - \beta_8 M + u_4 A - \mu M
\end{aligned} \tag{6}$$

The objective function including control variables for the model is given by:

$$J(u_i, \Lambda) = \int_0^T \left(A_1 S^2 + A_2 E^2 + A_3 D^2 + A_4 C^2 + A_5 A^2 + A_6 M^2 + w_1 u_1^2 + w_2 u_2^2 + w_3 u_3^2 + w_4 u_4^2 \right) dt \tag{7}$$

Here, Λ denotes set of all compartmental variables and A_i, i=1,2,…,6 are small positive constants to keep a balance in the size of the respective compartments. w_1, w_2, w_3 and w_4 are positive weight parameter which is associated with the control

functions u_1, u_2, u_3 and u_4 respectively. The objective of applied control theory is to stabilize the system by optimising these control functions. Optimal control condition is normalized from the weight constants w_1, w_2, w_3 and w_4.

Now, we will calculate the values of control variables from t=0 to t=T such that,

$$J\left(u_1(t),u_2(t),u_3(t),u_4(t)\right) = optimum\left\{J(u_i^*,\Lambda)/(u_1,u_2,u_3,u_4)\in\phi\right\} \tag{8}$$

Here, ϕ is a smooth function on the interval [0,1]. The optimal controls u_1^*, u_2^*, u_3^* and u_4^* are founded by accumulating all the integrands of equation (7) using the lower bounds and upper bounds respectively with the results of Fleming and Rishel (1975) (Fleming W. H. *et. al.*, 1975).

To optimise controls using the Pontrygin's principle, we construct a Lagrangian function which contains state equations and adjoint variables λ_1, λ_2, λ_3, λ_4, λ_5, and λ_6 as follows:

$$\begin{aligned}L(\Lambda,A) = {} & A_1S^2 + A_2E^2 + A_3D^2 + A_4C^2 + A_5A^2 + A_6M^2 + w_1u_1^2 + w_2u_2^2 + w_3u_3^2 + w_4u_4^2 \\ & + \lambda_1\left(B - \beta_1SE - u_1S - \mu S\right) + \lambda_2\left(\beta_1SE - \beta_2E - \beta_3E + u_1S - \mu E\right) \\ & + \lambda_3\left(\beta_2E + \beta_4C - \beta_5DA - u_2D - \mu D\right) + \lambda_4\left(\beta_3E - \beta_4C - \beta_6CA - u_3C - \mu C\right) \\ & + \lambda_5\left(\beta_5DA + \beta_6CA + \beta_8M - \beta_7A + u_2D + u_3C - u_4A - \mu A\right) \\ & + \lambda_6\left(\beta_7A - \beta_8M + u_4A - \mu M\right)\end{aligned} \tag{9}$$

The partially differentiation of the Lagrangian function with respect to each compartmental variable gives the adjoint equation variables A_i=(λ_1, λ_2, λ_3, λ_4, λ_5, λ_6) corresponding to the system:

$$\begin{aligned}-\frac{\partial L}{\partial S} &= -2A_1S + (\lambda_1-\lambda_2)\beta_1E + (\lambda_1-\lambda_2)u_1 + \mu\lambda_1 \\ -\frac{\partial L}{\partial E} &= -2A_2E + (\lambda_1-\lambda_2)\beta_1S + (\lambda_2-\lambda_3)\beta_2 + (\lambda_2-\lambda_4)\beta_5 + \lambda_2\mu \\ -\frac{\partial L}{\partial D} &= -2A_3D + (\lambda_3-\lambda_5)\beta_5A + (\lambda_3-\lambda_5)u_2 + \lambda_3\mu \\ -\frac{\partial L}{\partial C} &= -2A_4C + (\lambda_4-\lambda_3)\beta_4 + (\lambda_4-\lambda_5)\beta_6A + (\lambda_4-\lambda_5)u_3 + \lambda_4\mu \\ -\frac{\partial L}{\partial A} &= -2A_5A + (\lambda_3-\lambda_5)\beta_5D + (\lambda_4-\lambda_5)\beta_6C + (\lambda_5-\lambda_6)\beta_7 + (\lambda_5-\lambda_6)u_4 + \lambda_5\mu \\ -\frac{\partial L}{\partial M} &= -2A_6M + (\lambda_6-\lambda_5)\beta_8 + \lambda_6\mu\end{aligned} \tag{10}$$

The necessary conditions for Lagrangian function L to be optimal is:

$$-\frac{\partial L}{\partial u_i} = 0 \text{ , } i=1,2,3,4.$$

Hence,

$$u_1 = \frac{(\lambda_1 - \lambda_2)S}{2w_1}, u_2 = \frac{(\lambda_3 - \lambda_5)D}{2w_2}, u_3 = \frac{(\lambda_4 - \lambda_5)C}{2w_3}, \text{ and } u_4 = \frac{(\lambda_5 - \lambda_6)A}{2w_4}$$

Formulated required optimal controls are:

$$\begin{aligned} u_1^* &= \max\left\{a_1, \min\left(b_1, \frac{(\lambda_1 - \lambda_2)S}{2w_1}\right)\right\} \\ u_2^* &= \max\left\{a_2, \min\left(b_2, \frac{(\lambda_3 - \lambda_5)D}{2w_2}\right)\right\} \\ u_3^* &= \max\left\{a_3, \min\left(b_3, \frac{(\lambda_4 - \lambda_5)C}{2w_3}\right)\right\} \\ u_4^* &= \max\left\{a_4, \min\left(b_4, \frac{(\lambda_5 - \lambda_6)A}{2w_4}\right)\right\} \end{aligned} \tag{11}$$

Analytical results and variation in compartments of the model under impact of optimized controls have been visualised in numerical simulation section.

NUMERICAL SIMULATION

Numerical simulation of the present dynamical system is carried out to visualise it graphically and to analyse the behaviour of compartments before and after applying the controls.

Figure 2 represent overall flow of model in absence of controls. In about one month, rapid growth is seen in the class of exposed individuals under effect of pollutants present in polluted environment. It can be interpreted from the above figure 2 that under proper guidance and medication dyspnea and cough can be controlled and

Figure 2. variation in compartments with time

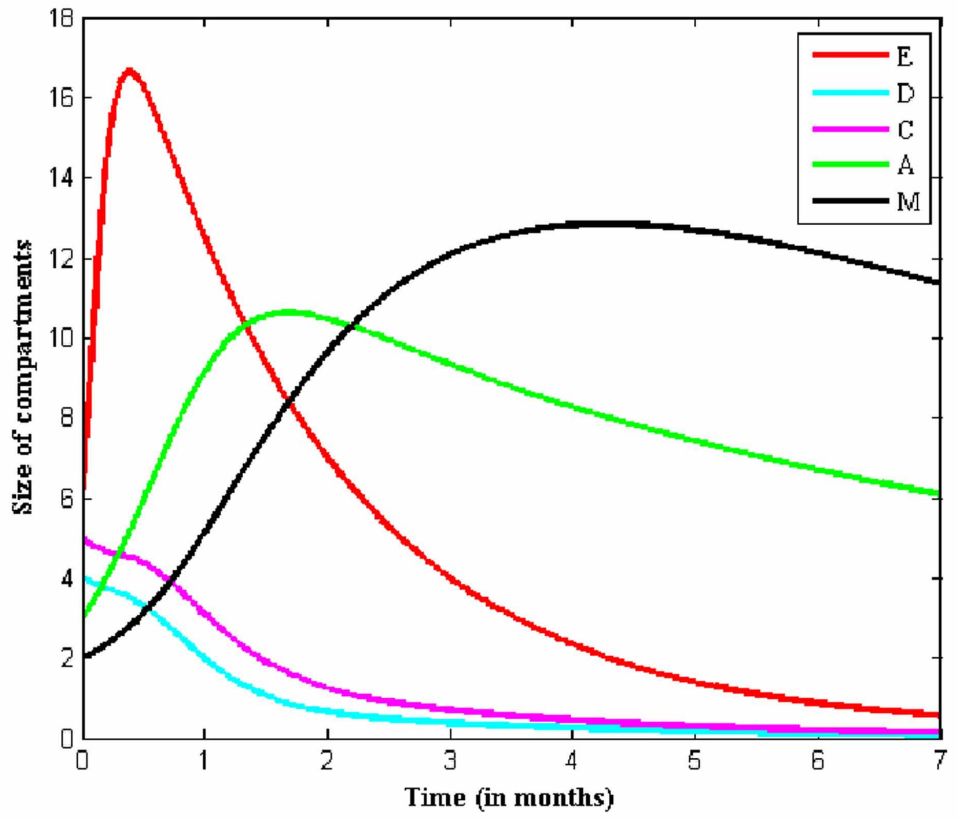
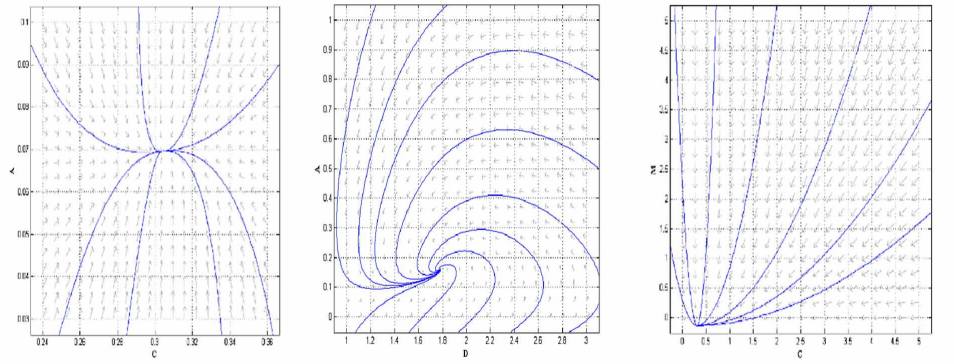


Figure 3. Representation of correlation of the diseases by curve and trajectory field



reduced in three months. As we know that asthma is lifelong and incurable in most cases, but its effects can be reduced by improving medication.

Direction and trajectory of first two graphs in figure 3 shows influence of dyspnea and cough in asthma exacerbation which suggests that controlling dyspnea and cough can help effectively to reduce asthma. Moreover, rapid and optimistic impact of medication on transmission of cough is seen in third graph.

The above three graphs given in figure 4 shows optimistic variation in class of individuals suffering from dyspnea, cough and asthma under combine influence of all the controls.

Change in class of medicated individuals with and without controls is shown in figure 5. Since control functions suggests improving medication, initially for 2-3 months more attention is required, with control, as compared to without control after that situation becomes normalised. Hence requirement of medication becomes low under impact of control strategy.

Figure 6 shows change in objective function with respect to time under influence of all the four controls.

Figure 4. Effect of controls on class of individuals suffer from dyspnea, cough and asthma

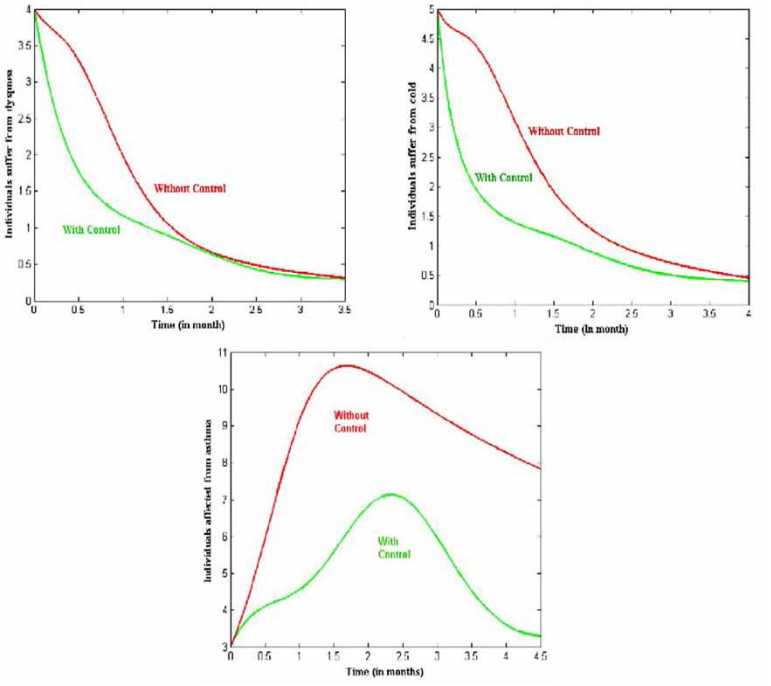


Figure 5. Variation in medication compartment under effect of all the controls

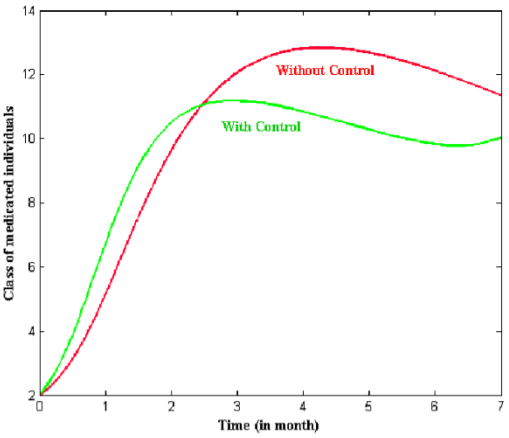
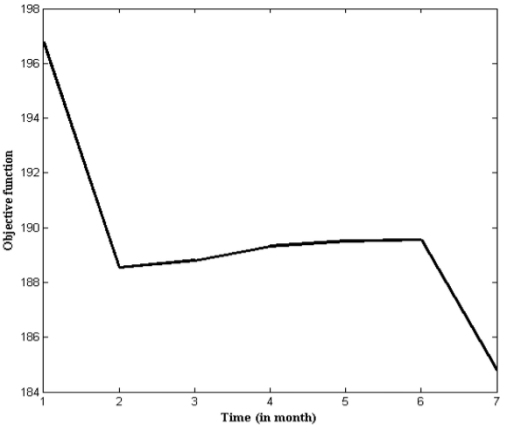


Figure 6. Change in objective function with time



CONCLUSION

Respiratory organs are influenced by infected and toxic air pollutants exhaust from vehicles, chemical industries and power plants. Exposed to air pollutants and allergens increases rate of morbidity and mortality due to frequent asthma episodes and other respiratory diseases. In the present study a compartmental mathematical model is generated and examine to analyse relation between air pollution and spread of respiratory diseases (dyspnea, cough and asthma). The model is constructed in such a way that it helps to analyse intensity of asthma under the influence of dyspnea and

cough. Local and global stability of the system is achieved under certain conditions. Here, Lyapunov function is used to prove global stability. Stability of the model is optimise by applying optimal control theory wherein four control variables are made based on different relative factors associated to air pollution and asthma exacerbation. Here, four control variables are used among which two controls are applied in such a way that helps to reduce spread and intensity of asthma caused by dyspnea and cough, and fourth control is associated with medication. By applying all the four controls, intensity or size of class of individuals having asthma can be decreased up to 32.92%. Which indicate that avoiding exposure to polluted air, following proper lifestyle and having appropriate medication for dyspnea, cough and asthma; positively affects the asthma exacerbation up to certain accepted level. Compared to without control applied, highest value of medication compartment is 12.85% lesser when all the controls are applied.

ACKNOWLEDGMENT

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Chapter 2
Analytical Study of Large-Scale Household Yagya Effects on Ambient Air Pollution:
A Study in NCR, India

Rohit Rastogi
https://orcid.org/0000-0002-6402-7638
Dayalbagh Educational Institute, India & ABES Engineering College, India

Devendra K. Chaturvedi
https://orcid.org/0000-0002-4837-2570
Dayalbagh Educational Institute, Agra, India

Mamta Saxena
Planning and Implementation, Ministry of Statistics, Delhi, India

Mayank Gupta
Tata Consultancy Services, Noida, India

Parul Singhal
ABES Engineering College, Uttar Pradesh, India

Mukund Rastogi
ABES Engineering College, India

Priyanshi Garg
ABES Engineering College, India

ABSTRACT

We all are living in such a world where the pollution and global warming are threats. Every year in India, at the time of festival seasons of Dussehra and Deewali, the smog and pollution are so much that millions of people suffer from different health

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issues. Also, the farmers of Punjab and Hariyana burn the Parali of their crops due to less awareness, and it becomes a challenge in the national capital, Delhi, to breathe. The government invests resources and the vehicles are allowed as per their even odd numbers. The authors team, including government officials, educationists, academicians, and students, along with IT experts, performed significant experiments on the ancient Indian Vedic science of Yajna and Mantra, and they found surprising results in the reduction of pollution on respective days. The chapter is an effort to present that scientific study conducted in 2018 and 2019 in random days after doing Yajna, and it was found that the pollution level was drastically decreased.

INTRODUCTION

Health Issues and Challenges in Global Scenario

Global Health is defined as the area of research, study and practice that places precedence on improving health and achieving rectitude in health for all people over world. There are certain issues and challenges that are faced by researchers till now. Some issues include Ebola, Yemen, Rohingya, Opioid Crisis, Gun Violence, Refugees, Tuberculosis and many more. Now to resolve these issues, there are some challenges. Some past challenges include Zika virus, MERS, H1N1 and many more. Now challenges are like "How to solve problem like Ebola?", "How to say goodbye to polio?" etc. Some efforts such as GHSA are also taken by 60 countries and various organizations to manage all issues.(Adleman,N.E., et al, 2002), (Yadav, V.,et al.,2018k) and (Chaturvedi D.K.,et al.,2013c)

Status of Healthcare in Indian Context

According to Indian Constitution, Each state government is responsible for improving its Healthcare facilities, raising the level of standard living and nutrition in their own state. In India, Healthcare system under public sector is free for those people who are under poverty line. Healthcare system under private sector consists of 81% doctors, 58% hospitals and 29% of beds in country. According to a survey, Private sector becomes the primary source of healthcare for rural and urban areas. In Medication, it is surveyed that in 2010, India consumes most number of antibiotic per head. But now in 2018, most of the antibiotics are substandard and fake and is not approved. Although there are 1.4 million doctors in India, yet India is not able to reach its Millennium Development Goals (eight international development goals for year 2015) related to health. Initiatives such as The Twelfth Plan, Public-private partnership, PM-JAY and

many more are taken for improving access of healthcare in India.(Brondino, N., et al, 2013), (Gupta, M.,et al.,2019a) and (Chaturvedi D.K.,et al.,2014b)

Arvind Kasthuri experimented in his research that studies the whole scenario of Indian healthcare system from ancient to modern one, the challenges that need to be overcome are represented in the form of 5 A's, that need to be solve in order to improve access and quality of healthcare system of India. First A represents "Awareness", this stresses upon the level of awareness of health among people. Second A represents "Access", this stresses upon the physical reach of healthcare facilities (allowed up to 5 km). Third A represents "Absence", this term stresses upon less number of personnel availability for Healthcare services. Fourth A represents "Affordability" or "Accommodation", this stresses upon the cost of healthcare system for people who are not able to afford it. Fifth A represents "Accountability" or "Acceptability", It defines rules and regulations to justify one party and to take responsibility for its activities. (2),(Chaturvedi D.K.,et al.,2013a) and (Hui Chu Tsai,et al.,2013)

Figure 1. 5 A's of Accessing Healthcare Services(Arvind Kasthuri, 2018)

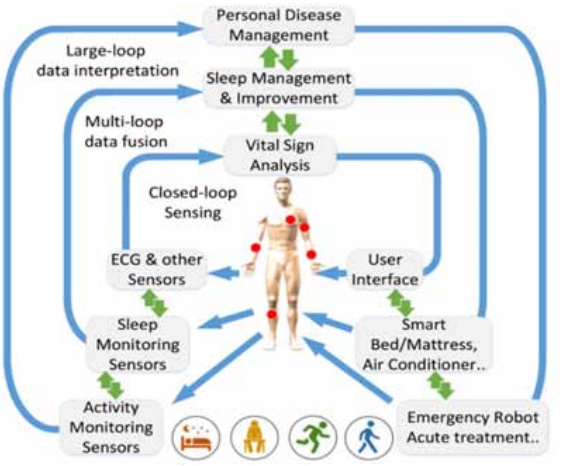
Healthcare 4.0

Healthcare 4.0 has been introduced by taking inspiration from "Industry 4.0". Healthcare 4.0 is basically the extension of concept of Industry 4.0 in such a way that a technically strict correlation develop between Patients & Healthcare Professionals and organization, technology & methodology. This is a patient-oriented system for data sharing amongst varied players. The major challenge for future is to make hospital a consolidated center to attend patient with personal care. Healthcare 4.0 is precisely linked with medicine and aims to identify the environmental, genetic and

way of living life approach of patient for providing patient with effective remedy. (Chauhan, S.et al.,2017a) and (Yadav, V.,et al.,2018j)

Zhibo Pang, Geng Yang, RidhaKhedri, Yuan-Ting Zhang experimented in their research that after taking this initiative, there happens significantly important progress in the direction of ideal vision of 8-P's of Healthcare. These 8 P's include Predictive, Participatory, Preventive, Patient-centered, Pre-emptive, Precision, Personalized and Persuasive healthcare. In this, Persuasive and Preventive Healthcare is achieved by installing smart and unobtrusive sensors in human body as well as in ambient environments through digitalization of living entity especially before that gets sick. Personalized and Personal Healthcare can be achieved by linking all the generic and healthcare data of an individual with the guaranteed privacy preservation in order to make it more precise. Patients are also provided with seamless consolidation of flows of patient and full scheduling and optimization of Healthcare process. A primary change by this initiative in Healthcare system is the shift of the paradigm design in a loop which is small to large, single to a multi and open to close one.(3) and (Chaturvedi D. K.,et al.,2013b)

Figure 2. The Healthcare 4.0 design paradigm shift: from open loop to closed loop; from single loop to multiple loops; and from small loop to large loop(Jhibo Pang et al., 2018)



Fog Computing

Fog computing, which is also known as Fog Networking is an system architecture that uses edge devices (devices which provides a point to enter into enterprise and

to the core network of service providers) to carry out computation, storage and communication locally. It can be perceived both in big data structures and large cloud systems. It consists of a control and a data plane. It also supports IoT concept. Both fog computing and cloud computing provides application, storage and data to their users. However, Fog computing has a larger geographical distribution and has a closer juxtaposition to end users. Fog computing help in providing the facilities for operation of storage, networking and computing services between end devices and cloud computing data centers (Rastogi, R.,et al.,28 Oct. 2018c) and (Saini H.,et al.,2019c).

Mist and Edge Computing

Rabindra K. Barik, Amaresh Chandra Dubey, AnkitaTripathi, T. Pratik, Sapna Sasane, Rakesh K. Lenka, Harishchandra Dubey, Kunal Mankodiya, Vinay Kumar experimented in their research that the term Mist Computing has been taken from the concepts of fog and edge computing by shifting some of the computation of the data to the edge of the work, devices and to the sensor which helps in building up the whole network for the cloud data center. Latency has been decreased and autonomy of a solution can be increased by using mist computing paradigm. Still it uses the cloud data center for processing, collecting and for providing availability to the user (Rastogi, R. et al., 2017b) and (Chaturvedi, D.K.et al.,2004)

Swarm Intelligence Techniques

Swarm intelligence technique is the emerging of the collection of intelligence of groups of simple agents. As computing tasks have to be well defined, fairly predictable, and needs to be completed in reasonable time, all these goals are the motivation behind the emergence of swarm intelligence. The three step process for designing swarm intelligence(SI) systems is, First “Identification of analogies” in systems of IT and swarm biology, Second “Understanding” the computer designing of realistic swarm biology, Third one is “engineering” in model simplifications and tuning for IT applications. Example of SI includes division of labor, group foraging of sociable insects etc.(Sharma, A.,et al.,2019a) and (Singh A.,et al.,2019d)

Pattern Classification

As there is presence of pattern in everything of universe, they can be observed either physically or mathematically. Pattern Recognition is the technique of recognizing patterns by making use of algorithms of machine learning. Pattern Recognition includes Pattern Classification which is done with the help of probabilistic classifiers

algorithm. Features of Pattern Recognition include recognizing familiar pattern accurately and quickly, unfamiliar objects, shapes and objects from various angles (even when they are partly hidden) with automaticity.(Sharma, P.,et al.,2018d) and (Gulati, M.,et al.,2019e)

YAJNA AND MANTRA SCIENCE

The word Yajna is derived from Sanskrit word "Yaj" which means worship of deities. Yajna lessen electromagnetic radiation levels from mobile phones, electronic and electric devices. Bhopal gas leak tragedy is one of the best examples of significance of yajna i.e. On the evening of third December 1984, the toxic MIC gas spilled from the carbide production line at Bhopal, many individuals kicked the bucket and a large number of individuals were hospitalized. In any case, one mile away from the industrial facility, two families were not influenced by the gas. It happened in light of the fact that they performed Yajna normally. In these families, no one passed on, no one was even hospitalized. This incident proved that Yajna is an antidote to pollution. (English Daily:" The Hindu"; news under the heading 'Vedic Ways to Beat Pollution')During Yajna, Saints chant Mantras which have calmative and encouraging effects on Human Beings and animals (Rastogi, R.,et al.,2017b) and (Chaturvedi D.K,et al.,2012a).

Ancient Vedic Tradition and Yajna

Vedicism was a sacrificial religion of Hindus which involves worship of numerous deities either male or female, who were related to natural resources and phenomena as Indra (God of thunder), Varuna (God of air) Agnidev (God of fire), etc. At that time Brahmans were appointed for worship of deities because society was divided on the basis of caste and Brahmans were supreme of them. Ancient Vedic Ceremonies centered on ritual sacrifice of animals and Yajna. Vedic rites were performed by sacrificing his/her things, which is more valuable than life, into the sacred fire of Yajna.The society thought that by immolation they could attain heaven (Vyas, P.,et al.,2018e) and (Gulati, M.,et al.,2018f).

Effect of Mantra and Yajna

The continuous release of unwanted industrial waste, which affects human beings and ecosystems,alters the physical, chemical and biological properties of soil and water. Yajna and Mantra takes care of human health and treat them. It also purifies air so that humans can breathe in natural and fresh air. In Yajna, various sorts of

prescriptions and herbs are vaporized. Yajnopavita offers them in the fire, when they go into the human body through pores of skin and nose, it is the simplest, least unsafe, least dangerous and best strategy for regulating a medication to arrive at every cell of the body. The whole process is known as Yagnopathy.(Agrawal, A.,et al.,2018g) and (Yadav, V.,et al.,2019b)

Technical Aspects of Mantra and Yajna Therapies

Pt. Shriram Sharma Acharya experimented in his research that The physical world has two basic energy systems: warmth and sound. In performing Yajna, these are two energies heat from fire of Yajna and the sound of Mantras is joined to accomplish wanted physical, physiological and otherworldly advantages. To evaluate different compound changes, it is important to realize the different substances offered in Yajna. They are portrayed beneath: Wood(Sandal-wood, Agar and Tagar wood, Deodar, Mango, Bilva, Bargad), Odoriferous substance (Pollachi, musk, tagar, javitri, Chandan, saffron, jaiphal, agar), Sweet Substances(sugar, honey, dried grapes, chhuhara),Substances with Healthy Constituents(munga, milk, chana, butter (ghee), rice, barley, masur or peas, til, kangu, arhar,fruits and cereals like wheat) and etc. The mixture of all these sacred things is known as Havisya.(13) and (Chaturvedi D.K.,et al.,2014a)

Scientific Study on Impact of Yajna on Air Purification

The air which we breathe is now filled with harmful gases like NO2,CO,SPM and RSPM. Gayatri and Yajna are the pillars of Indian philosophy and culture. Scientific study has also shown that yajna helps in air purification. The various Ahuties used in yajna like cow butter (ghee), Pipal wood (Ficus religiosa), Havan samagri (kapurkachari, gugal, nagarmotha, balchhaar or jatamansi, narkachura, sugandhbela, illayachi, jayphal, cloves and dalchini etc.) has powerful impact on the harmful gases present in earth's atmosphere. This is the most easy way in which we can reduce air pollution. The main thing of this is that there is no use of any chemical, all the things are natural so there is no harm to any element of earth in any way.(Singh, P.,et al.,2018h) and (Singh, V.,et al.,2018i)

Pushpendra K. Sharma, S. Ayub, C.N.Tripathi, S. Ajnavi and S. K. Dubey experimented in their research that pollution is the most dangerous problem in today's world. Air pollution is increasing at a very fast pace day by day. There is a great urge to develop some innovative ideas to stop this fast growing air pollution. Scientific study shows that yajna has a very positive impact on air purification.The Ahuties used in yajna like cow butter (ghee), Pipal wood (Ficus religiosa), Havan samagri (kapurkachari, gugal, nagarmotha, balchhaar or jatamansi, narkachura,

sugandhbela, illayachi, jayphal, cloves and dalchini etc.) proved very much beneficial in coping air pollution problem.There is no need to add any chemicals. Only these natural products help in air purification. The harmful gases present in the atmosphere like NO2, CO, SPM and RSPM gets reduced after the yajna.(20) and (Chaturvedi D.K.,et al.,2015a)

Effect of Yajna and Mantra on Human Health

There is a great impact of yajna and mantra on human health. By the fragrance of yajna many allergies of people get cured. Yajna also helps in air purification. Gayatri mantra and Yajna are the pillars of Indian philosophy and culture. In a *Yajna,* medicines and herbs are vaporized by offering them into the sacrificial fire, and when they enter the human body through the nose, lungs and the pores of the skin. This is the easiest, least toxic, less risky and most effective method of administrating a medicine to reach every cell of the body. There is no use of any type of harmful chemical in this process. This new concept is known as yagopathy. In the days to come it will become a very popular way of curing people as it is very easy and safe and it is directly related to Indian culture.(Saini, H.,et al.,2018j) and (Chaturvedi D K,et al.,2015b)

Rahul Raveendran Nair experimented in his research that one should take care of his/her health as if health is good then only person will be able to do work. In today's world everyday a new disease is taking birth. So technology should also work in same manner as to cope up with this issue. The yagopathy has proved beneficial in maintaining human health. The purpose behind the practice of Agnihotra Yajna is "letting incessant flow of energy (LIFE)" through our meridian lines and acupuncture points. This is the most easiest way of curing people. It is actually the Indian tradition and culture. The yajna helps in the betterment of human health as the ahuties that are given to sacrificial fire has very good advantages that can improve the health of humans without the use of any harmful chemical (Chaturvedi, D.K., 2012) and (Chaturvedi D.K.,et al.,2019).

METHODOLOGY

Instruments Required

In our research for pollution checking with collaboration of Central Pollution Control Board (CPCB), we have used many checkers to measure the various parameters that decide the level of pollution at certain place. We install Purifiers which can absorb the harmful particles present in air such as exhausts, dust and particles,

Chemical Balls which when absorb gases such as sulphur dioxide can change their color so that we come to know about the level of certain gas present in air, PM level Checkers which check the quantity of particulate matter present in air, Aura meter which detects the amount of water and minerals present in air and Energy Scanners which help in study of human health (Richa,et al.,2016a) and (Richa,et al.,2016b).

Bovi's Energy of Different Items, Places and Yagya

There is immense energy generation after Yajna. This energy is life force as has been mentioned in ancient Vedic scriptures as 'Prana Parjanya'. Prana or Life force or Chi as is known by Chinese, is the energy which is present in all living beings including plants, insects, animals and humans. This energy is also measure by people dealing in Vaastu shastra to determine the energy of a place. The Bovis energy measuring instrument is a pendulum in chain called dowsing pendulum and works on the dowsing principle. It is not considered as a scientific instrument and is under controversy amongst the present day material scientisits. In the experiments related to human health following data values would be required:

1. Bovis points of the Yagya Centre before and after Yagya
2. Bovis points of any one of the patient's home before and after their Yagya.

Bovis point is central point of our research paper. Add the relationship of Bovis energy point as one of the explanation point why Jan to mar point do not decrease. Thereby we will be able to address both points: 1. Yagya impact 2. Pranic Urja, in one go.

People may ignore the Energy in Bovis scale but in general people/science knows that such centre/temple have lot more positive energy then at least they can think with this perspective. Plotting simple relationship between Bovi's energy difference and rate of improvement can show and make people think it as serious point.

The energy in tap water is 13000 Bovis, fresh fruits is around 13000 to 20000Bovis, most of the bakery products it is negative. Cooked and refrigerated vegetables when fresh range between 8000 to 11000 but if old say two days old they have negative energy. Energy in hot milk tea was found to be extremely negative .

Energy in my house in my bedroom where we do daily havan, used to be 24000 Bovis and after Chandrayaan it is between 28000 to 31000 Bovis. Energy in water kept in havan, is 18000 after havan and in ghritavghran kal is very high.

Well you will be surprised to know that the actual energy in Ghritavghran jal is 50,00,000 Bovis or 5 million units!!!!

We can measure our pranic energy as well through Bovis. It's an interesting study. You can measure your energy before jap and after jap and there is an increase of

4 to 5000 after jap of 45 minutes. After havan there is a jump of another 4000 to 8000 Bovis. But after srinink ghritavaghran jal the energy is increased instantly by at least 30000 Bovis which is stupendous.

It is literally pranic energy that rains after completion of Yagya and even with short Yagya/ Agnihotra of 25 minutes there is such huge energy that remains throughout the day though starts diminishing after 2 hrs

Background of the Experiments

Gruhe Gruhe Yagya (GGY) program of Shantikunj, there was Yagya done on 24th June, 2018 and again on 2nd June, 2019 in about 5000 homes in and around Delhi at the same time simultaneously during the morning hours 9.00 am to 12.00 am. The format was same for all. It was demonstrated through video prepared and released from Shantikunj and local GayatriParivar (GP) people's efforts. Almost all the areas were covered in Delhi.(Chaturvedi D. K.,et al.,2012b)

Since a lot of medicinal fumes were released through Yagya simultaneously on the same day, one of the author cum researcher Dr. Mamta Saxena tried to check whether there was any lowering of air pollutants in the surrounding atmosphere. To her pleasant surprise the results were encouraging. In most of the Delhi centers, where live monitors were installed and data was captured, there was a perceptible reduction in the pollution levels on the day of havan. Next day it was increased in few centers. In few cases it has decreased in 20 to 22 centers out of 22 centers in Delhi.

She had tried to check the random status of lowering of pollutants on any given day in all the stations and have found that on neither instance, there was such a large reduction in so many stations.

RESULTSAND DISCUSSION

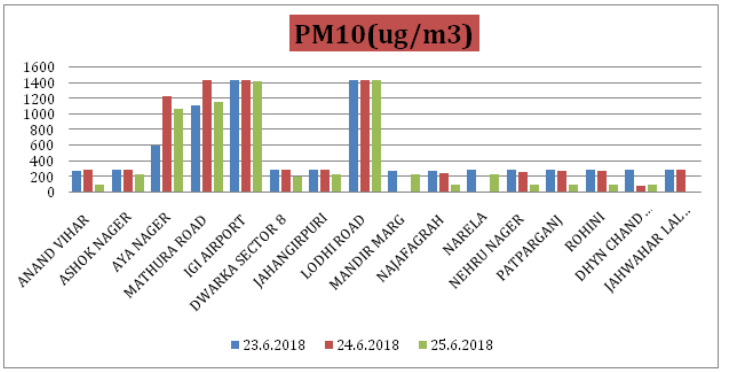
The Graphical Presentation of Various Pollutants day before (background), On the day of GGY

This experiment was conducted on June 24th, 2018 first and then repeated on 2ndJune 2019, almost after one year. The data has been collected from one day before (for background) to one day after (Post), from about 23 live monitoring stations in Delhi. The list of stations is given in the graphs below. This is just to see if there was any impact of such large scale yagya's conducted in individual homes spread all over in Delhi, on the gaseous pollution in the ambient air.

Observations

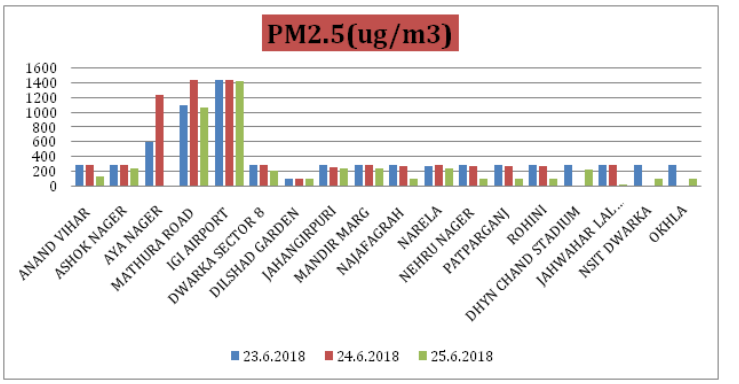
Pl. Refer the Annexure 1 for Air Pollution data in different centers of Delhi in 2018.
Graphical representation of 23,24,25 June 2018 in Delhi
PM 10 was reduced or remained same in almost 14 stations out of 16 in Delhi on the day of Yagya.

Figure 3. PM10 measurement on 3 days of June, 2018



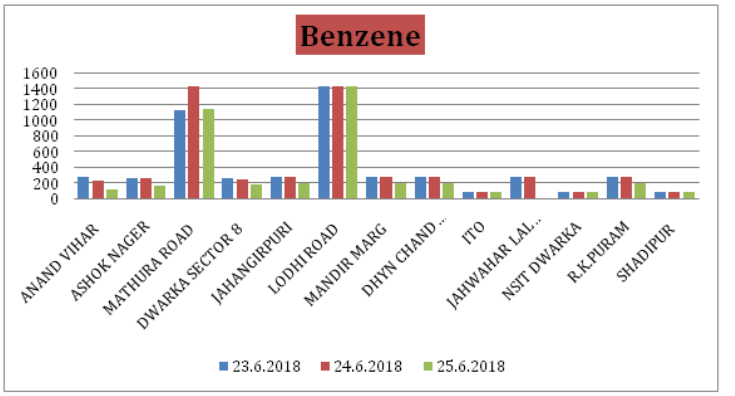
PM 2.5 was reduced or remained same in almost 14 stations out of 16 in Delhi on the day of Yagya.

Figure 4. PM 2.5 measurement on 3 days of June, 2018



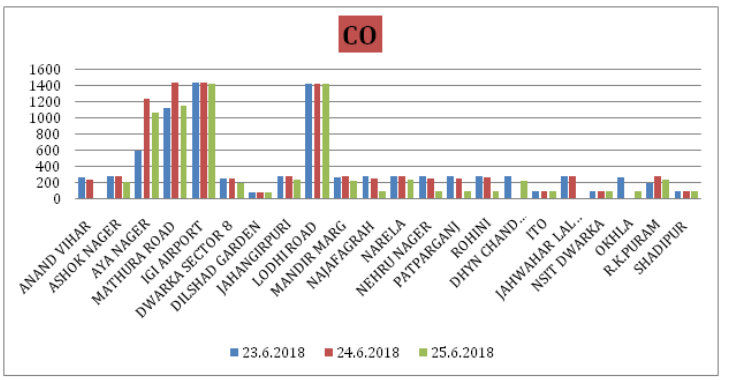
Benzene(ug/m3) Benzene reduced in almost 18/20 Centres on the day of Yagya

Figure 5. Benzene measurement on 3 days of June, 2018



Reduced on 20 centres out of total 22 Centres in Delhi, on the day of Yagya.
NO2 (ppm)

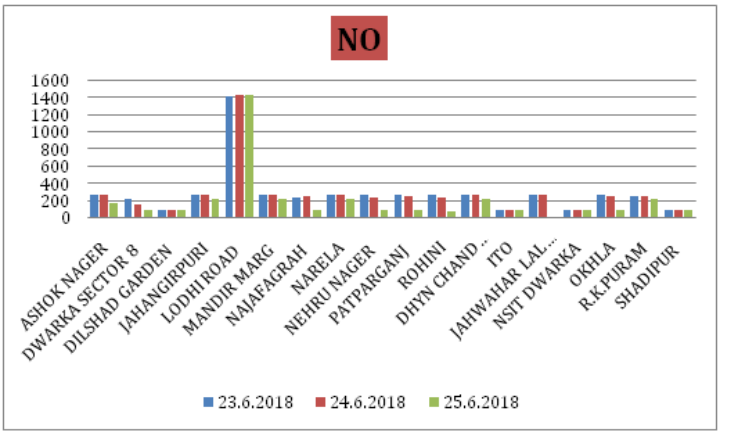
Figure 6. 5NO2 measurement on 3 days of June, 2018



NO2, Reduced or remained same in 23/25 Centres

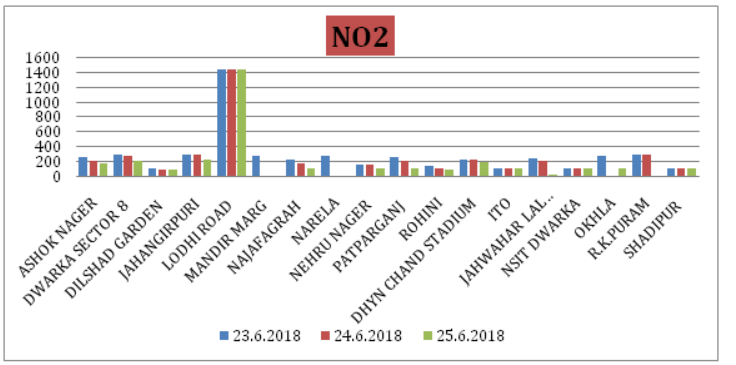
NOx Reduced or remained same in 24/26 stations on Yagya Day

Figure 7. NOx measurement on 3 days of June, 2018



Ozone Reduced in 19/25 Centres in Delhi

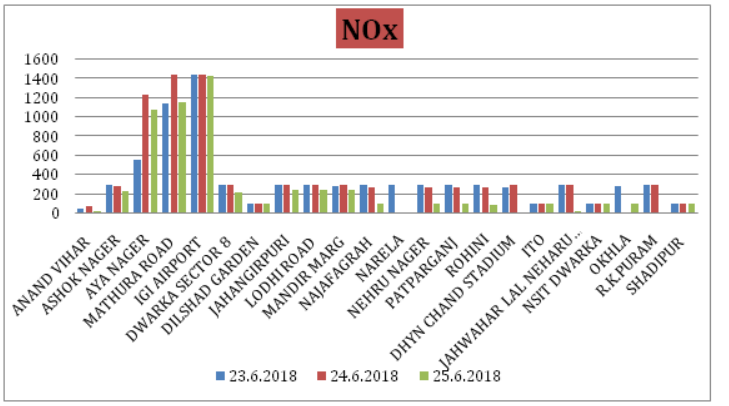
Figure 8. Ozone measurement on 3 days of June, 2018



The same experiment of conducting large scale Yagya was conducted after almost one year on June 2, 2019 in about 5000 homes spread all over Delhi and the results are as under:

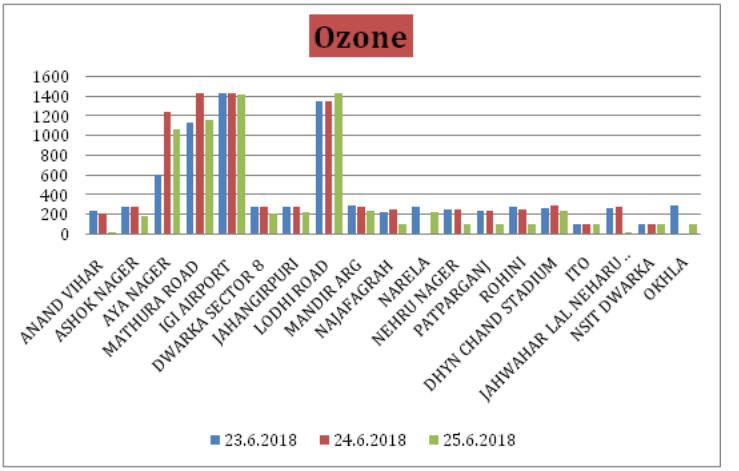
Results of Impact of Yagya on Different Pollutants on 2nd June 2019 in Delhi
Pl. Refer the Annexure 2 for Air Pollution data in different centers of Delhi in 2012.
PM 10
PM10 reduced in 16/16 centers which recorded data

Figure 9. PM10 measurement in 3 days of June, 2019



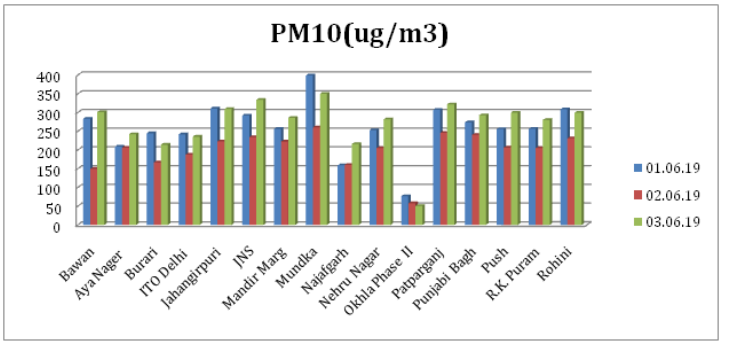
PM 2.5
PM 2.5 reduced at 16/19 centers where it was recorded

Figure 10. PM 2.5 measurement in 3 days of June, 2019



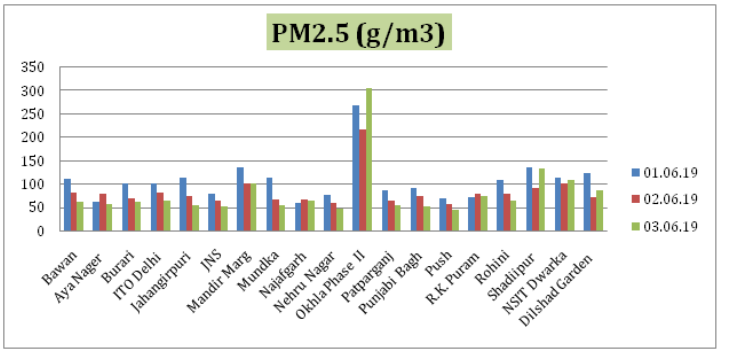
CO (mg/m3)
CO reduced at 18/19 Centers in Delhi

Figure 11. CO measurement in 3 days of June, 2019



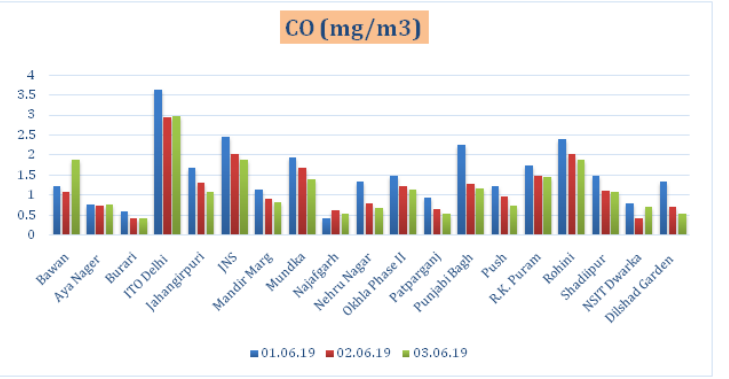
NO2 (ug/m3)

Figure 12. NO2 measurement in 3 days of June, 2019



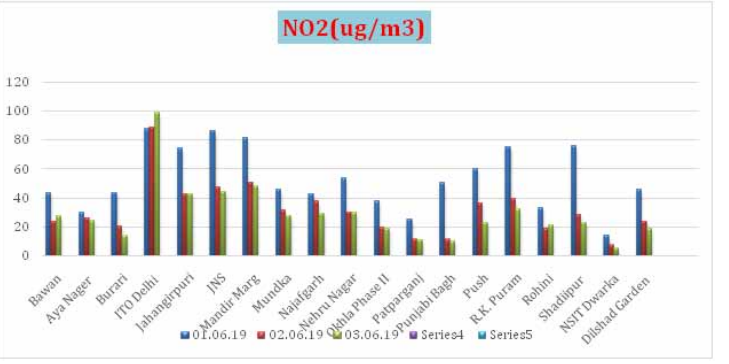
Reduced at 19/19 Centers on the day of Yagya in Delhi
NO(ug/m3

Figure 13. NO measurement on 3 days of June, 2019



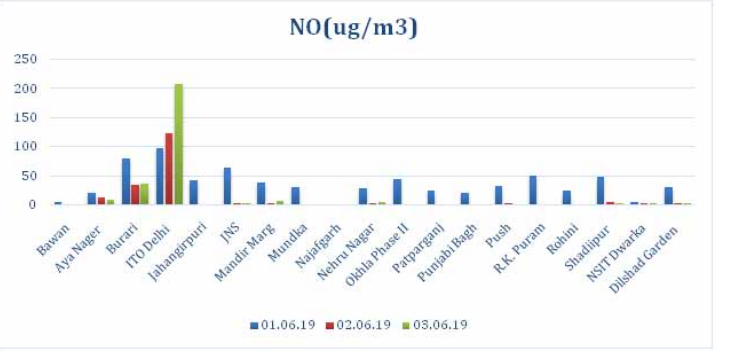
NO reduced on 18/19 Centres in Delhi on the day of Yagya
NOx (ppb)

Figure 14. NOx measurement in 3 days of June, 2019



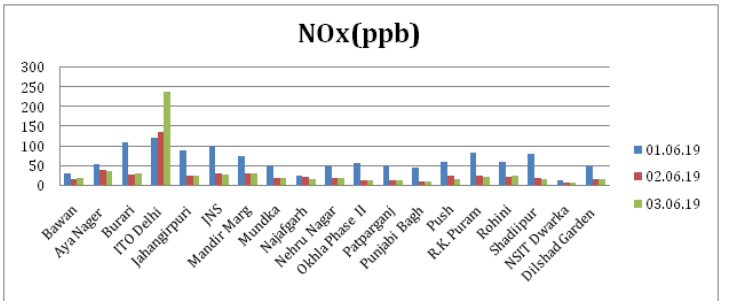
Reduced on 18/19 Centers in Delhi on the day of Yagya
Ozone (ug/m3)

Figure 15. Ozone measurement on 3 days of June, 2019



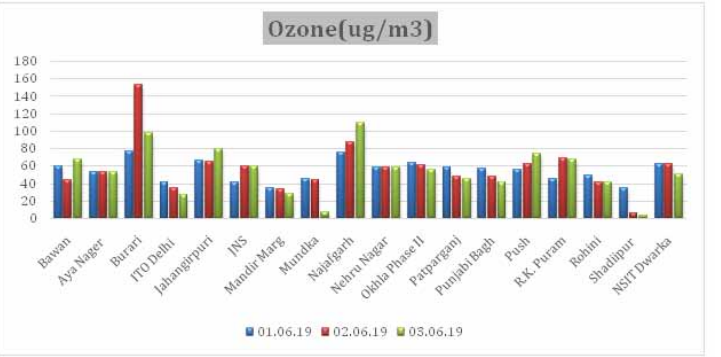
Reduced at 18/19 Centers in Delhi on the day of Yagya

Figure 16. Ozone measurement in 3 days of June, 2019



SO2 (ug/m3)

Figure 17. SO2 measurement in 3 days of June, 2019



SO2 reduced or remained equal in almost all 17 centers in Delhi

The above data is sufficient enough to explain that the Yagya Process reduced significantly the above particles from atmosphere. It has an overall impact even with small individual Yagyas/ Agnihotras performed in individual homes simultaneously. When other days data was examined for seeing the random trend it was observed that at any point of day data had increased at 50% or more stations, which was the random trend. Two dates were selected randomly 21st and 22nd June, 2018 in the years 2018 and 2019 to see the trend of pollutants in various centres in Delhi. The data collected on these dates has been compared and it is found that pollution is increasing on more than half the centres for almost all the pollutants. Hence getting a trend of lowering of pollutants in almost 90% or more centres shows statistically significant trend.

This experiment is indicative of the trend and needs to be done at larger levels eg in about 15000 to 20000 houses, simultaneously in Delhi

NOVELTY IN OUR WORK

The yagya and Mantra therapy is completely different form of treatment form those which we have today. The treatment method today involved eating up of medicines which are made from different chemicals and in some cases have found to have ill-effect in our body if taken for long time. Some methods of treatment prevalent today are based on injecting some medicines in the body of the patient by mean of

injection or other pins. The homeopathy method of treatment also require eating of chemicals though they are given in mix with sugar balls. This method of treatment is slow and requires long time to cure a patient. Also the disease first rises to its maximum level and then start to cure in this method of treatment. The ayurvedic method of treatment which has roots in ancient Indian heritage is also a popular method of treatment especially in villages and now is also becoming popular in cities. But this method also requires eating of Spices and other useful and healthy natural product. These products are mostly bitter and therefore patient don't prefer the ayurvedic Method. The "Yagya and Mantra therapy" is a completely different method of treatment. It neither require eating of costly and chemical rich medicine nor it require injecting of chemicals in the body of the patient. The disease is cured by the fumes which rises from doing a special type of "Yagya" in a closed room. The material for the "Yagya" is prepared from extensive research. The fumes have some feature which when inhaled effect the disease causing bacteria or virus and thus cure the disease. It is also not a slow process the considerable result is seen at the end of the "Yagya" in the patient. The different experiment conducted at different places also has proven the usefulness and power of the "Yagya and Mantra therapy". As the method don't involve consumption of medicine or any type of injection, the cost to perform the therapy is too low. It also don't require expensive machine to perform operations. Thus the "Yagya and Mantra therapy" is the treatment method which can revolutionize the era of medical science and the way disease are cured will also change significantly by the use of this therapy. It also has the power of curing deadly disease like cancer which now require doing Kemo therapy which is very painful and costly. Thus "Yagya and Mantra Therapy" can also be called as treatment of the future.

RECOMMENDATIONS

The "Yagya and Mantra therapy" is very useful for patient suffering from Diabetes. The current form of treatment available is to eat the medicine lifelong which will just control disease form increasing. But no complete cure is of diabetes is there. The "yagya and mantra therapy" has been found to be very useful while dealing with diabetes. The experiment done on various patient has proved that it has significantly affected the disease. The "Yagya and Mantra therapy "is thereby a strong medium of treatment especially in case of diabetes.

For other diseases the research is still going to find similar method of treatment. The therapy is also good for lowering the pollution content from atmosphere.

FUTURE SCOPE, LIMITATIONS, APPLICATIONS Of YAGYA THERAPY And MANTRA THERAPY

Future Scope

There are many scopes of Yagya Therapy and Mantra Therapy. Some are mentioned below:-

1- Diseases like hypertension, migraine, depression, asthma, arthritis, blood pressure, anxiety etc. will be cured by yagya therapy and mantra therapy.
2- Water can be purified through yagya therapy.
3- Fertility of the soil can be improved by these therapies.
4- Different skin diseases can also be cured.
5- Growth of organic farming can be increased through Yagya Therapy.
6- It also helps to increase yield in the field which helps farmers.
7- It also helps in increasing rain in the drought regions.
8- Strep throat, urinary tract infections and tuberculosis which is caused by bacteria's can easily be cured.
9- Ringworm and athlete's foot which is caused by fungi can also be cured.
and malaria, swine flu can also be cured through these therapies.

Limitations

1- FAITH ISSUE - Many people or patients have belief on homeopathy and allopathic that they will be cured easily and feel better within few days. So, people have less belief on Yagya and Mantra Therapy. So, they treated it as unfaithful and have less profit.
2- KNOWLEDGE - The number of subjects is less within patients. Patients don`t know about this therapy.
3- TREATMENT - Duration of treatment is long. It takes long time to recover.
4- DURATION - Patient arrives late for their treatment.

Applications

In a Yagya, medicines and herbs are vaporized by offering them into the sacrificial fire, and they enter the human body in a vaporous form through the nose, lungs and the pores of the skin. This might be proved to be easiest, least toxic, less risky and most effective method of administrating a medicine to reach every single cell of the body. It is used for the treatment of physical and mental disease. It consists of various Samidha which create desired effects. It may lead to the development of a

scientifically established Yagyopathy and also in other therapies of the world such as Allopathy, Homeopathy, Chromopathy, Naturopathy, etc. It is used in herbal/ plant medicinal preparation is used in anti-tuberculosis yagya.

CONCLUSION

From Pattern Classification in the study, it can be concluded that the proposed schemes GA with nearest neighbor techniques and GA with PNN are the efficient techniques. From Expert Design, it can be concluded that it is undeniably reliable in terms of providing reasonable and highly valuable decisions. Knowledge and experiences from a human expert can lead to the critical decision-making in achieving success. From Artificial Intelligence, AI is at the centre of a new enterprise to build computational models of intelligence. The main assumption is that intelligence (human or otherwise) can be represented in terms of symbol structures.

From Machine Learning, it can be concluded that it is a technique of training machines to perform the activities a human brain can do, even though bit faster and better than an average human-being. From Swarm Intelligence Techniques, it can be concluded that basically it is a set of algorithms and can be used in the context of forecasting problems. From Robotics, it can be concluded that robots are useful in many ways. For instance, it boosts economy because businesses need to be efficient to keep up with the industry competition. Therefore, having robots helps business owners to be competitive, because robots can do jobs better and faster than humans can. From Advanced numerical computation and optimization, it can be concluded that We have achieved an asymptotically optimal scale-up of the numerical effort with the number of spatial discretization points based on inexact SQP with an inner generalized Newton Picard preconditioned LISA which features extensive structure exploitation in a two-stage solution process for the possibly no convex QPs for which we have developed a condensing approach and a PASM.

From quantum inspired soft computing, we can conclude that it explores the use of a hybrid soft-computing paradigm for the prediction of the adsorption capacity of an environmentally-friendly and low-cost adsorbent. From intelligent control, we can conclude that it is a class of control techniques that use various artificial intelligence computing approaches like neural networks, Bayesian probability, fuzzy logic, machine learning, reinforcement learning, evolutionary computation and genetic algorithms. From Applications and experience with deployed systems, it can be concluded that it is the set of contents in the box. An application needs at least one deployment type, as it determines how to install the app. From ambient learning, it can be concluded that it is the idea that we don't need to interact directly with any devices. As computing becomes more pervasive, it vanishes into the environment

around us. From Learning Classifier System, it can be concluded that the trained model is a set of rules/classifiers rather than any single rule/classifier and it was commonly defined as the combination of 'trial-and-error' reinforcement learning with the global search of a genetic algorithm.

From hybrid intelligent system, we can conclude that it have resulted in the fusion of knowledge-based system, artificial neutral network etc. In software engineering and management, we have looked at some key concepts, themes and skills related to software development and allow us to develop the fundamental knowledge, understanding, and analysis and synthesis skills. From neuron-fuzzy systems, it can be concluded, it describes several adaptive neural and fuzzy networks and introduces the associative memory class of systems - which describe the similarities and differences existing between fuzzy and neural algorithms. From focus on the physics and biology - based approaches and algorithms. It is based on swarm intelligence, biological systems, physical and chemical systems.

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Chapter 3
Spread of Tuberculosis Among Smokers: A Mathematical Model

Purvi M. Pandya
Department of Mathematics, Gujarat University, Ahmedabad, India

Ekta N. Jayswal
Department of Mathematics, Gujarat University, Ahmedabad, India

Yash Shah
GCS Medical College, Ahmedabad, India

ABSTRACT

Smoking tobacco has some hazardous implications on an individual's physical, physiological, and psychological health; health of the passive smokers near him or her; and on the surrounding environment. From carcinomas to auto-immune disorders, smoking has a role to play. Therefore, there arises a need to frame a systemic pathway to decipher relationship between smoking and a perilous disease such as tuberculosis. This research work focuses on how drugs or medications can affect individuals who are susceptible to tuberculosis because of smoking habits and also on individuals who have already developed symptoms of tuberculosis due to their smoking addiction. The mathematical model is formulated using non-linear ordinary differential equations, and then threshold is calculated for different equilibrium points using next generation matrix method. Stability analysis along with numerical simulations are carried out to validate the data.

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INTRODUCTION

One of the most crucial yet common issue that humans can face, are health issues. It may be attributed to earning their livelihood. It sometimes shudders mental health of people who can't withstand them positively. These causes mental imbalance which sometimes leads to behavioral changes and also becomes the reason for life-threatening habits such as smoking, drinking, drugs addiction etc. Smoking which is found to be very common among all age groups may lead to pulmonary, hepatic, cardiac or gastrointestinal diseases, carcinomas, ulcers, etc. turning out to be the main reasons for extensive mortality and morbidity all around the world. People adopt these habits for the sake of pleasure or as a form of stress release or succumbing to peer pressure. Disease like tuberculosis has become common issue among smokers. It can be cured if they complete prescribed course within a given time interval. Also, advanced medical science has helped to curtail severity of health issues prevailing among smokers by utilizing novel methods such as nicotine containing chewing gum, nicotine patch, nicotine spray, nicotine based nasal formulations etc. Different advanced programs are also developed in various areas to spread awareness regarding how to use new methods affectively to quit smoking.

Many researchers have given extraordinary contributions to this field opening new insights of customized and impactful drug delivery system. Several theoretical as well as practical aspects were brought into light. An epidemiological relation between smoking and individuals suffering from tuberculosis was reviewed from the UK, India, china and the USA (Davies *et al.,* 2006). Also, a question was discussed to whether the age of individual matters on the impact of cigarette smoking on tuberculosis (Feng *et al.,* 2014). Even many statistical analyses were done along with surveys of different areas. To visualize the transmission dynamics and spread of the disease many deterministic models are designed. The basic models such as *SIS*, *SIR*, *SIRS*, *SEIR*, *SEIRS*, etc. were constructed to study the transmission of tuberculosis among smokers along with many latent stages using different conditions and assumptions. A compartmental mathematical model to estimate the impact of tuberculosis and its control was projected together with incorporated changing trends in smoking (Basu *et al.*, 2011). Models are constructed with different latent stages for tuberculosis transmission found in the regions of Asia-pacific (Trauer *et al.*, 2014). Also, modelling and data analysis for tuberculosis epidemic in Russia (Perelman *et al.*, 2004), model of tuberculosis with drug resistance effect (Ronoh *et al.*, 2016), model to simulate disease among population with stability analysis (Koriko and Yusuf, 2008), global dynamics of tuberculosis containing three stages susceptible, latent and active stage (Guo, 2005) and global stability with early latent stage (Guo and Li, 2006) were discussed. A model with exogenous reinfection for tuberculosis consisting of four compartments (Feng *et al.*, 2000), model with

recurrent infection and vaccination based on *SEIR*-model with force of infection for tuberculosis (Nainggolan *et al.*, 2013), practical aspects of backward bifurcation with seven compartments containing both fast and slow progression levels of infection and two different models (Gerberry, 2016), model concluding the prospects of controlling and improving case detection of tuberculosis (Okuonghae and Omosigho, 2011) were formulated and analyzed. So, different models along with stability analysis, bifurcation analysis, etc. were explored and analyzed. An epidemiological model with six compartments containing uninfected, latent stage and highly infected stage was considered as well. This was to investigate the effects of different cases on tuberculosis (murphy *et al.*, 2002). Later an extension to this model was formulated and the influence of backward bifurcation was modelled to study the transmission of epidemic tuberculosis with reinfection (Singer and Kirschner, 2004). Even many case-control studies were conducted related to smoking and death in India (Jha *et al.*, 2008). A similar eco-epidemiological model was formulated for prey and predators with saturated incidences (Saifuddin *et al.*, 2016), the stability analysis, existence of equilibrium points, etc. of this model was also discussed (Saifuddin *et al.*, 2017) and the epidemiological association between Smoking and tuberculosis and immunopathogenesis was discussed (Davies *et al.*, 2006). A study on tuberculosis recurrences was done and concluded that it plays a very important role in certain population and they can again be infected easily (De Viedma *et al.*, 2002).

In section below, model is formulated mathematically using system of non-linear differential equations along with computation of basic reproduction number. Next, stability is analyzed followed by numerical simulation and finally concluding the findings is in last section.

Mathematical Model

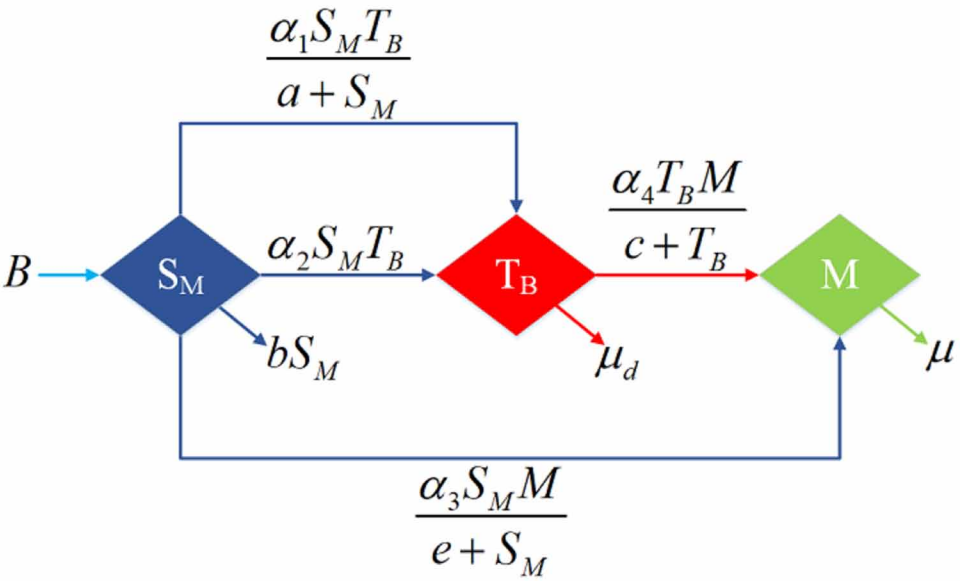
The transmission of tuberculosis among smokers is scrutinized in this model which is based on *SIR*-model. As shown in Figure 1, it comprises of three compartments namely smokers, tuberculosis and medication. Let S_M and T_B denote the smokers and individuals suffering from tuberculosis respectively and M denotes medication. we have considered intra-class competition in smokers (b) as smokers and individuals suffering from tuberculosis cannot have the same competing ability. Some saturated incidences are considered for transmission of disease as well as for medication. Other notations used in this research and which indicates the transmission rates among different compartments is described in Table 1.

Using parametric values, the mathematical model is formulated. The system of non-linear differential equations is as follows:

Table 1. Notations and Parametric values

Notation	Description	Parametric values
B	New recruitment rate	0.6
a	Half-saturation constant for disease transmission	4
b	Intra-class competition between smokers	2
c	Half-saturation constant of medication for TB patients	2
e	Half-saturation constant of medication for smokers	4
c_1	Conversion efficiency of medication on smokers	3
c_2	Conversion efficiency of medication on TB patients	7
α_1	Rate of infection with saturated incidence	0.5
α_2	Rate at which smokers get TB infection	0.7
α_3	Rate at which smokers starts medication to quit smoking	0.2
α_4	Rate at which TB patients procuring medication	0.6
μ_d	Death rate of infected TB patients	0.2
μ	Natural Death rate	0.2

Figure 1. Mathematical model



$$\frac{dS_M}{dt} = BS_M - \frac{\alpha_1 S_M T_B}{a + S_M} - \alpha_2 S_M T_B - \frac{\alpha_3 S_M M}{e + S_M} - bS_M{}^2$$

$$\frac{dT_B}{dt} = \frac{\alpha_1 S_M T_B}{a + S_M} + \alpha_2 S_M T_B - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B \tag{1}$$

$$\frac{dM}{dt} = \frac{c_1 \alpha_3 S_M M}{e + S_M} + \frac{c_2 \alpha_4 T_B M}{c + T_B} - \mu M$$

where $S_M+T_B+M \leq N$. Also, $S_M>0$; $T_B, M \geq 0$.

From above system (1), we can obtain

$$\frac{dS_M}{dt} \leq S_M (B - bS_M),$$

$$\frac{dT_B}{dt} \leq \alpha_2 S_M T_B - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B, \tag{2}$$

$$\frac{dM}{dt} \leq (c_1 \alpha_3 S_M + c_2 \alpha_4 T_B - \mu) M$$

From system (2) we get,

$$\limsup_{t \to \infty} S_M(t) \leq \frac{B}{b}.$$

Now, from the second equation of the system (2) we get,

$$\frac{dT_B}{dt} \leq \left(\alpha_2 S_M - \frac{\alpha_4 M}{c + T_B} - \mu_d \right) T_B \leq \frac{T_B}{c + T_B} \left(\alpha_2 c \frac{B}{b} - \alpha_4 M - \mu_d c - T_B \left(\mu_d - \alpha_2 \frac{B}{b} \right) \right)$$

$$\therefore \limsup_{t \to \infty} T_B(t) \leq \frac{\alpha_2 cB - \alpha_4 Mb - \mu_d cb}{\mu_d b - \alpha_2 B}.$$

Again, from the third equation of the system (2) we get,

$$\frac{dM}{dt} \le \left(c_1\alpha_3 S_M + c_2\alpha_4 T_B - \mu\right) M \le \left(c_1\alpha_3 \frac{B}{b} - c_2 c\alpha_4 - \frac{c_2{\alpha_4}^2 Mb}{\mu_d b - \alpha_2 B} - \mu \right) M$$

$$\therefore \limsup_{t\to\infty} M(t) \le \frac{\left(\alpha_2 B - \mu_d b\right)\left(\mu b + c_2 c\alpha_4 b - c_1\alpha_3 B\right)}{b^2 c_2 {\alpha_4}^2}.$$

Using it we get, $\limsup_{t\to\infty} T_B(t) \le \frac{b\mu - c_1\alpha_3 B}{bc_2\alpha_4}$.

Now,

$$\limsup_{t\to\infty}(S_M(t) + T_B(t) + M(t)) \le \limsup_{t\to\infty} S_M(t) + \limsup_{t\to\infty} T_B(t) + \limsup_{t\to\infty} M(t)$$
$$= \frac{c_2 b\alpha_4\left(B\alpha_4 + c\left(\alpha_2 B - b\mu_d\right)\right) + \left(b\alpha_4 + \alpha_2 B - b\mu_d\right)\left(b\mu - c_1\alpha_3 B\right)}{b^2 c_2 {\alpha_4}^2}$$

Therefore, the feasible region of the model is

$$\Lambda = \left\{ (S_M, T_B, M) \in R^3 : S_M + T_B + M \le \frac{\begin{array}{c} c_2 b\alpha_4\left(B\alpha_4 + c\left(\alpha_2 B - b\mu_d\right)\right) \\ + \left(b\alpha_4 + \alpha_2 B - b\mu_d\right)\left(b\mu - c_1\alpha_3 B\right) \end{array}}{b^2 c_2 {\alpha_4}^2} \right\}.$$

Now, the disease-free equilibrium point of the model is $Y_0 = \left(\frac{B}{b}, 0, 0\right)$, infection-free point is

$$Y_1 = \left(\frac{e\mu}{c_1\alpha_3 - \mu}, 0, \frac{ec_1\left(B\left(c_1\alpha_3 - \mu\right) - be\mu\right)}{\left(c_1\alpha_3 - \mu\right)^2} \right),$$

medication-free point is

$$Y_2 = \left(r_1, \frac{(a+r_1)(B\alpha_2 - b\mu_d) + r_1\alpha_1 b}{\alpha_2(\alpha_1 + \alpha_2(a+r_1))}, 0 \right)$$

where

$$r_1 = RootOf\left(\alpha_2 Z^2 + (a\alpha_2 - \mu_d + \alpha_1)\,Z - \mu_d a\right)$$

and endemic point is $Y^* = \left(S_M{}^*, T_B{}^*, M^*\right)$ where, $S_M{}^* = r_2$,

$$T_B{}^* = \frac{c\left(e\mu - r_2\left(c_1\alpha_3 - \mu\right)\right)}{r_2\left(\left(c_1\alpha_3 - \mu\right) + c_2\alpha_4\right) + e\left(c_2\alpha_4 - \mu\right)} \text{ and}$$

$$M^* = \frac{\left[\begin{array}{l}
(e+r_2)\{(r_2b-B)((c_1-c_2)(a\alpha_4c_2e((a\alpha_2+\alpha_1)(\alpha_3c_1+\alpha_4c_2)-\alpha_4c_2)) + \mu_d a\alpha_4{c_2}^2(a+e) \\
(2\mu-\alpha_3c_1-\alpha_4c_2)) - \mu_d a^2\alpha_3c_1c_2(r_2\alpha_4bc_2 - B(\alpha_3c_1+\alpha_4c_2)) + 2r_2a^2\alpha_2\alpha_4b{c_2}^2e\mu\} \\
+(c_1-c_2)\{(a-e)(\alpha_2{}^2ce^2\mu(a+r_2)(\alpha_4c_2-\mu) + ae\mu(\alpha_2e\mu(r_2b-B) - \mu_d r_2b(\alpha_3c_1-\mu)) \\
+\alpha_2e(\alpha_1ce\mu(\alpha_4c_2-\mu) - a\alpha_4c_2(2Be\mu - \mu_d\alpha_3cc_1)) + \mu_d e\mu(\alpha_1c(\alpha_3c_1+\alpha_4c_2) - Ba\mu)) \\
+\alpha_2ce(a+r_2)(\mu(\alpha_1e(\alpha_3c_1+\alpha_4c_2) + a\alpha_3c_1(\alpha_2e+2\mu_d)) - \mu_d(\alpha_3c_1(a\alpha_3c_1+e\mu) - \\
\mu(a-e))) + (\alpha_3c_1-\mu)(\mu_d a\alpha_3c_1e(ar_2b-\alpha_1c) + \alpha_1ce^2\mu(a\alpha_2+\alpha_1)) - ae^2\mu(r_2b-B) \\
(\alpha_3c_1(a\alpha_2+\alpha_1)+\alpha_1(\alpha_4c_2-\mu)) + \mu_d e\mu(a+e)(\alpha_2\alpha_4cc_2e + Ba\alpha_3c_1) + 2r_2a\alpha_2\alpha_4bc_2e^3\mu \\
+ae(\alpha_2\alpha_4c_2\mu(\mu_d ce + Br_2a) - \mu_d\alpha_3c_1(\alpha_1\alpha_4cc_2 - Ba\mu)) - \alpha_1e\mu(a\alpha_4c_2(e+r_2)(r_2b-B) \\
-ce(\alpha_1\alpha_4c_2 - r_2\alpha_2\mu))\} + (a-e)\{(r_2b-B)(\mu_d c_2\mu(r_2a(\alpha_3c_1-\mu) - \alpha_4c_1e(a+r_2)) + r_2\alpha_2e\mu^2 \\
(c_1e-ac_2)) + (\alpha_3c_1-\mu)(\mu_d acc_2(\alpha_3c_1+\alpha_4c_2) - \mu_d e\mu(r_2c_1(r_2b-B)+\alpha_1cc_2)) - \mu_d c\mu(c_2 \\
(a-e)(\mu_d\alpha_3c_1+\alpha_2e\mu) - \mu(\mu_d c_2(a+e) - \alpha_1e(c_1-c_2))) - \alpha_4{c_2}^2e\mu(\mu_d c(a\alpha_2+\mu_d+\alpha_1) \\
+Br_2a\alpha_2)\} - \mu_d ae(a+r_2)(B\alpha_3{c_1}^2(\alpha_3c_1+\alpha_4c_2) + \alpha_2\alpha_4c{c_2}^2\mu) + \mu_d r_2a\alpha_3b{c_1}^2(r_2e(\alpha_3c_1 \\
+\alpha_4c_2) - ac_2(r_2\alpha_3-\alpha_4e)) + r_2\alpha_2be\mu(a^2c_1c_2(r_2(\alpha_3-\alpha_4)-2\alpha_4e) + r_2e(\alpha_4c_2(c_1e-2ac_2) \\
-a\alpha_3{c_1}^2)) + \mu_d\alpha_2cc_2e\mu(a^2c_1(\alpha_3+\alpha_4) + r_2\alpha_4c_2e) + (ac_2-c_1e)\{(r_2b-B)(r_2\mu(\mu_d a\alpha_3c_1 \\
-\alpha_1e\mu)) + r_2\alpha_1e\mu(\alpha_3c_1+\alpha_4c_2)(r_2b-B) - Br_2\alpha_2e\mu(a\alpha_3c_1 - \alpha_4c_2e)\}
\end{array}\right]}{\left(e\mu + a\left(\alpha_3\left(c_1-c_2\right)-\mu\right)\right)\left(\begin{array}{l}
e\left(c_1\alpha_3+c_2\alpha_4-\mu\right) \\
\left(\alpha_4\left(c_1\left(a\alpha_2+\alpha_1\right)-c_2\left(a\alpha_2-\mu_d+\alpha_1\right)\right)+\mu\left(a\alpha_2-\mu_d+\alpha_1\right)\right) \\
-\alpha_2e^2\left(c_2\alpha_4-\mu\right)\left(\mu+\alpha_4\left(c_1-c_2\right)\right)-\mu_d a\left(c_1\alpha_3+c_2\alpha_4-\mu\right)^2
\end{array}\right)}$$

where,

$$r_2 = \text{Root of} \begin{pmatrix} \left(b\left(\alpha_3 c_1 - \mu\right) + \alpha_4 b c_2\right) Z^3 + \left(\left(\alpha_3 c_1 - \mu\right)\left(ab - \alpha_2 c - B\right) + be\left(\alpha_4 c_2 - \mu\right)\right. \\ \left. + c_2\left(\alpha_4\left(ab - B\right) + \alpha_2 \alpha_3 c\right)\right) Z^2 + \left(e\left(\left(ab - B\right)\left(\alpha_4 c_2 - \mu\right) + \alpha_2 c\mu\right)\right. \\ \left. + c_2\left(\alpha_3 c\left(a\alpha_2 - \mu_d\right) + \alpha_1 \alpha_3 c - Ba\alpha_4\right)\left(\alpha_3 c_1 - \mu\right)\left(a\left(\alpha_2 c + B\right) + \alpha_1 c\right)\right) Z \\ + c\left(e\mu\left(a\alpha_2 + \alpha_1\right) - \mu_d a\alpha_3 c_2\right) - Bae\left(\alpha_4 c_2 - \mu\right) \end{pmatrix}$$

Now, we evaluate the basic reproduction number known as threshold R_0 using next generation matrix method (Diekmann *et al.*, 2009).

Let $X' = (S_M, T_B, M)'$ and $X' = \dfrac{dX}{dt} = F\left(X\right) - V\left(X\right)$ where, $F(X)$ denotes the rate of arrival of new individual in the compartment and $V(X)$ denotes the rate of transmission of tuberculosis who procures medication among smokers which are given by

$$F\left(X\right) = \begin{bmatrix} \left(\alpha_1 S_M T_B / a + S_M\right) + \alpha_2 S_M T_B \\ \left(c_1 \alpha_3 S_M M / e + S_M\right) + \left(c_2 \alpha_4 T_B M / c + T_B\right) \\ 0 \end{bmatrix} \text{ and}$$

$$V\left(X\right) = \begin{bmatrix} \left(\alpha_4 T_B M / c + T_B\right) + \mu_d T_B \\ \mu M \\ -BS_M + \left(\alpha_1 S_M T_B / a + S_M\right) + \alpha_2 S_M T_B + \left(\alpha_3 S_M M / e + S_M\right) + bS_M{}^2 \end{bmatrix}$$

Now, $DF(X_0) = \begin{bmatrix} f & 0 \\ 0 & 0 \end{bmatrix}$ and $DV(X_0) = \begin{bmatrix} v & 0 \\ J_1 & J_2 \end{bmatrix}$, where f and v are 3×3 matrices defined as $f = \left[\dfrac{\partial F_i(\mathrm{X}_0)}{\partial \mathrm{X}_j}\right]$ and $v = \left[\dfrac{\partial V_i(\mathrm{X}_0)}{\partial \mathrm{X}_j}\right]$. Finding f and v, we get

$$f=\begin{bmatrix} \left(\alpha_1 S_M/a+S_M\right)+\alpha_2 S_M & 0 & \begin{pmatrix}\left(\alpha_1 T_B/a+S_M\right)\\ -\left(\alpha_1 S_M T_B/\left(a+S_M\right)^2\right)+\alpha_2 T_B\end{pmatrix} \\ \begin{pmatrix}\left(c_2\alpha_4 M/c+T_B\right)\\ -\left(c_2\alpha_4 T_B M/\left(c+T_B\right)^2\right)\end{pmatrix} & \begin{pmatrix}\left(c_1\alpha_3 S_M/e+S_M\right)\\ +\left(c_2\alpha_4 T_B/c+T_B\right)\end{pmatrix} & \begin{pmatrix}\left(c_1\alpha_3 M/e+S_M\right)\\ -\left(c_1\alpha_3 S_M M/\left(e+S_M\right)^2\right)\end{pmatrix} \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$v=\begin{bmatrix} \begin{pmatrix}\mu_d+\left(\alpha_4 M/c+T_B\right)\\ -\left(\alpha_4 T_B M/\left(c+T_B\right)^2\right)\end{pmatrix} & \left(\alpha_4 T_B/c+T_B\right) & 0 \\ 0 & \mu & 0 \\ \left(\alpha_1 S_M/a+S_M\right)+\alpha_2 S_M & \left(\alpha_3 S_M/e+S_M\right) & \begin{pmatrix}-B+\left(\alpha_1 T_B/a+S_M\right)-\left(\alpha_1 S_M T_B/\left(a+S_M\right)^2\right)\\ +\alpha_2 T_B+\left(\alpha_3 M/e+S_M\right)-\left(\alpha_3 S_M M/\left(e+S_M\right)^2\right)\\ +2bS_M\end{pmatrix} \end{bmatrix}$$

Here, v is non-singular matrix.

For this model, by using matrix fv^{-1} the basic reproduction (threshold) R_0 at Y_0 is obtained

$$R_0=\frac{B\mu\left(eb+B\right)\left(\alpha_1 b+\alpha_2\left(ab+B\right)\right)+c_1\alpha_3 Bb\mu_d\left(ab+B\right)}{b\mu_d\mu\left(ab+B\right)\left(eb+B\right)} \tag{3}$$

STABILITY

In this section, local as well as global stability is analysed for every equilibrium point.

Local Stability

In this section, for all equilibrium points local stability is discussed.

Theorem 3.1.1 (Stability at Y_0): $Y_0 = \left(\frac{B}{b}, 0, 0\right)$ is locally asymptotically stable if following conditions are satisfied:

i) $c_1\alpha_3 B<\mu(eb+B)$ and

ii) $(ab+B)(\mu_d b - B\alpha_2)>\alpha_1 Bb$

Proof: The Jacobian matrix J_0 at Y_0 is given by

$$J_0 = \begin{bmatrix} -B & -\left(\alpha_1 B/b\left(a+B/b\right)\right)-\alpha_2 B/b & -\left(\alpha_3 B/b\left(e+B/b\right)\right) \\ 0 & \left(\alpha_1 B/b\left(a+B/b\right)\right)+\left(\alpha_2 B/b\right)-\mu_d & 0 \\ 0 & 0 & \left(c_1\alpha_3 B/b\left(e+B/b\right)\right)-\mu \end{bmatrix}$$

The eigenvalues of Jacobian J_0 are $\lambda_1=-\mu<0$,

$$\lambda_2 = \frac{c_1\alpha_3 B - be\mu - B\mu}{eb+B} < 0 \text{ if } c_1\alpha_3 B < \mu\left(eb+B\right)$$

and

$$\lambda_3 = \frac{\alpha_1 Bb - \left(ab+B\right)\left(\mu_d b - B\alpha_2\right)}{b\left(ab+B\right)} < 0 \text{ if } \left(ab+B\right)\left(\mu_d b - B\alpha_2\right) > \alpha_1 Bb\,.$$

Therefore, the equilibrium point is locally asymptomatically stable if

i) $c_1\alpha_3 B<\mu(eb+B)$

ii) $(ab+B)(\mu_d b - B\alpha_2)>\alpha_1 Bb$

Theorem 3.1.2 (Stability at Y_1): $Y_1 = \left(\frac{e\mu}{c_1\alpha_3-\mu}, 0, \frac{ec_1\left(B\left(c_1\alpha_3-\mu\right)-be\mu\right)}{\left(c_1\alpha_3-\mu\right)^2}\right)$ is locally asymptotically stable if $x_1,x_4<0$.

Proof: The Jacobian matrix J_1 at Y_1 is given by

$$J_1 = \begin{bmatrix} \frac{\mu\left(c_1\alpha_3\left(-be+B\right)-\mu\left(be+B\right)\right)}{\alpha_3 c_1\left(c_1\alpha_3-\mu\right)} & -\frac{e\left(\left(\alpha_2 e-\alpha_1-a\alpha_2\right)\mu+c_1\alpha_3\left(a\alpha_2+\alpha_1\right)\right)\mu}{\left(c_1\alpha_3-\mu\right)\left(\left(e-a\right)\mu+a\alpha_3 c_1\right)} & -\frac{\mu}{c_1} \\ 0 & \begin{pmatrix} \frac{e\mu\left(\left(\alpha_2 e-\alpha_1-a\alpha_2\right)\mu+c_1\alpha_3\left(a\alpha_2+\alpha_1\right)\right)}{\left(c_1\alpha_3-\mu\right)\left(\left(e-a\right)\mu+a\alpha_3 c_1\right)} \\ -\frac{\alpha_4 e c_1\left(B\left(c_1\alpha_3-\mu\right)-be\mu\right)}{\left(c_1\alpha_3-\mu\right)^2 c}-\mu_d \end{pmatrix} & 0 \\ \frac{c_1\alpha_3 B-\mu\left(eb+B\right)}{\alpha_3} & \frac{c_1\left(c_1\alpha_3 B-\mu\left(eb+B\right)\right)ec_2\alpha_4}{\left(c_1\alpha_3-\mu\right)^2 c} & 0 \end{bmatrix}$$

Let

$$\frac{\mu\left(c_1\alpha_3\left(-be+B\right)-\mu\left(be+B\right)\right)}{\alpha_3 c_1\left(c_1\alpha_3-\mu\right)}=x_1, \frac{c_1\alpha_3 B-\mu\left(eb+B\right)}{\alpha_3}=x_2,$$

$$\frac{e\left(\left(\alpha_2 e-\alpha_1-a\alpha_2\right)\mu+c_1\alpha_3\left(a\alpha_2+\alpha_1\right)\right)\mu}{\left(c_1\alpha_3-\mu\right)\left(\left(e-a\right)\mu+a\alpha_3 c_1\right)}=x_3,$$

$$\frac{e\mu\left(\left(\alpha_2 e-\alpha_1-a\alpha_2\right)\mu+c_1\alpha_3\left(a\alpha_2+\alpha_1\right)\right)}{\left(c_1\alpha_3-\mu\right)\left(\left(e-a\right)\mu+a\alpha_3 c_1\right)}-\frac{\alpha_4 e c_1\left(B\left(c_1\alpha_3-\mu\right)-be\mu\right)}{\left(c_1\alpha_3-\mu\right)^2 c}-\mu_d=x_4,$$

$$\frac{c_1\left(c_1\alpha_3 B-\mu\left(eb+B\right)\right)ec_2\alpha_4}{\left(c_1\alpha_3-\mu\right)^2 c}=x_5$$

and $\frac{\mu}{c_1}=x_6$.

The characteristic equation of Jacobian matrix J_0 is $\lambda^3+a_1\lambda^2+a_2\lambda+a_3=0$ where a_1= -x_1–x_4, a_2= x_1x_4+x_2x_6 and a_3= -$x_2x_4x_6$.

Here equilibrium point Y_1 is locally asymptotically stable (Routh, 1877), if x_1,x_4<0.

Theorem 3.1.3 (Stability at Y_2): $Y_2=\left(r_1, \frac{\left(a+r_1\right)\left(B\alpha_2-b\mu_d\right)+r_1\alpha_1 b}{\alpha_2\left(\alpha_1+\alpha_2\left(a+r_1\right)\right)}, 0\right)$ where

$$r_1 = RootOf\left(\alpha_2 Z^2 + (a\alpha_2 - \mu_d + \alpha_1) Z - \mu_d a\right)$$

is locally asymptotically stable if following conditions are satisfied:

i) $x_{14} = \frac{c_1\alpha_3 r_1}{e + r_1} + \frac{c_2\alpha_4 x_7}{c + x_7} - \mu < 0 \Rightarrow \frac{c_1\alpha_3 r_1}{e + r_1} + \frac{c_2\alpha_4 x_7}{c + x_7} < \mu$ and

ii) $Br_1\left(\frac{\alpha_1}{a + r_1} + \alpha_2\right) + x_1\mu_d\left(\frac{a\alpha_1}{(a + r_1)^2} + \alpha_2\right) + 2br_1\mu_d < B\mu_d + 2br_1^2\left(\frac{\alpha_1}{a + r_1} + \alpha_2\right)$

Proof: Let $\frac{(a + r_1)(B\alpha_2 - b\mu_d) + r_1\alpha_1 b}{\alpha_2(\alpha_1 + \alpha_2(a + r_1))} = x_7$. The Jacobian matrix J_2 at Y_2 is given by

$$J_2 = \begin{bmatrix} x_8 & -x_{10} & -x_{12} \\ x_9 & x_{11} & -x_{13} \\ 0 & 0 & x_{14} \end{bmatrix}$$

where

$$x_8 = B - \frac{a\alpha_1 x_7}{(a + r_1)^2} - \alpha_2 x_7 - 2br_1, x_9 = \frac{a\alpha_1 x_7}{(a + r_1)^2} + \alpha_2 x_7, x_{10} = \frac{\alpha_1 r_1}{a + r_1} + \alpha_2 r_1,$$

$$x_{11} = \frac{\alpha_1 r_1}{a + r_1} + \alpha_2 r_1 - \mu_d, x_{12} = \frac{\alpha_3 r_1}{e + r_1}, x_{13} = \frac{\alpha_4 x_7}{c + x_7},$$

and $x_{14} = \frac{c_1\alpha_3 r_1}{e + r_1} + \frac{c_2\alpha_4 x_7}{c + x_7} - \mu$

Jacobian J_2 has eigenvalues, $\lambda_1 = x_{14}$,

$$\lambda_2 = \frac{x_8 + x_{11}}{2} + \frac{\sqrt{(x_8 - x_{11})^2 + 4x_9 x_{10}}}{2} \text{ and}$$

$$\lambda_3 = \frac{x_8 + x_{11}}{2} - \frac{\sqrt{(x_8 - x_{11})^2 + 4x_9 x_{10}}}{2}$$

Here, these eigenvalues are negative if

i) $x_{14} = \frac{c_1\alpha_3 r_1}{e+r_1} + \frac{c_2\alpha_4 x_7}{c+x_7} - \mu < 0 \Rightarrow \frac{c_1\alpha_3 r_1}{e+r_1} + \frac{c_2\alpha_4 x_7}{c+x_7} < \mu$ and

ii)

$$Br_1\left(\frac{\alpha_1}{a+r_1}+\alpha_2\right) + x_1\mu_d\left(\frac{a\alpha_1}{(a+r_1)^2}+\alpha_2\right) + 2br_1\mu_d < B\mu_d + 2br_1^2\left(\frac{\alpha_1}{a+r_1}+\alpha_2\right)$$

Therefore, equilibrium point Y_2 is locally asymptotically stable if both these conditions are satisfied.

Theorem 3.1.4 (Stability at Y^*): Endemic point Y^* is locally asymptotically stable if the following conditions hold:

i) $x_{15}, x_{19}, x_{23} < 0$ and

ii) $x_{16}x_{20}x_{21} > x_{17}x_{18}x_{22}$

Proof: We determine the local stability behavior of endemic equilibrium point Y^* by using the Jacobian matrix J^* which is given by

$$J^* = \begin{bmatrix} x_{15} & -x_{18} & -x_{21} \\ x_{16} & x_{19} & -x_{22} \\ x_{17} & x_{20} & x_{23} \end{bmatrix}$$

where,

$$x_{15} = B - \frac{a\alpha_1 T_B^*}{(a+S_M^*)^2} - \frac{e\alpha_3 M^*}{(e+S_M^*)^2} - \alpha_2 T_B^* - 2bS_M^*, x_{16} = \frac{a\alpha_1 T_B^*}{(a+S_M^*)^2} + \alpha_2 T_B^*, x_{17} = \frac{c_1 e\alpha_3 M^*}{(e+S_M^*)^2},$$

$$x_{18} = \frac{\alpha_1 S_M^*}{a+S_M^*} + \alpha_2 S_M^*, x_{19} = \alpha_2 S_M^* + \frac{\alpha_1 S_M^*}{a+S_M^*} - \frac{c\alpha_4 M^*}{(c+T_B^*)^2} - \mu_d, x_{20} = \frac{cc_2\alpha_4 M^*}{(c+T_B^*)^2}, x_{21} = \frac{\alpha_3 S_M^*}{e+S_M^*},$$

$$x_{22} = \frac{\alpha_4 T_B^*}{c+T_B^*}, x_{23} = \frac{c_1\alpha_3 S_M^*}{e+S_M^*} + \frac{c_2\alpha_4 T_B^*}{c+T_B^*} - \mu .$$

The corresponding characteristic equation of Jacobian matrix is

$$\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0$$

where,

$$b_1 = -x_{15} - x_{19} - x_{23},$$

$$b_2 = x_{16}x_{18} + x_{17}x_{21} + x_{19}x_{23} + x_{15}x_{19} + x_{20}x_{22} + x_{15}x_{23}$$

and

$$b_3 = x_{16}x_{20}x_{21} - x_{17}x_{18}x_{22} - x_{16}x_{18}x_{23} - x_{17}x_{19}x_{21} - x_{15}x_{19}x_{23} - x_{15}x_{20}x_{22}$$

The equilibrium point is locally asymptotically stable (Routh, 1877), if following conditions hold:

i) $x_{15}, x_{19}, x_{23} < 0$ and
ii) $x_{16}x_{20}x_{21} > x_{17}x_{18}x_{22}$

Global Stability

Now, global stability behavior is analyzed for each and every equilibrium points using Lyapunov's function and curl.

Theorem 3.2.1 (Stability at Y_0): The disease-free equilibrium point Y_0 is globally asymptotically stable.

Proof: Consider the Lyapunov's function $L_{1(t)}=S_{M(t)}+T_{B(t)}$ then,

$$L_1'(t) = S_M'(t) + T_B'(t)$$
$$= BS - \frac{\alpha_3 S_M M}{e + S_M} - bS^2 - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B$$

we get $\frac{dL_1}{dt} < 0$ whereas $\frac{dL_1}{dt} = 0$ only if $T_B, M=0$.

Therefore, Y_0 is globally asymptotically stable (LaSalle, 1976).

Theorem 3.2.2 (Stability at Y_1): The infection-free equilibrium point Y_1 is globally asymptotically stable if

(i) $c_1\alpha_3 < \min\left\{\mu, \left(1-\frac{e}{a}\right)\mu\right\}$ and

(ii) $c_2>1$

Proof: Consider the Lyapunov's function $L_2(t)=T_B(t)+M(t)$ then,

$$\begin{aligned} L_2'(t) &= T_B'(t) + M'(t) \\ &= \frac{\alpha_1 S_M T_B}{a+S_M} + \alpha_2 S_M T_B - \frac{\alpha_4 T_B M}{c+T_B} - \mu_d T_B + \frac{c_1\alpha_3 S_M M}{e+S_M} + \frac{c_2\alpha_4 T_B M}{c+T_B} - \mu M \\ &= e\mu T_B\left(\frac{\alpha_1(c_1\alpha_3-\mu)+\alpha_2(a(c_1\alpha_3-\mu)+e\mu)}{(c_1\alpha_3-\mu)(a(c_1\alpha_3-\mu)+e\mu)}\right) + \frac{ec_1\alpha_4 T_B(B(c_1\alpha_3-\mu)-be\mu)(c_2-1)}{(c+T_B)(c_1\alpha_3-\mu)^2} - \mu_d T_B \end{aligned}$$

we get $\frac{dL_2}{dt} < 0$ if

(i) $c_1\alpha_3 < \min\left\{\mu, \left(1-\frac{e}{a}\right)\mu\right\}$ and

(ii) $c_2>1$

whereas $\frac{dL_2}{dt} = 0$ only if $T_B=0$.

Therefore, Y_1 is globally asymptotically stable (LaSalle, 1976).

Theorem 3.2.3 (Stability at Y_2): The infection-free equilibrium point Y_2 is globally asymptotically stable if

(i) $B<br_1$ and

(ii) $(a+r_1)(B\alpha_2-b\mu_d)+r_1\alpha_1 b < r_1\alpha_2(\alpha_1+\alpha_2(a+r_1))$.

Proof: Consider the Lyapunov's function

$$L_3(t) = \frac{S_M^2(t)+T_B^2(t)+M^2(t)}{2}$$

then,

$$L_3'(t) = S_M(t)S_M'(t) + T_B(t)T_B'(t) + M(t)M'(t)$$
$$= S_M{}^2\left(B - bS_M\right) + \left(\frac{\alpha_1 S_M T_B}{a + S_M} + \alpha_2 S_M T_B\right)\left(T_B - S\right)$$
$$+ \frac{\alpha_3 S_M M}{e + S_M}\left(c_1 M - S_M\right) + \frac{\alpha_4 T_B M}{c + T_B}\left(c_2 M - T_B\right) - \mu M^2 - \mu_d T_B{}^2$$

substituting the values of Y_2, we get $\frac{dL_3}{dt} < 0$ if

(i) $B < br_1$ and
(ii) $\left(a + r_1\right)\left(B\alpha_2 - b\mu_d\right) + r_1\alpha_1 b < r_1\alpha_2\left(\alpha_1 + \alpha_2\left(a + r_1\right)\right)$.

Therefore, Y_2 is globally asymptotically stable (LaSalle, 1976).

Theorem 3.2.4 (Stability at Y^*): Consider a piecewise smooth vector field

$$g\left(S_M, T_B, M\right) = \left\{g_1\left(S_M, T_B, M\right), g_2\left(S_M, T_B, M\right), g_3\left(S_M, T_B, M\right)\right\}$$

on Λ^* that satisfies the condition $\left(curl\ g\right)\cdot\vec{n} < 0,\ g\cdot f = 0$ inside Λ^*, where f= (f_1, f_2, f_3) is a Lipschitz continuous field inside Λ^*, $\vec{n}$ is a normal vector to Λ^* and

$$curl\ g = \left(\frac{\partial g_3}{\partial T_B} - \frac{\partial g_2}{\partial M}\right)\hat{i} - \left(\frac{\partial g_3}{\partial S_M} - \frac{\partial g_1}{\partial M}\right)\hat{j} + \left(\frac{\partial g_2}{\partial S_M} - \frac{\partial g_1}{\partial T_B}\right)\hat{k}.$$

Then, the system of differential equations $S_M = f_1$, $T_B = f_2$, $M = f_3$ has no homoclinic loops, periodic solutions and oriented phase polygons inside Λ^* if $c_1, c_2 > 1$ (Awan *et al.*, 2017).

Proof: Suppose $\Lambda^* = \left\{\left(S_M, T_B, M\right) : S_M + T_B + M = 1, S_M > 0, T_B \geq 0, M \geq 0\right\}$.

Also, it can easily be proved that Λ^* is a subset of Λ, Λ^* is a positively invariant and endemic equilibrium Y^* belongs to Λ^*. Let f_1, f_2 and f_3 represents the right-hand side of equations in set of equations (1) respectively. Using $S_M + T_B + M = 1$ to write f_1, f_2 and f_3 in the equivalent forms, we get

$$f_1\left(S_M, T_B\right) = BS_M - \frac{\alpha_1 S_M T_B}{a + S_M} - \alpha_2 S_M T_B - \frac{\alpha_3 S_M M}{e + S_M} - bS_M{}^2$$

$$f_1\left(S_M, \mathrm{M}\right) = BS_M - S_M\left(1 - S_M - M\right)\left(\frac{\alpha_1}{a + S_M} + \alpha_2\right) - \frac{\alpha_3 S_M M}{e + S_M} - bS_M{}^2$$

$$f_2\left(S_M, T_B\right) = \frac{\alpha_1 S_M T_B}{a + S_M} + \alpha_2 S_M T_B - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B$$

$$= \frac{\alpha_1 S_M T_B}{a + S_M} + \alpha_2 S_M T_B - \frac{\alpha_4 T_B\left(1 - S_M - T_B\right)}{c + T_B} - \mu_d T_B$$

$$f_2\left(T_B, M\right) = \frac{\alpha_1 S_M T_B}{a + S_M} + \alpha_2 S_M T_B - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B$$

$$= \frac{\alpha_1 T_B\left(1 - T_B - M\right)}{a + \left(1 - T_B - M\right)} + \alpha_2 T_B\left(1 - T_B - M\right) - \frac{\alpha_4 T_B M}{c + T_B} - \mu_d T_B$$

$$f_3\left(S_M, M\right) = \frac{c_1 \alpha_3 S_M M}{e + S_M} + \frac{c_2 \alpha_4 M\left(1 - S_M - M\right)}{c + \left(1 - S_M - M\right)} - \mu M$$

$$f_3\left(T_B, M\right) = \frac{c_1 \alpha_3 M\left(1 - T_B - M\right)}{e + \left(1 - T_B - M\right)} + \frac{c_2 \alpha_4 T_B M}{c + T_B} - \mu M$$

Suppose, g=(g_1,g_2,g_3) be a vector field such that

$$g_1 = \frac{f_3\left(S_M, M\right)}{S_M M} - \frac{f_2\left(S_M, T_B\right)}{S_M T_B}$$

$$= \frac{c_1 \alpha_3}{e + S_M} + \frac{c_2 \alpha_4\left(1 - S_M - M\right)}{S_M\left(c + \left(1 - S_M - M\right)\right)} - \frac{\mu}{S_M} - \frac{\alpha_1}{a + S_M} - \alpha_2 + \frac{\alpha_4\left(1 - S_M - T_B\right)}{S_M\left(c + T_B\right)} + \frac{\mu_d}{S_M}$$

$$g_2 = \frac{f_1(S_M, T_B)}{S_M T_B} - \frac{f_3(T_B, M)}{T_B M}$$
$$= \frac{B}{T_B} - \frac{\alpha_1}{a + S_M} - \alpha_2 - \frac{\alpha_3 M}{T_B(e + S_M)} - \frac{bS_M}{T_B} - \frac{c_1\alpha_3(1 - T_B - M)}{T_B(e + (1 - T_B - M))} - \frac{c_2\alpha_4}{c + T_B} + \frac{\mu}{T_B}$$

$$g_3 = \frac{f_2(T_B, M)}{T_B M} - \frac{f_1(S_M, M)}{S_M M}$$
$$= \frac{(1 - T_B - M)}{M}\left(\frac{\alpha_1}{(a + (1 - T_B - M))} + \alpha_2\right) - \frac{\alpha_4}{c + T_B} - \frac{1}{M}(\mu_d + B - bS_M)$$
$$+ \frac{\alpha_3}{e + S_M} + \frac{(1 - S_M - M)}{M}\left(\frac{\alpha_1}{a + S_M} + \alpha_2\right)$$

As, the alternate forms of f_1, f_2 and f_3 are equivalent in Λ^*, so

$$g \cdot f = g_1 f_1 + g_2 f_2 + g_3 f_3 = 0 .$$

Now, using normal vector $\vec{n}$, where $\vec{n} = (1,1,1)$ to Λ^*, we have

$$(curl\ g) \cdot \vec{n} = \frac{\alpha_1 - b(a + S_M)^2}{M(a + S_M)^2} + \frac{\alpha_4(c(1 - c_2) + 1)}{S_M(c + T_B)^2}$$
$$- \frac{b}{T_B} + \frac{\alpha_3(e(1 - c_1) + 1)}{T_B(e + S_M)^2} < 0 \text{ if } c_1, c_2 > 1$$

So, the system (1) has no homoclinic loops, periodic solutions and oriented phase polygons in the interior of Λ^* if $c_1, c_2 > 1$. $\therefore Y^*$ is globally asymptotically stable in the interior of Λ^*.

Numerical Simulations

In this section, simulation is carried out to validate the data and their results are interpreted which helps to know the behaviour of individuals in each compartment.

Figure 2. Transmission pattern of tuberculosis among smokers

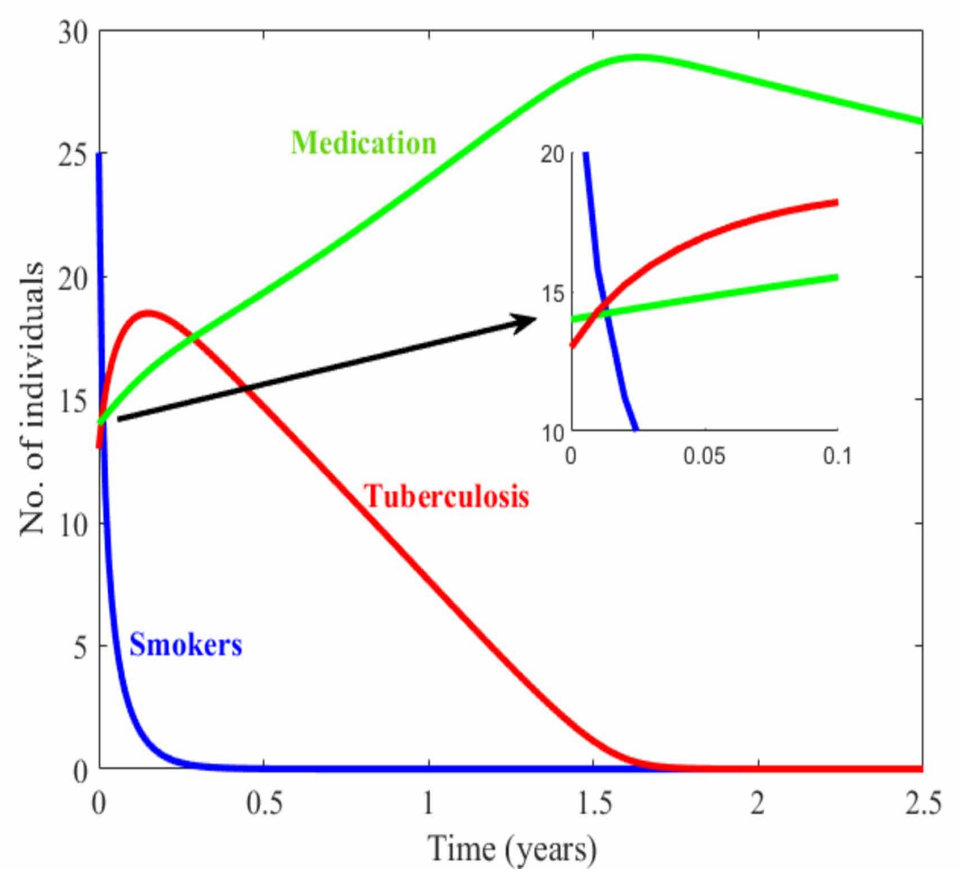
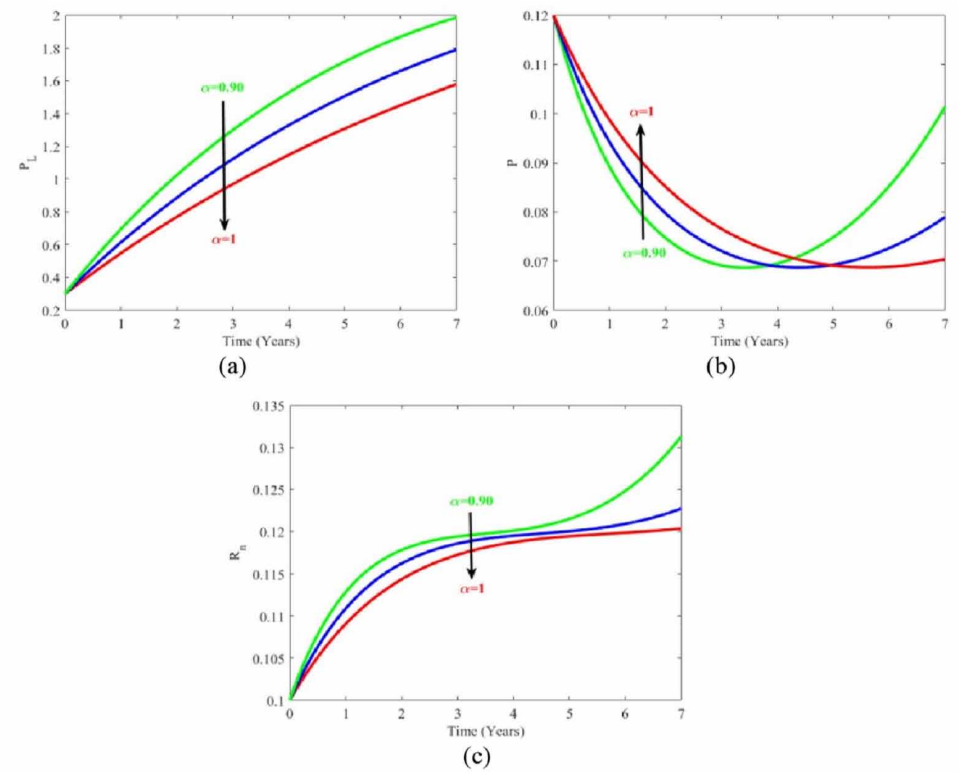


Figure 2 shows the transmission pattern of smokers suffering from tuberculosis. Approximate 15 smokers who have symptoms of tuberculosis procures medication after approximate 0.02 years. But sometimes they again get infected due to carelessness or ignorance while following proper and exact procedures carefully while procuring medication. Also, using our assumed data, the above figure shows that at approximately 0.3 years, around 18 individuals again suffer from tuberculosis due to such sloppiness/negligence while completing prescribed course. Individuals starts getting cured if they complete prescribed course with utmost precision for given time interval.

Figure 3 shows the effect of rates α_2 (the rate where smokers suffers from tuberculosis) on tuberculosis patients, α_3 (the rate from smokers who procures medication) and α_4 (the rate from tuberculosis patients procuring medication) on medication. In Figure 3(a), increase in α_2 by 20% results in increase of individuals

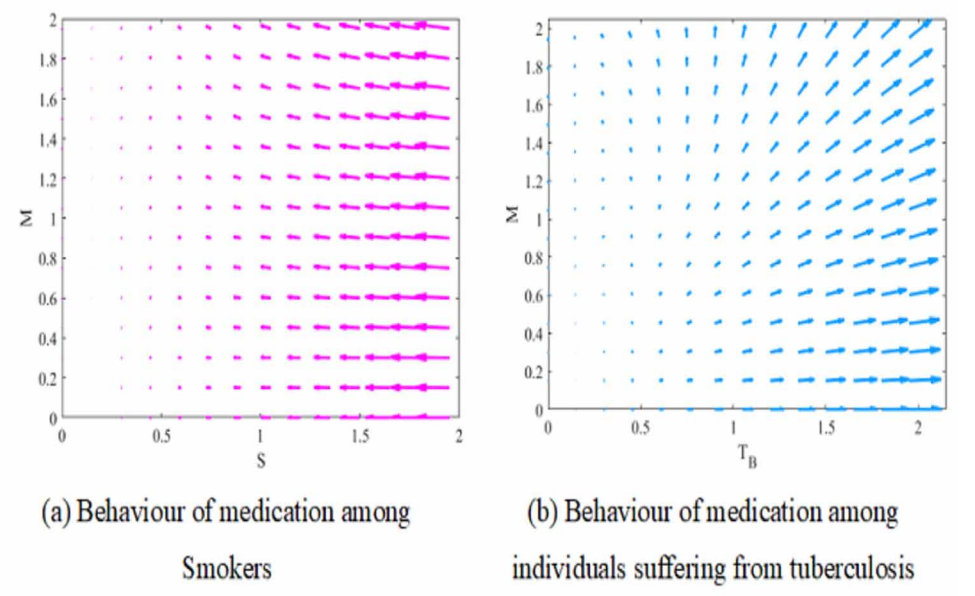
suffering from tuberculosis by approximately 11%. Figure 3(b) displays the impact of change in α_3 on medication. If it is increased by 20% then it results in increase in medication by 6%. Even change in α_4 by 20% as shown in Figure 3(c), will affect the medical rate which increases by 16%.

Figure 3.



Here, the quiver graphs shown in Figure 4 displays the behaviour of medication among smokers and individuals suffering from tuberculosis. Figure 4(a) shows the intensity of smokers moving towards medication which connotes that it decreases as they start procuring medication in terms of joining rehabilitation centre to quit smoking. Also, Figure 4(b) shows that intensity of individuals suffering from tuberculosis increases if they don't complete prescribed course (medication) for particular interval of time.

Figure 4.



(a) Behaviour of medication among Smokers

(b) Behaviour of medication among individuals suffering from tuberculosis

In pie chart shown in Figure 5, it is observed that due to 16% smokers, 45% individual suffers from tuberculosis and overall 39% individual procures proper medication.

Figure 5. Percentage of medication due to Tuberculosis among smokers

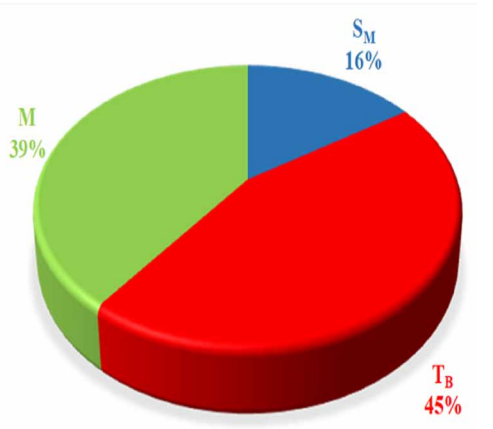
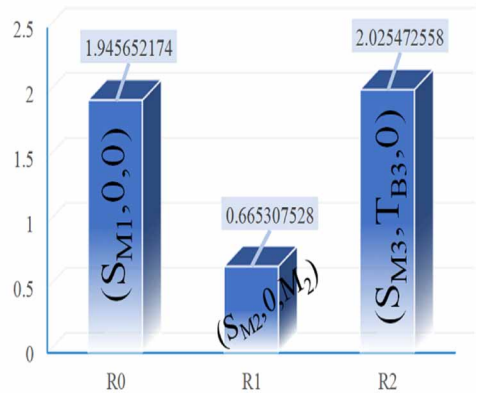


Figure 6. Graphical representation of basic reproduction number



The graphical representation of basic reproduction number (threshold) R_0, R_1 and R_2 for equilibrium point Y_0, Y_1 and Y_2 respectively are shown in Figure 6. Here it shows that the value is minimum where smokers quits smoking or procures medication to curtail the spread of infection. Whereas the maximum effect is observed where no medication is opted. In this case an infected individual affects approximately 2 others individuals.

CONCLUSION

This proposed chapter comprises of a mathematical model to study the flow of individuals suffering from tuberculosis due to smoking. A system of nonlinear differential equation is formulated to analyse the spread of tuberculosis infection smokers who either quits smoking due to awareness of situation or procures medication. Here, the basic reproduction number (R_0) is 1.94 which connotes that a smoker suffering from tuberculosis spreads this infection to approximately 2 other individuals and also that the issue is epidemic in nature. Also, stability analysis is carried out using Lyapunov function and curl. Along with it, to justify the results numerical simulation validates that with the increase in the rate of individuals suffering from tuberculosis due to smoking will increase the mortality rate all over the world. Also, quiver graphs explain the flow or behavior of individuals in respective compartments. Constructive modifications in lifestyle can reduce the overall occurrence of tuberculosis. With recent advancements in novel drug delivery systems, the complexities of various pulmonary infections can be resolved.

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Chapter 4
Dynamic Analysis of the Effect of Quitting Smoking Applications on Smoking Cessation

A. George Maria Selvam
https://orcid.org/0000-0003-2004-3537
Sacred Heart College (Autonomous), India

Mary Jacintha
Sacred Heart College (Autonomous), India

ABSTRACT

In this chapter, the authors considered a smoking cessation model formulated with a non-linear system of differential equations and obtained the continuous fractional order model and through discretization its discrete form to study the effectiveness of quitting smoking applications in giving up smoking. The existence of smoking free equilibria and smoking present equilibria are discussed, and the dynamical analysis of these two equilibria is put forward with the assistance of the smoking generation number. The numerical simulations aided by time series, phase portraits, and bifurcation diagrams confirm the results that are obtained analytically.

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INTRODUCTION

Smoking is hazardous to healthiness, as cigarettes include substances like nicotine and carbon monoxide. When the ingredients of a cigarette are burnt, essentially 7,000 chemicals are generated, several of those chemicals are toxic and nearly 69 of them are connected to cancer. Smoking leads to a variety of unending bodily complications which leaves lasting effects on the body systems. Smoking intensifies the risk of a variety of problems over the years, but some of the bodily effects are instantaneous. Stained teeth, bad breath, coughing and high blood pressure, are the foremost consequences of short-term smoking. Cancer of the mouth, throat, lung, cardiovascular diseases, stomach ulcers which are life-threatening are the result of long-term smoking.

World Health Organization (WHO), reports that smoking presently accounts for above five million fatalities in the world each year. It also predicts that deaths due to smoking could reach to ten million by 2020, resulting in almost 18 percent of all demises in the technologically advanced world. The mortality rate for smokers is thrice that of non-smokers. Smoking is the most common "preventable cause of death" around the world. Smoking is injurious, not only for smokers but also to those who share their environment. The WHO claims that each year above six million deaths are the consequence of non-smokers being subjected to second-hand smoke, which is risky particularly to the unborn babies and children.

In general, adults mostly begin to smoke at the age of adolescence, a time in life of great vulnerability to social influences, where many attitudes change which also comprises the attitude concerning smoking. One of the components in cigarettes is a mood-altering drug called nicotine, which reaches the brain in mere seconds and provides an energized feeling for a while. But as the effect decreases, tiredness sets in with a craving for more. Nicotine is exceedingly habit-forming, thus making smoking so tough to quit. The governments around the globe are promoting policies and agendas to control smoking, and to this end, academic and medical world is contributing its efforts through development and study of various mathematical models and psychological studies.

Mathematical models play a vital role in understanding the spread and control of a disease. In 2000, a simplistic mathematical model was formulated for giving up smoking, wherein the total constant population was sub divided as potential smokers (P), non-smokers who may start smoking in the future, smokers (S), and persons (former smokers) who would quit smoking permanently (Q) (Castillo-Garsow et al., 2000).

The smoking cessation models have generated much interest (Sharomi et al., 2008), particularly, the qualitative behaviour of giving up smoking model (Zaman, 2011), smoking cessation model with media campaigns and bifurcation analysis (Sharma

et al., 2015), a mathematical model with a saturated incidence rate to explore the effect of controlling smoking (Pang et al, 2015), global dynamics of mathematical model on smoking with media campaigns (Vinay et al., 2015),the stability analysis on dynamics of giving up smoking model with education campaign (Piyarat et al., 2015), the study on qualitative and sensitivity analysis of the effect of electronic cigarettes on smoking cessation (Jae et al., 2018).

Derivatives of fractional order are an efficient tool to elucidate the dynamical behaviour of complex biomaterials and systems. The forte of these operators consists in its unique nonlocal characteristics. This property implies that the next stage of a model depends not only to its present stage but also to all of its past stages. Erturk et al. initiated derivatives of fractional into the giving up smoking model and studied it numerically (Erturk et al., 2012), Zeb et al. explored a fractional order giving up smoking model (Zeb et al., 2012), Khalid et al. expounded on the fractional mathematical model of giving up smoking (Khalid et al., 2016), Jagdev et al. introduced an innovative continuous fractional model for giving up smoking (Jagdev et al., 2017).

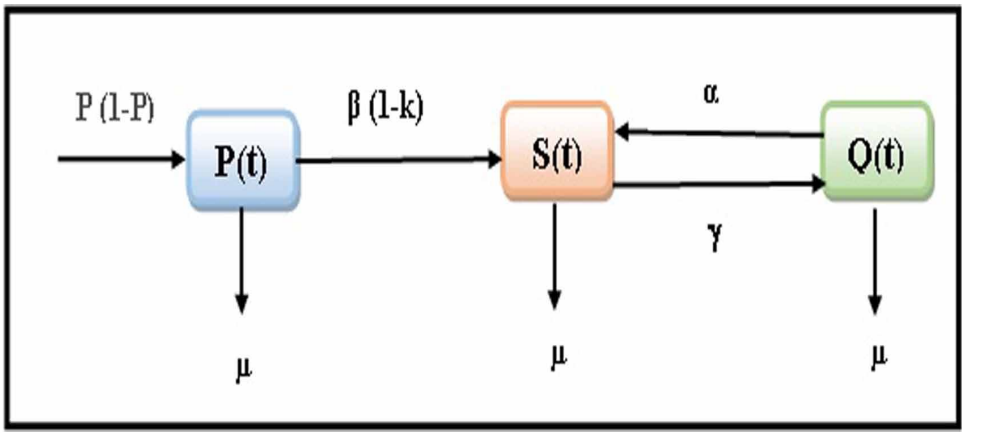
Finding active stratagems to support young adults in quitting smoking is a priority because of their excessive smoking rates and minimum utilisation of accessible cessation means. Considering their active presence in the mobile phone market, several researchers perceived mobile phone apps as a constructive way to reach young smokers. Presently over 500 smoking cessation apps are accessible which are met with eagerness confirmed by the significant number of downloads. Young adults essentially want cessation support via apps in comparison to other interventions. Based on this premise, authors intend to study the usefulness of quit smoking apps in smoking cessation. A mathematical model is proposed in keeping with (Piyarat et al., 2015) to study the addictive nature of smoking and the effectiveness of quit smoking apps in smoking cessation.

The chapter is further presented as follows: The mathematical model is formulated with a system of differential equations which are non-linear in section 1. The continuous fractional order model and its discrete version are presented in section 2. In Section 3, smoking free and smoking present equilibria along with the smoking generation number are obtained. The stability analysis of the smoking free and smoking present equilibrium point are undertaken in section 4. Numerical results and analysis are in section 5 while bifurcation diagrams are provided in section 6. The chapter ends with a brief conclusion.

FORMULATION OF THE MODEL

The conventional epidemiologic prototype of agent, host, vector and environment is an apt tool to analyse the relationship of varied effects on patterns of tobacco use in populations. Based on the epidemic models the present model is formulated, as person who has not smoked before, comes into contact with persons who smoke and due to their stimulus start smoking. Let *N(t)* represents the total population size at time t. The population *N(t)* is subdivided into potential smokers *P(t)*, smokers *S(t)* and quitters *Q(t)*.

Figure 1. Flow chart of the transmission of the smoking model



The dynamics of the smoking model is represented by the system of differential equations which are non-linear with three state variables as given below:

$$\frac{dP}{dt} = P(1-P) - \beta(1-k)PS - \mu P$$
$$\frac{dS}{dt} = \beta(1-k)PS + \alpha SQ - \gamma S - \mu S \quad (1)$$
$$\frac{dQ}{dt} = \gamma S - \alpha SQ - \mu Q$$

where μ represents natural mortality rate of human population,

β is the interaction rate between potential smokers and smokers,

α is the interaction rate between smokers and quitters who revert back to smoking,
k is the effectiveness of quit smoking mobile apps,
γ is the rate of quitting smoking,
N is the total population, with initial conditions given by $P(0) = P_0 \geq 0, S(0) = S_0 \geq 0$ and $Q(0) = Q_0 \geq 0$.

Model Analysis: Boundedness of Solution

Let $N=P+S+Q$, consider

$$\frac{dN}{dt} = \frac{dP}{dt} + \frac{dS}{dt} + \frac{dQ}{dt},$$

$$\frac{dN}{dT} = P(1-P) - \beta(1-k)PS - \mu P + \beta(1-k)PS + \alpha SQ - \gamma S - \mu S + \gamma S - \alpha SQ - \mu Q,$$

$$= P(1-P) - \mu P - \mu S - \mu Q,$$

$$= P(1-P) - \mu(P+S+Q),$$

$$\frac{dN}{dT} = P(1-P) - \mu N.$$

Thus, $\frac{dN}{dt} = 0$ if $\mu N = P(1-P)$, which is an indication that the population is constant (Zaman, 2011). As system (1) considers human population, the parameters and state variables for all $t \geq 0$ are non-negative.

And also, all feasible solutions of system (1) are bounded and enter the region

$$\Gamma = \left\{ (P,S,Q) \in \Re_+^3 \mid P+S+Q \leq N \right\}.$$

FRACTIONAL ORDER MODEL AND ITS DISCRETE VERSION

In recent years, researchers are drawn to fractional calculus as fractional derivatives are evolving as an powerful instrument to explicate the dynamical behaviour of various physical systems. Hence the fractional-order form of model (1) is given as

$$
\begin{aligned}
D_t^\sigma P &= P(1-P) - \beta(1-k)PS - \mu P, \\
D_t^\sigma S &= \beta(1-k)PS + \alpha SQ - \gamma\, S - \mu S, \\
D_t^\sigma Q &= \gamma S - \alpha\, SQ - \mu Q.
\end{aligned} \tag{2}
$$

where σ is the fractional order satisfying σ∈(0,1] and $D^\sigma = \frac{d^\sigma}{dt^\sigma}$ is in the sense of Caputo derivative (Caputo, 1967). The initial conditions of system (2) are $P(0) = P_0, S(0) = S_0, Q(0) = Q_0$. It is observed that richer dynamical behaviour is exhibited by the discretized system than its corresponding continuous fractional-order forms. Thus, applying the discretization method of piecewise constant arguments (Agarwal, 2013) to system (2) yields the fractional order discrete model

$$
\begin{aligned}
P_{n+1} &= P_n + \frac{h^\sigma}{\Gamma(1+\sigma)}\left[P_n(1-P_n) - \beta(1-k)P_nS_n - \mu P_n\right], \\
S_{n+1} &= S_n + \frac{h^\sigma}{\Gamma(1+\sigma)}\left[\beta(1-k)P_nS_n + \alpha S_nQ_n - \gamma\, S_n - \mu S_n\right], \\
Q_{n+1} &= Q_n + \frac{h^\sigma}{\Gamma(1+\sigma)}\left[\gamma S_n - \alpha\, S_nQ_n - \mu Q_n\right],
\end{aligned} \tag{3}
$$

where *h* is the step size.

EXISTENCES OF THE EQUILIBRIA AND SMOKING GENERATION NUMBER

Letting the right-hand side of the equations in system (1) to zero, two equilibrium points are obtained:

1. **Smoking free equilibrium point (E_0)**: As there are no smokers, by setting S=0 and Q=0 in system (1) leads to $E_0(P,S,Q) = (1-\mu, 0, 0)$.
2. 2. **Smoking present equilibrium point (E_1)**: For the case $P \neq 0, S \neq 0, Q \neq 0$,

$$
E_1\left(P^*, S^*, Q^*\right) = \left(1 - \beta(1-k)S^* - \mu, S^*, \frac{\gamma S^*}{\alpha S^* + \mu}\right) \text{with}
$$

$$B_1 S^{*2} + B_2 S^* + B_3 = 0,\ B_1 = \alpha\beta^2 (1-k)^2,\ B_2 = \mu\left[\alpha + \beta^2 (1-k)^2\right] - \alpha(\mu+\gamma)R_0,$$

$$B_3 = \mu(1-R_0)(\mu+\gamma), \text{ and } S^* = \frac{-B_2 \pm \sqrt{(B_2)^2 - 4B_1 B_3}}{2B_1}.$$

Here, for all parameter values, the coefficient B_1 is always positive. But the sign of B_2 depends on parameters while B_3 on R_0. Positive solutions do not exist when R_0=1 as B_3=0. If, R_0>1, then B_3<0 and $\sqrt{(B_2)^2 - 4B_1 B_3} > B_2$, so only one positive solution exists. If R_0<1, then B_1>0, B_2>0 and B_3>0, positive solutions do not exit. These results are summarized as follows.

Theorem 1: The smoking-free equilibrium point $E_0(P,S,Q) = (1-\mu,0,0)$ always exists for system (3). As for the existence of a smoking present equilibrium point (E_1), it can have three cases:

(i) if R_0<1, positive equilibrium point does not exist,
(ii) if R_0>1, one positive equilibrium point exists,
(iii) if R_0=1, positive equilibrium point does not exist.

Smoking Generation Number

The smoking generation number R_0 is obtained by calculating the spectral radius of the next generation matrix as in [Vanet al., 2008]. Let $X=(S,Q,P)^T$, so system (1) can be expressed as $X^{'} = F(X) - V(X)$, where

$$F(X) = \begin{bmatrix} \beta(1-k)PS \\ 0 \\ 0 \end{bmatrix} \text{ and } V(X) = \begin{bmatrix} \gamma S + \mu S - \alpha SQ \\ \alpha SQ + \mu Q - \gamma S \\ \beta(1-k)PS + \mu P - P(1-P) \end{bmatrix}.$$

By obtaining, the Jacobian of $F(X)$ and $V(X)$, given by $\frac{d}{dX}F(X) = F$ and $\frac{d}{dX}V(X) = V$,

$$F=\begin{pmatrix}\beta(1-k)S & \beta(1-k)P & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix} \text{ and}$$

$$V=\begin{pmatrix}0 & \gamma+\mu-\alpha S & -\alpha S\\ 0 & \alpha Q-\gamma & \alpha S+\mu\\ \beta(1-k)S+\mu+2P-1 & \beta(1-k)P & 0\end{pmatrix}.$$

At E_0=(1 – μ,0,0),

$$F=\begin{pmatrix}0 & \beta(1-k)(1-\mu) & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix} \text{ and } V=\begin{pmatrix}0 & \gamma+\mu & 0\\ 0 & -\gamma & \mu\\ (1-\mu) & \beta(1-k)(1-\mu) & 0\end{pmatrix}.$$

Hence, the next generation matrix

$$FV^{-1}=\frac{1}{\mu(1-\mu)(\gamma+\mu)}\begin{pmatrix}\beta(1-k)(1-\mu)^2\mu & 0 & 0\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}.$$

The spectral radius $\rho\left(FV^{-1}\right)=\frac{\beta(1-k)(1-\mu)}{(\gamma+\mu)}$, which is R_0, the smoking generation number.

STABILITY ANALYSIS

The Jacobian matrix of system (3) is

$$J(E_0)=\begin{pmatrix}1+A[1-2P-\beta(1-k)S-\mu] & -A\beta(1-k)P & 0\\ A\beta(1-k)S & 1+A[\beta(1-k)P+\alpha Q-(\mu+\gamma)] & A\alpha S\\ 0 & A[\gamma-\alpha Q] & 1-A[\mu+\alpha S]\end{pmatrix}$$

Stability Analysis of the smoking free equilibrium point $E_0(P,S,Q)$=(1 – μ,0,0).

Theorem 2: The smoking free equilibrium of system (3) at E_0, is local asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

Proof: The Jacobian matrix of system (3) assessed at $E_0=(1-\mu,0,0)$ is

$$J(E_0)=\begin{pmatrix} 1-A(1-\mu) & -A\beta(1-k)(1-\mu) & 0 \\ 0 & 1+A\left[\beta(1-k)(1-\mu)-(\mu+\gamma)\right] & 0 \\ 0 & A\gamma & 1-A\mu \end{pmatrix}, \text{ where}$$

$$A=\frac{h^{\sigma}}{\Gamma(1+\sigma)}.$$

The eigen values of $J(E_0)$ are found by solving $\det(J(E_0)-\lambda I)=0$. Thus, the eigen values are

$$\lambda_1=1-A(1-\mu),\lambda_2=1+A\left[\beta(1-k)(1-\mu)-(\mu+\gamma)\right],\lambda_3=1-A\mu.$$

Theorem 3: The smokers present equilibrium point of system (3) at E_1, is local asymptotically stable if $R_0>1$ and unstable if $R_0<1$.

Proof: The Jacobian matrix of system (3) assessed at $E_1=(P^*,S^*,Q^*)$ is

$$J(E_1)=\begin{pmatrix} a_{11} & a_{12} & 0 \\ a_{21} & a_{22} & 0 \\ 0 & a_{32} & a_{33} \end{pmatrix} \text{ and } J(E_1)-\lambda I=\begin{pmatrix} a_{11}-\lambda & a_{12} & 0 \\ a_{21} & a_{22}-\lambda & 0 \\ 0 & a_{32} & a_{33}-\lambda \end{pmatrix}.$$

The characteristic equation is given by $\lambda^3-d_1\lambda^2+d_2\lambda-d_3=0$, where

$$d_1=-(a_{11}+a_{22}+a_{33}),d_2=a_{22}a_{33}+a_{11}a_{33}+a_{22}a_{11}-a_{12}a_{21},d_3=a_{33}(a_{12}a_{21}-a_{11}a_{22}),$$

$$a_{11}=1-A\left[(1-\mu)-\beta(1-k)S^*\right],a_{12}=-A\beta(1-k)(1-\mu)+A\beta^2(1-k)^2S^*,a_{21}=A\beta(1-k)S^*,$$

$$a_{22}=1+A\left[\beta(1-k)(1-\mu)-\beta^2(1-k)^2S^*+\frac{\alpha\gamma S^*}{\alpha S+\mu}-(\mu+\gamma)\right],a_{32}=A\gamma\left[\frac{\mu}{\alpha S^*+\mu}\right],$$

$$a_{33}=1-A\left[\alpha S^*+\mu\right].$$

The three eigen values of $\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3 = 0$ will have negative real parts if they satisfy the Routh-Hurwitz criteria: (a) $d_1>0$, (b) $d_3>0$ and (c) $d_1d_3–d_3>0$.

NUMERICAL RESULTS AND ANALYSIS

Here, taking into account R_0 termed as smoking generation number which calculates the average number of new smokers generated by an individual smoker in the potential smoker's population. If $R_0<1$ then the total number of smokers in the population can be reduced to zero which implies that a small inflow of smokers into the population will not generate large number of smokers on the contrary for $R_0>1$ the total number of smokers in the population rises in other words a small influx of smokers into the population will give rise to large number of smokers.

In this segment, some numerical solutions of system (3) are pictorialized for varied parametric values comparing it with the qualitative results.

Example 1: Stability Analysis of the smoking free equilibrium point $E_0(1–\mu,0,0) = (0.9700,0,0)$:

The following parametric values are utilized

$\beta = 0.15$,k = 0.4, $\mu = 0.03$, $\alpha = 0.22$,$\gamma = 0.25$,$\sigma = 0.5$, h = 3.3

with initial conditions P(0)=0.95, S(0)=0.05, Q(0)=0.0 *such that* P+S+Q=1. The Jacobian matrix at E_0 of system (3) is

$$J\left(0.9700,0,0\right) = \begin{pmatrix} -0.9883 & -0.1789 & 0 \\ 0 & 0.6050 & 0 \\ 0 & 0.5125 & 0.9385 \end{pmatrix}.$$

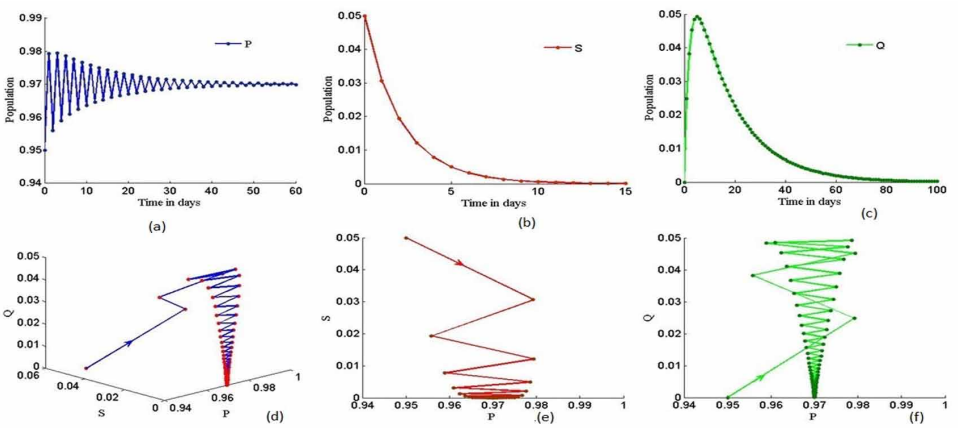
The eigen values and the smoking generation number are

$$\lambda_1 = -0.9883, \lambda_2 = 0.6050, \lambda_3 = 0.9385,\ |\lambda_1| = 0.9883<1, |\lambda_2| = 0.6050<1, |\lambda_3| = 0.9385<1,$$

and $R_0=0.3118<1$. Since all of the eigen values are less than one and smoking generation number is less than one, the smoking free equilibrium point will be local asymptotically stable as seen in *Figure 2*.

For, R_0=0.3118<1, Figure 2(a) illustrates that the number of potential smokers' ascents and reaches a stable point. In Figure 2(b) the number of the smokers decrease and tends to zero. In Figure 2(c) quitters increase at first and later tend to zero. From Figure 2(d), 2(e) and 2(f) for the given initial conditions, the solution curves approach the equilibrium E_0, when R_0<1, hence, system (3) is local asymptotically stable about E_0 with respect to given set of values.

Figure 2. Time series of system (3) with (a) Potential smokers (b) Smokers (c) Quitters and Phase portraits in (d) (P, S, Q) plane (e) (P, S) plane (f) (P, Q) plane



Example 2: The following parametric values are considered:

$$\beta = 0.15,\ \mathrm{k} = 0.4,\ \mu = 0.03,\ \alpha = 0.22,\ \gamma = 0.25, h = 2.1,$$

varying the fractional order parameter σ=0.5, σ=0.7 and σ=0.9 with initial conditions P(0)=0.95, S(0)=0.05, Q(0)=0.0.

In Figure 3(a), for σ=0.5 it is observed that the number of potential smokers increase and attain stability quickly in comparison to σ=0.7 and σ=0.9 whereas in figures 3(b), for σ=0.9 the number of smokers decrease in comparison to σ=0.7 and σ=0.5 in 3(c) for σ=0.9 the number of quitters increase in comparison to σ=0.7 and σ=0.5. Thus clearly illustrating the variations in numbers, for different fractional order parameter σ values .

In this case when the value of k increases, the value of (1–k) decreases and R_0 value also decreases and the Time series is in Figure 4.

Figure 3. Time series of system (3) with (a) Potential smokers (b) Smokers (c) Quitters for σ=0.5, σ=0.7, σ=0.9 with h=2.1

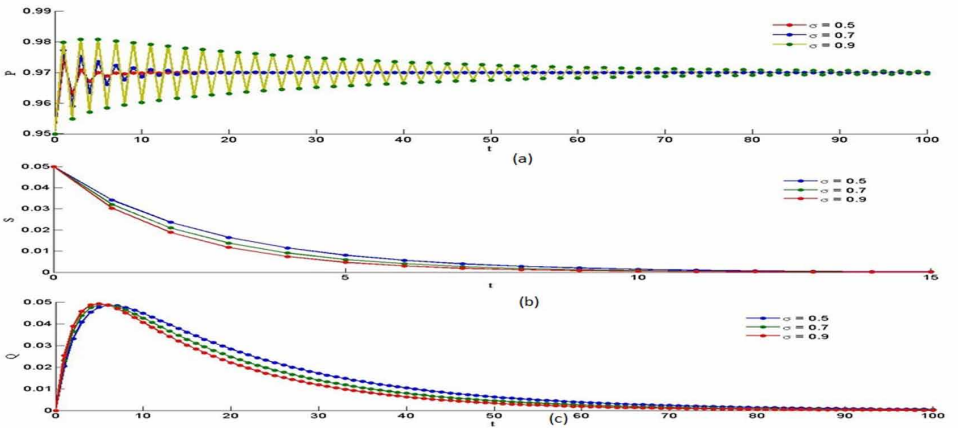


Figure 4. Time series of system (3) with (a) Potential smokers (b)Smokers (c) Quitters for k=0.2, 0.4, 0.6, 0.8, 0.9, h=2.1 and σ=0.5

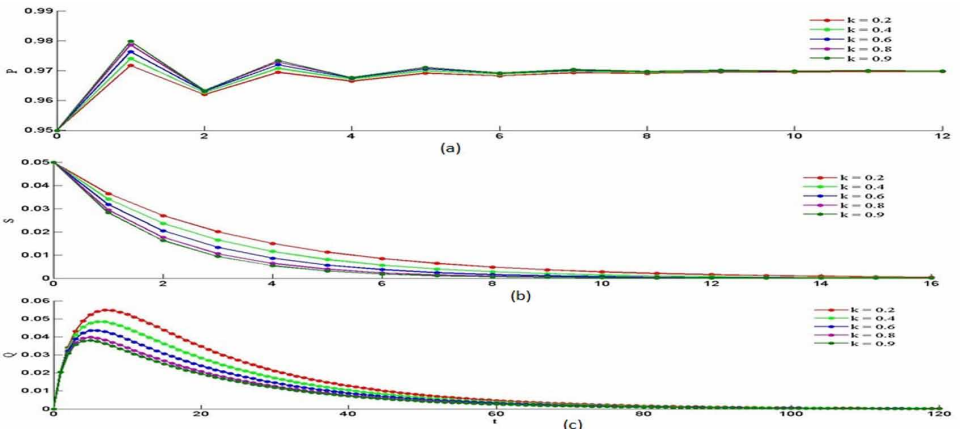


Table 1.

k	R_0
0.2	0.4157<1
0.4	0.3118<1
0.6	0.2079<1
0.8	0.1039<1
0.9	0.0520<1

Example 3: Stability Analysis of the smoking present equilibrium point $E_1(P^*,S^*,Q^*)$:

The following parametric values are considered

$\beta = 0.99$, k = 0.1, $\mu = 0.2$, $\alpha = 0.92$, $\gamma = 0.3$,$\sigma = 0.5$, h = 3.0

with initial conditions P(0)=0.95, S(0)=0.5, Q(0)=0 such that P+S+Q=1.

The Jacobian matrix of system (3) at E_1 is

$$J\left(0.3222,0.5363,0.2320\right)=\begin{pmatrix} 0.3704 & -0.5610 & 0 \\ 0.9339 & 1.0010 & 0 \\ 0 & 0.1691 & -0.3552 \end{pmatrix}.$$

The eigen values and the smoking generation number are λ_1=-0.3552, $\lambda_{2,3}$=0.6857$\pm i$0.6515 so that $|\lambda_1|$=0.3552<1, $|\lambda_{2,3}|$=0.9459<1 and R_0=1.4256>1. Since all of the eigen values are less than one and smoking generation number is greater than one, the smoking present equilibrium point is local asymptotically stable as seen in Figure 5

For, R_0=1.4256>1, Figure 5(a) displays the variation in the number of potential smokers and reaching stability. There is a surge in the number of smokers and they approach stability also there is an increase in the number of quitters. From Figure 5(b), 5(c), 5(d) and 5(e) for the given initial conditions, the solution curves tend

Figure 5. Time series of system (3) with (a) Potential Smokers, Smokers and Quitters (b)Phase portrait in (P, S, Q) plane (c) Phase portrait in (P, S) plane (d) Phase portrait in (P, Q) plane (e) Phase portrait in (S, Q) plane

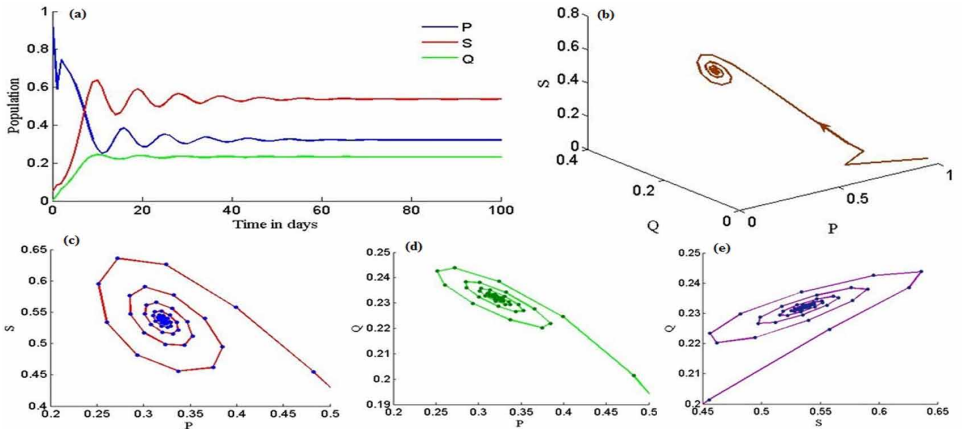


Table 2.

k	E_1	R_0
0.1	(0.3222, 0.5363, 0.2320)	1.4256>1
0.2	(0.3595, 0.5561, 0.2344)	1.2672>1
0.3	(0.4097, 0.5632, 0.2353)	1.1088>1
0.4	(0.4837, 0.5325, 0.2316)	0.9504<1

Figure 6. Time series of system (3) with (a) Potential smokers (b) Smokers (c) Quitters for σ=0.5, σ=0.7, σ=0.9 with h=2.0

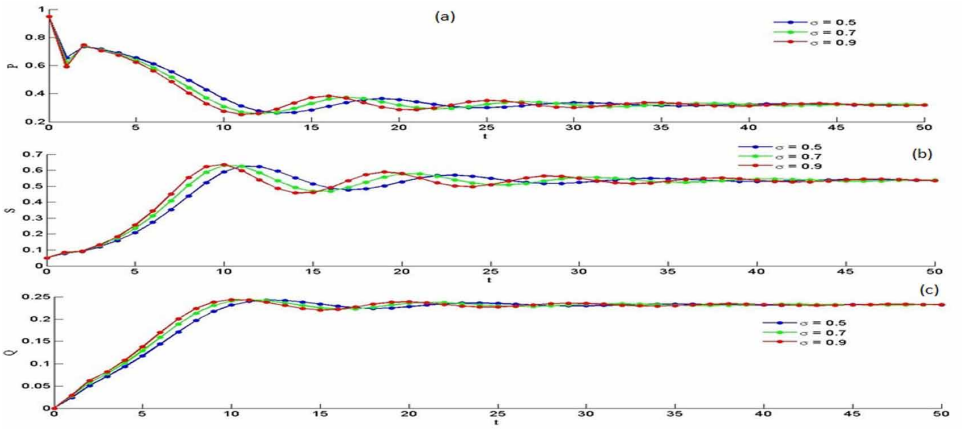
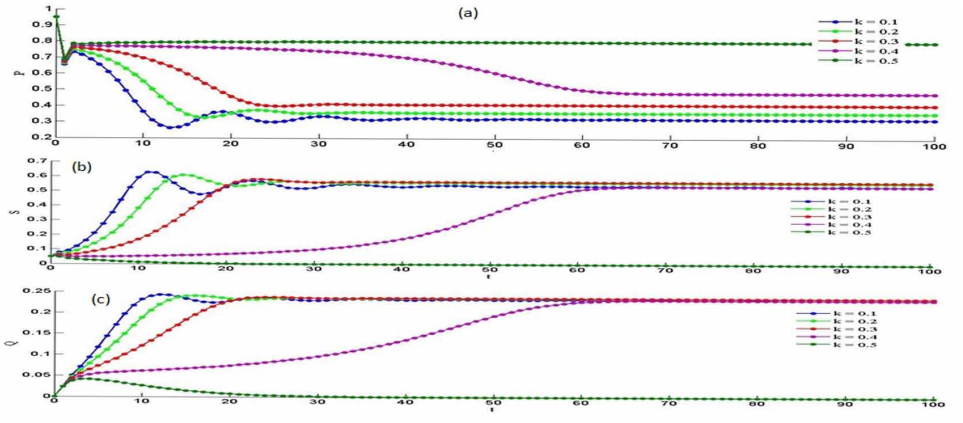


Figure 7. Time series of system (3) with (a) Potential smokers (b) Smokers (c) Quitters for k=0.1, 0.2, 0.3, 0.4, 0.5, h=2.0 and σ=0.5



to the equilibrium E_1, when $R_0>1$, hence, system (3) is local asymptotically stable about E_1 for the given set of values.

In Figure 6 (a), 6(b) and 6(c), it is clearly observed that the change in the number of potential smokers, smokers and quitters when σ values are varied making the rich dynamics of the fractional order system to be known.

For different values of k, the time series is seen in Figure 7.

For k=0.1, the equilibrium point is stable and also as k increases, (1–k) decreases and R_0 also decreases.

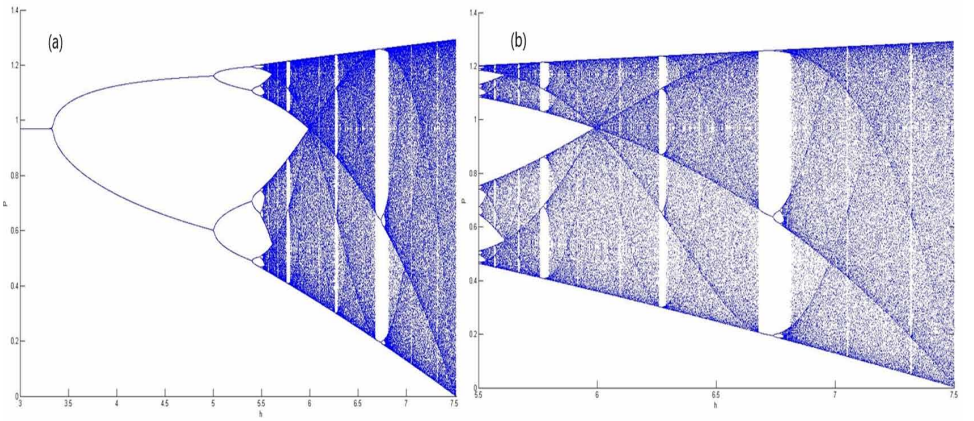
BIFURCATION DIAGRAMS

In dynamical systems, bifurcation sets in when a small smooth change made to the parameter values (the bifurcation parameters) of a system results in a sudden qualitative or topological change in its behaviour. Generally, at bifurcation point, changes in the local stability properties of equilibria, periodic orbits or other invariant sets are observed (Sohel, 2015).

The parametric values are:

P(0)=0.95, S(0)=0.05, Q(0)=0.0, β = 0.15, k = 0.4, μ = 0.03,α= 0.22, γ = 0.25, σ = 0.5

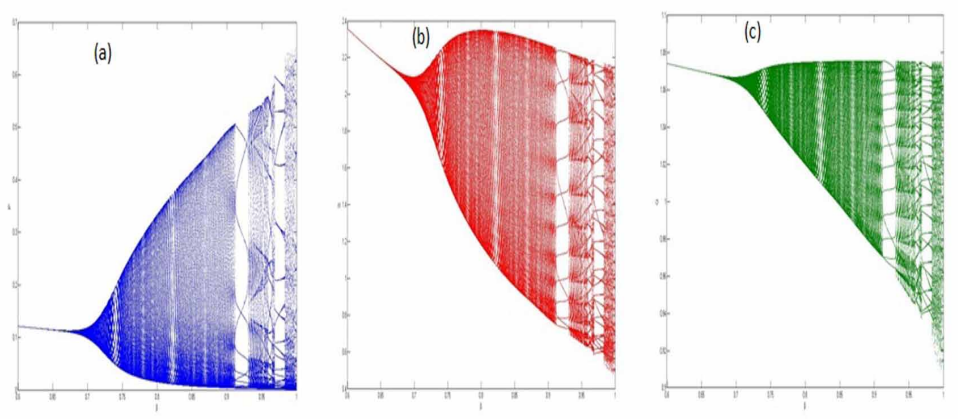
Figure 8. (a) Bifurcation diagram for system (3) in (h, P) plane with h∈[3,7.5] (b) Local Amplification for h∈[5.5,7.5]



The parametric values are:

P(0)=0.95, S(0)=0.05, Q(0)=0.0, h=5.0, k = 0.4, μ = 0.03,α= 0.22, γ = 0.25, σ = 0.5

Figure 9. Bifurcation diagram for system (3), with (a) (β, P) plane (b) (β, Q) plane (c) (β, Q) plane with β∈[0.6,1.0]



CONCLUSION

The purpose of this chapter is to analyze the effectiveness of quit smoking mobile apps in smoking cessation. To this end, a mathematical model was formulated with a system of nonlinear differential equations. The population was divided into three classes namely Potential Smokers P(t), Smokers(S) and Quitters(Q). From the described model, a continuous fractional order model was attained and with the process of discretization using piecewise constant arguments method, discrete fractional order system (3) was obtained. The smoking generation number is calculated with the help of spectral radius of the next generation matrix. The smoking generation number is $R_0 = \frac{\beta(1-k)(1-\mu)}{(\gamma+\mu)}$. In this study, it is showed that there exists a smoking free equilibrium point which is local asymptotically stable if R_0<1 indicating the absence of the smokers in the population. The smoking present equilibrium point which is local asymptotically stable if R_0>1 indicating the presence of the smokers in the population. It is also observed that as k increases, (1–k) decreases and R_0 also decreases. It is seen that the smokers will decrease when effectiveness of quit smoking apps increases. The usage of these apps, may somewhat reduce the number

of smokers but it fails to surge the number of quitters considerably. It is the responsibility, of the society and individuals to promote awareness about the detrimental bearings of smoking on health which is crucial for prevention of smoking. For these apps to be effective in smoking cessation, they need to be user friendly, possibly developed in accordance to the place and language and finally awareness programs are to be promoted.

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APPENDIX

A list of MATLAB functions created for simulation of fractional order discrete system:

```
clear
set(0,'DefaultAxesFontsize',13)
time=100;
beta = 0.15; % Contact rate
k = 0.4; % Effect of Mobile Apps
mu = 0.03; % Natural death rate
alpha = 0.22; % Contact rate between smokers and Quitters
theta = 0.25; % Rate of Quitting and Smoking
sigma = 0.5; % Fractional order
h = 3.3; % Step size
P (1) =0.95;
S (1) =0.05;
Q (1) =0.0;
for t=1: time
P (t + 1) = P(t) + (h^(sigma)/gamma (sigma + 1)) * (P(t)*(1-P(t))-beta*(1-k)
    *P(t)*S(t)-mu*P(t));
S (t + 1) = S(t) + (h^(sigma)/gamma (sigma + 1)) * (beta*(1-k) *P(t)*S(t) +
    alpha*S(t)*Q(t)-theta*S(t)-mu*S(t));
Q (t + 1) = Q(t) + (h^(sigma)/gamma (sigma + 1)) * (theta*S(t)- alpha*S(t)*Q(t)-
    mu*Q(t));
end
%Figure 2-(a): Time series of system (3) with Potential smokers
plot ([0:1: time],P,'b-','lineWidth',2)
legend('P')
xlabel ('Time in days'), ylabel('Population');
%Figure 2-(b): Time series of system (3) with Smokers
plot ([0:1: time], S,'b-','lineWidth',2)
legend('S')
xlabel ('Time in days'), ylabel('Population');
%Figure 2-(c): Time series of system (3) with Quitters
plot ([0:1: time], Q,'b-','lineWidth',2)
legend('Q')
xlabel ('Time in days'), ylabel('Population');
%Figure 2-(d): Phase portraits in (P, S, Q) plane
plot3(P, S, Q,'b-','lineWidth',2)
xlabel ('P'), ylabel('S'), zlabel('Q');
```

```
%Figure 2-(e): Phase portraits in (P, S) plane
plot (P, S,'b-','lineWidth',2)
xlabel ('P'), ylabel('S');
%Figure 2-(f): Phase portraits in (P, Q) plane
plot (P, Q,'b-','lineWidth',2)
xlabel ('P'), ylabel('Q');
```

Chapter 5
Bifurcation and Chaos in a Discrete Fractional Order Prey-Predator System Involving Infection in Prey

A. George Maria Selvam
https://orcid.org/0000-0003-2004-3537
Sacred Heart College (Autonomous), India

R. Dhineshbabu
https://orcid.org/0000-0003-0647-6184
Sacred Heart College (Autonomous), India

ABSTRACT

This chapter considers the dynamical behavior of a new form of fractional order three-dimensional continuous time prey-predator system and its discretized counterpart. Existence and uniqueness of solutions is obtained. The dynamic nature of the model is discussed through local stability analysis of the steady states. Qualitative behavior of the model reveals rich and complex dynamics as exhibited by the discrete-time fractional order model. Moreover, the bifurcation theory is applied to investigate the presence of Neimark-Sacker and period-doubling bifurcations at the coexistence steady state taking h as a bifurcation parameter for the discrete fractional order system. Also, the trajectories, phase diagrams, limit cycles, bifurcation diagrams, and chaotic attractors are obtained for biologically meaningful sets of parameter values for the discretized system. Finally, the analytical results are strengthened with appropriate numerical examples and they demonstrate the chaotic behavior over a range of parameters. Chaos control is achieved by the hybrid control method.

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INTRODUCTION

Epidemiology and ecology are two important research fields of mathematical biology. The first breakthrough work of Lotka (Lotka, 1925) and Volterra (Volterra, 1926) model with coupled nonlinear continuous prey predator systems are discussed in the modern mathematical biology. Moreover, the study of epidemiology and ecology is combined as eco-epidemiology. The dynamics of interacting species in prey predator model with disease spreads are analyzed in eco-epidemiology. Eco-epidemiology population growth dynamics with spread of diseases due to parasites and viruses are efficiently taken care of by complex nonlinear mathematical models. These models are also used to determine steady states, their local stability, periodic solutions, various types of bifurcation diagrams and nature of chaotic attractors (if exist). A huge number of statistical and mathematical models have been used in the study of eco-epidemiological models.

Mathematical models play a vital role in understanding the spread and control of a disease. Mathematical models are apt to model realistic situations like dynamics in population biology, bacterial or viral growth, population of an endangered species, a human population involving its age distribution or not and so on (Edelstein Keshet, 2005). Ecology, the study of relationship between environment and their species is now an enormous field considering competition and prey - predator interactions, multi-species societies, evolution of pesticide resistant strains, ecological and plant - herbivore models. There is also a lot of practical applications for single-species in the biomedical sciences.

In the past years, especially, the study of prey-predator models has been a hot topic for many researchers. Differential equations have played an important role in the investigation of prey-predator interactions and it will continue as an efficient tool in forthcoming explorations. The infectious diseases in population dynamics in a prey-predator system has been garnering great attention because natural species do not exist in isolation, the development of diseases appears due to interaction with other species for food or they are predated by other species. The authors (Prasenjet et al., 2014) studied the prey predator with infectious disease in the prey population or predator population. The nonlinear feedback controls, positive controls have been applied by many researchers to control the chaos in prey-predator system. The adaptive control methods are rapidly developing and finding its application in various fields such as electrical engineering, ecological systems, neural networks and others.

Recently, many novel forms of models have been developed to investigate the natural and social processes that enlarge over time. Now-a-days, these models are referred to as dynamical systems (Frederick, 2006). Dynamical systems are divided into two general categories, i.e. deterministic models and stochastic models respectively. Deterministic models are employed when the number of quantities involved in the

process being modeled is relatively small and all the underlying scientific principles are fairly well understood. The tools of dynamical systems enable the researchers to better understand and investigate the different nonlinear characteristics, which exhibit new phenomena of the systems from various disciplines. Particularly, the tools of the dynamical models such as the applied bifurcation theories are successfully employed to discuss the qualitative behavior of nonlinear systems. This includes the enquiry in to existence of steady states and their stability and bifurcations of periodic orbits, chaotic attractors and synchronization.

Fractional derivative is new form of important tool to describe the dynamical behavior of complex ecological models. The strength of these operators is their nonlocal characteristic which does not exist in the non-fractional order derivatives. This property means that the next aspect of a model relates not only to its present state but also to all of its historical states. In recent decades, many authors (Oldham, 1974) have indicated that fractional-order models are more suitable to study biological dynamics due to memory and hereditary properties exhibited by fractional derivatives. Hence the use of fractional-order differential equations (FDEs) helps us to have a better understanding of the biological system behaviors. Objective of this work is to propose and discuss the discrete version of a fractional order prey-predator model involving infection in prey population by using Caputo fractional derivative and to enquire the inevitable consequences of nonlocal properties induced by fractional-order derivatives.

The rest of this chapter is organized as follows: the basic model is reduced from seven parameters to four parameters and fractional order SI prey and predator interaction involving infection in prey is proposed with its discretization in section 2. Existence and Uniqueness results are discussed in section 3. Local Stability analysis of steady states of the discretized system is discussed in section 4. Bifurcation theory is used to study the existence of Flip and Neimark-Sacker bifurcations of the discretized system in section 5. In section 6, hybrid control strategy is implemented for system (7) to control the chaos due to Flip and Neimark-Sacker bifurcations. Numerical simulations validate the analytical results, including bifurcation diagrams and phase portraits in section 7. Finally the chapter ends with a brief conclusion.

FRACTIONAL - ORDER SYSTEM WITH DISCRETIZATION

Here a three species prey - predator model with Michaelis-Menten-Holling type II functional response (Wuhaid and Abu Hasan, 2012) is considered. The prey population splits into two types, susceptible prey, infected prey and one species predating on both species (S&I). Now the following mathematical model is taken for

investigation of stability and other qualitative properties. In this system, susceptible prey follows the logistic growth

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right)-bSI,\\
\frac{dI}{dt} &= bSI - f\left(I,P\right)P,\\
\frac{dP}{dt} &= e\,f\left(I,P\right)P - gP.
\end{aligned} \tag{1}$$

In system (1), S, I and P denote the number of susceptible prey, infected prey and predator populations at time t. All the system parameters r,K,b,e,g have positive values that stand for susceptible prey intrinsic rate, the environmental carrying capacity, the rate of transmission from susceptible to infected prey population, the conversion efficiency rate and the death rate of predator population respectively. Also $f\left(I,P\right)=\dfrac{\gamma I}{P+\gamma\theta I}$ is the Michaelis-Menten-Holling functional response of infected prey by predator, γ is the total violence rate for predator and θ is the treatment time of predator to infected prey.

In order to reduce the parameters of the model (1), the following transformation is carried out:

$$x=\frac{S}{K}, y=\frac{I}{K}, z=\frac{P}{\gamma\theta K} \text{ and } t=rt.$$

This leads to the following non-dimensional form:

$$\begin{aligned}
\frac{dx}{dt} &= x\left(1-x\right)-kxy,\\
\frac{dy}{dt} &= kxy-\beta\left(\frac{yz}{y+z}\right),\\
\frac{dz}{dt} &= \mu\left(\frac{yz}{y+z}\right)-\alpha z,
\end{aligned} \tag{2}$$

where $k=\dfrac{bK}{r}, \beta=\dfrac{\gamma}{r}, \mu=\dfrac{e}{r\theta}$, and $\alpha=\dfrac{g}{r}$ subject to the following initial values $x(0)=x_0\geq 0$, $y(0)=y_0\geq 0$ and $z(0)=z_0\geq 0$.

The fractional calculus introduced the notion of a non-integer order derivative which provides a new modeling approach for systems with complex dynamical properties. Furthermore, fractional order differential equations has been found especially advantageous in automatic control and system theory, where fractional order differential equations are used to attain more accurate description of the dynamical systems, develop the characteristics of control loops and enhance the novel control strategies. The model under consideration is a new form of fractional order continuous three dimensional prey - predator system with infection in prey populations

$$\begin{aligned} {}_0^C D_t^q x(t) &= x(1-x) - kxy,\ x(0) = x_0, \\ {}_0^C D_t^q y(t) &= kxy - \beta\left(\frac{yz}{y+z}\right),\ y(0) = y_0, \\ {}_0^C D_t^q z(t) &= \mu\left(\frac{yz}{y+z}\right) - \alpha z,\ z(0) = z_0, \end{aligned} \tag{3}$$

where q is the fractional order $0<q\leq 1$, especially when $q=1$, the system (3) is a classical integer order system and

$${}_0^C D_t^q f(t) = \frac{1}{\Gamma(n-q)} \int_0^t \frac{f^{(n)}(s)}{(t-s)^{q-n+1}} ds,$$

for $n-1<q<n$ (Caputo, 1967) is the standard Caputo definition of fractional derivatives.

Discretization Process

In applied mathematics, the process of transforming continuous models described by differential equations into its discrete counterpart is termed as discretization (Agarwal et al, 2013). The discrete version of the model is more suitable for implementing numerical methods and computer simulations. The system (3) is discretized with piecewise constant arguments process (Elsadany and Matouk, 2015) are given as

$$\begin{aligned} {}_0^C D_t^q x(t) &= x([t/h]h)\left(1 - x([t/h]h)\right) - kx([t/h]h)\, y([t/h]h), \\ {}_0^C D_t^q y(t) &= kx([t/h]h)\, y([t/h]h) - \beta\left(\frac{y([t/h]h)\, z([t/h]h)}{y([t/h]h) + z([t/h]h)}\right), \\ {}_0^C D_t^q z(t) &= \mu\left(\frac{y([t/h]h)\, z([t/h]h)}{y([t/h]h) + z([t/h]h)}\right) - \alpha z([t/h]h). \end{aligned} \tag{4}$$

First, taking $0 \le t < h$, so $0 \le (t/h) < 1$. Thus,

$$\begin{aligned}
{}_0^C D_t^q x(t) &= x_0\left(1-x_0\right) - kx_0y_0, \\
{}_0^C D_t^q y(t) &= kx_0y_0 - \beta\left(\frac{y_0z_0}{y_0+z_0}\right), \\
{}_0^C D_t^q z(t) &= \mu\left(\frac{y_0z_0}{y_0+z_0}\right) - \alpha z_0.
\end{aligned} \tag{5}$$

The solution of (5) is

$$\begin{aligned}
x_1(t) &= x_0 + J^q\left(x_0\left(1-x_0\right) - kx_0y_0\right) = x_0 + \frac{t^q}{q\Gamma(q)}\left(x_0\left(1-x_0\right) - kx_0y_0\right), \\
y_1(t) &= y_0 + J^q\left(kx_0y_0 - \beta\left(\frac{y_0z_0}{y_0+z_0}\right)\right) = y_0 + \frac{t^q}{q\Gamma(q)}\left(kx_0y_0 - \beta\left(\frac{y_0z_0}{y_0+z_0}\right)\right), \\
z_1(t) &= z_0 + J^q\left(\mu\left(\frac{y_0z_0}{y_0+z_0}\right) - \alpha z_0\right) = z_0 + \frac{t^q}{q\Gamma(q)}\left(\mu\left(\frac{y_0z_0}{y_0+z_0}\right) - \alpha z_0\right).
\end{aligned}$$

Secondly, with $h \le t < 2h$, so $1 \le (t/h) < 2$. Then,

$$\begin{aligned}
{}_0^C D_t^q x(t) &= x_1\left(1-x_1\right) - kx_1y_1, \\
{}_0^C D_t^q y(t) &= kx_1y_1 - \beta\left(\frac{y_1z_1}{y_1+z_1}\right), \\
{}_0^C D_t^q z(t) &= \mu\left(\frac{y_1z_1}{y_1+z_1}\right) - \alpha z_1,
\end{aligned}$$

which have the following solution

$$\begin{aligned}
x_2(t) &= x_1(h) + \frac{(t-h)^q}{q\Gamma(q)}\left(x_1(h)\left(1-x_1(h)\right) - kx_1(h)y_1(h)\right), \\
y_2(t) &= y_1(h) + \frac{(t-h)^q}{q\Gamma(q)}\left(kx_1(h)y_1(h) - \beta\left(\frac{y_1(h)z_1(h)}{y_1(h)+z_1(h)}\right)\right), \\
z_2(t) &= z_1(h) + \frac{(t-h)^q}{q\Gamma(q)}\left(\mu\left(\frac{y_1(h)z_1(h)}{y_1(h)+z_1(h)}\right) - \alpha z_1(h)\right).
\end{aligned}$$

Proceeding like this up to t times, one arrives at

$$\begin{aligned} x_{t+1}(t) &= x_t(th) + \frac{(t-th)^q}{q\Gamma(q)}\Big(x_t(th)\big(1-x_t(th)\big) - kx_t(th)y_t(th)\Big), \\ y_{t+1}(t) &= y_t(th) + \frac{(t-th)^q}{q\Gamma(q)}\left(kx_t(th)y_t(th) - \beta\left(\frac{y_t(th)z_t(th)}{y_t(th)+z_t(th)}\right)\right), \\ z_{t+1}(t) &= z_t(th) + \frac{(t-th)^q}{q\Gamma(q)}\left(\mu\left(\frac{y_t(th)z_t(th)}{y_t(th)+z_t(th)}\right) - \alpha z_t(th)\right), \end{aligned} \tag{6}$$

where $th \le t < (t+1)h$. As $t \to (t+1)h$, then the system (6) becomes

$$\begin{aligned} x_{t+1} &= x_t + \frac{h^q}{q\Gamma(q)}\Big(x_t\big(1-x_t\big) - kx_t y_t\Big), \\ y_{t+1} &= y_t + \frac{h^q}{q\Gamma(q)}\left(kx_t y_t - \beta\left(\frac{y_t z_t}{y_t+z_t}\right)\right), \\ z_{t+1} &= z_t + \frac{h^q}{q\Gamma(q)}\left(\mu\left(\frac{y_t z_t}{y_t+z_t}\right) - \alpha z_t\right), \end{aligned} \tag{7}$$

where $q \in (0,1]$,

$$J^q f(t) = \frac{1}{\Gamma(q)}\int_0^t (t-s)^{q-1} f(s)ds$$

and $h>0$ is defined as time interval of production.

EXISTENCE AND UNIQUENESS RESULTS

Let us consider the fractional order prey predator model (3) in the region $\Omega \times (0,T]$ where

$$\Omega = \left\{\big(x,y,z\big) \in \mathbb{R}_+^3 : \max\big(|x|,|y|,|z| \le Q\big)\right\}$$

and $\Omega \times (0,T]$ is taken as a region for existence and uniqueness. The approach used in (Hong-LiLi et al, 2016) is utilized. The fractional order system (3) can be expressed as follows

$$D^q X(t) = G\big(X(t)\big),\ t \in (0,T], X(0) = X_0,$$

where

$$X = \begin{bmatrix} x \\ y \\ z \end{bmatrix},\ X_0 = \begin{bmatrix} x_0 \\ y_0 \\ z_0 \end{bmatrix}, G(X) = \begin{bmatrix} x(1-x) - kxy \\ kxy - \beta\left(\frac{yz}{y+z}\right) \\ \mu\left(\frac{yz}{y+z}\right) - \alpha z \end{bmatrix}.$$

Define the maximum norm as $\|X\| = \max_{t\in(0,T]} |X(t)|$.

The norm of the matrix $N=[n_{ij}(t)]$ is defined by $\|N\| = \max_j \sum_i |n_{ij}|$ (Matouk et al., 2015).

The solution of fractional order system (3) is obtained as

$$L(X) = X = X_0 + \frac{1}{\Gamma(q)} \int_0^t (t-s)^{q-1} G\big(X(s)\big) ds.$$

So

$$L(X_1) - L(X_2) = \frac{1}{\Gamma(q)} \int_0^t (t-s)^{q-1} \Big(G\big(X_1(s)\big) - G\big(X_2(s)\big)\Big) ds\,.$$

Thus, one gets the following inequality

$$\big|L(X_1) - L(X_2)\big| \le \frac{1}{\Gamma(q)} \int_0^t \big|(t-s)^{q-1}\big| . \Big|\big(G\big(X_1(s)\big) - G\big(X_2(s)\big)\big)\Big| ds. \tag{8}$$

Since

$$\begin{aligned} \big|G(X_1) - G(X_2)\big| &\le \left[(1+2Q(1+k))|x_1 - x_2| + \left(2kQ + \frac{(\beta+\mu)}{4}\right)|y_1 - y_2| + \left(\alpha + \frac{(\beta+\mu)}{4}\right)|z_1 - z_2| \right] \\ &\le \left\{ 1+2Q(1+k); 2kQ + \frac{(\beta+\mu)}{4}; \alpha + \frac{(\beta+\mu)}{4} \right\} |X_1 - X_2|. \end{aligned} \tag{9}$$

Substituting equation (9) in (8), one obtains

$$\left|L(X_1)-L(X_2)\right| \le \frac{1}{\Gamma(q)}\int_0^t \left|(t-s)^{q-1}\right|.\left\{1+2Q(1+k);2kQ+\frac{(\beta+\mu)}{4};\alpha+\frac{(\beta+\mu)}{4}\right\}\left|X_1-X_2\right|ds,$$

that yields

$$\left\|L(X_1)-L(X_2)\right\| \le \frac{T^q}{q\Gamma(q)}\max\left\{1+2Q(1+k);2kQ+\frac{(\beta+\mu)}{4};\alpha+\frac{(\beta+\mu)}{4}\right\}\left\|X_1-X_2\right\|$$

$$\left\|L(X_1)-L(X_2)\right\| \le \phi\left\|X_1-X_2\right\|,$$

where

$$\phi = \frac{T^q}{q\Gamma(q)}\max\left\{1+2Q(1+k);2kQ+\frac{(\beta+\mu)}{4};\alpha+\frac{(\beta+\mu)}{4}\right\}.$$

If $\phi<1$, then the mapping $X=L(X)$ is a contraction mapping.
Now, one is ready to state the following theorem.

Theorem 1: In the specified region $\Omega\times(0,T]$, sufficient condition for existence and uniqueness of the solution of fractional order system (3) is

$$\phi = \frac{T^q}{q\Gamma(q)}\max\left\{1+2Q(1+k);2kQ+\frac{(\beta+\mu)}{4};\alpha+\frac{(\beta+\mu)}{4}\right\}<1.$$

EXISTENCE AND STABILITY ANAYSIS OF STEADY STATES OF THE DISCRETIZED SYSTEM

In this section, existence of the steady state of the discretized fractional order system (7) is established. The Variation matrix of the system (7) is evaluated and local stability analysis of the steady state of (7) is studied based on the Jury conditions.

Existence of Steady States of the Discretized System

In order to obtain the steady states of the discretized system (7), consider the system of algebraic equations as given by:

$$\begin{cases} x = x + \dfrac{h^q}{q\Gamma(q)}\Big(x(1-x) - kxy\Big), \\ y = y + \dfrac{h^q}{q\Gamma(q)}\left(kxy - \beta\left(\dfrac{yz}{y+z}\right)\right), \\ z = z + \dfrac{h^q}{q\Gamma(q)}\left(\mu\left(\dfrac{yz}{y+z}\right) - \alpha z\right). \end{cases} \tag{10}$$

Obviously, the algebraic system (10) has always three non-negative steady states,

(i) $S_0=(0,0,0)$
(ii) $S_1=(1,0,0)$
(iii) τ_1, τ_2 and τ_3,

where

$$x^* = \frac{\beta(\mu-\alpha)}{k\mu}, y^* = \frac{1}{k}\left(1-x^*\right) \text{and } z^* = \frac{(\mu-\alpha)}{k\alpha}\left(1-x^*\right).$$

The following theorem summarises the existence of steady states of the system (7).

Theorem 2: The existence of steady states satisfies:

- *The trivial and boundary steady states* S_0 and S_1 always exists.
- *If* $\mu>\alpha$, then the coexistence steady state S_2 exists.

Stability Analysis of the Steady States of the Discretized System

In this section, the nonlinear dynamical behavior of the discretized system is investigated (7). Now, the criteria for stability analysis in the neighborhood of each steady state is dicussed. At any steady state, the Variation matrix $V(x,y,z)$ of the system (7) has the form

$$V(x,y,z)=\begin{bmatrix} 1+\frac{h^q}{q\Gamma(q)}(1-2x-ky) & -\frac{h^q}{q\Gamma(q)}kx & 0 \\ \frac{h^q}{q\Gamma(q)}ky & 1+\frac{h^q}{q\Gamma(q)}\left(kx-\frac{\beta z^2}{(y+z)^2}\right) & -\frac{h^q}{q\Gamma(q)}\left(\frac{\beta y^2}{(y+z)^2}\right) \\ 0 & \frac{h^q}{q\Gamma(q)}\left(\frac{\mu z^2}{(y+z)^2}\right) & 1+\frac{h^q}{q\Gamma(q)}\left(\frac{\mu y^2}{(y+z)^2}-\alpha\right) \end{bmatrix} \tag{11}$$

In the following theorem, the stability for discrete fractional order the system (7) is examined through the relations satisfied by Jury condition. For this purpose, following theorem (Grove and Ladas, 2004) is needed.

Theorem 3: Let us consider the cubic equation of the form

$$\lambda^3+\sigma_1\lambda^2+\sigma_2\lambda+\sigma_3=0, \tag{12}$$

where σ_1, σ_2 and σ_3 are constants. The roots of the cubic equation (12) lie within the open unit disk if and only if the following conditions are satisfied:

$|\sigma_1+\sigma_3|<1+\sigma_2, |\sigma_1-3\sigma_3|<3-\sigma_2$ and $\sigma_3^2+\sigma_2-\sigma_3\sigma_1<1$.

Here the trivial and boundary steady states are not considered for discussion since the Variation matrix does not exist.

Coexistence Steady State

At the coexisting steady state S_2=(x^*,y^*,z^*), the Variation matrix V_{S_2} is

$$V(S_2)=\begin{bmatrix} 1-\frac{h^q}{q\Gamma(q)}x^* & -\frac{h^q}{q\Gamma(q)}x^*k & 0 \\ \frac{h^q}{q\Gamma(q)}\left(1-x^*\right) & 1+\frac{h^q}{q\Gamma(q)}\xi_1 x^*k & -\frac{h^q}{q\Gamma(q)}\xi_1^2\beta \\ 0 & \frac{h^q}{q\Gamma(q)}x^*k\xi_2 & 1-\frac{h^q}{q\Gamma(q)}x^*k\xi_3 \end{bmatrix}.$$

The characteristic equation at S_2 is

$$P(\lambda) = \lambda^3 + \tau_1\lambda^2 + \tau_2\lambda + \tau_3 = 0, \tag{13}$$

where $\tau_1 = \left(\frac{h^q}{q\Gamma(q)}\right)x^*\left(1-k(\xi_1-\xi_3)\right)-3$,

$$\tau_2 = 3-2\left(\frac{h^q}{q\Gamma(q)}\right)x^*\left(1-k(\xi_1-\xi_3)\right)-\left(\frac{h^q}{q\Gamma(q)}\right)^2 x^*k\left[x^*\left(1+\xi_1-\xi_3+k\xi_1\xi_3\right)-\left(1+\beta\xi_1^2\xi_2\right)\right]$$

and

$$\tau_3 = \left(\frac{h^q}{q\Gamma(q)}\right)x^*\left(1-k(\xi_1-\xi_3)\right)+\left(\frac{h^q}{q\Gamma(q)}\right)^2 x^*k\left[x^*\left(1+\xi_1-\xi_3+k\xi_1\xi_3\right)-\left(1+\beta\xi_1^2\xi_2\right)\right]$$
$$-\left(\frac{h^q}{q\Gamma(q)}\right)^3 x^{*2}k\left[k\xi_3\left(x^*+\xi_1x^*-1\right)-\beta\xi_1^2\xi_2\right]-1, \tag{14}$$

such that

$$\xi_1 = \frac{\alpha}{\mu}, \xi_2 = \frac{\mu-\alpha}{\beta}, \text{ and } \xi_3 = \frac{\alpha}{\beta}. \tag{15}$$

Theorem 4: If the condition $\mu>\alpha$ is satisfied, then the coexistence state S_2 of system (7) is a sink and it is locally asymptotically stable if

(i) $|\tau_1+\tau_3|<1+\tau_2$,
(ii) $|\tau_1-3\tau_3|<3-\tau_2$ and
(iii) $\tau_3^2+\tau_2-\tau_1\tau_3<1$, where $\tau1$, $\tau2$ and $\tau3$ are as in (14).

BIFURCATIONS OF THE COEXISTENCE STEADY STATE OF THE DISCRETIZED FRACTIONAL - ORDER SYSTEM

Bifurcation analysis is an interesting and fruitful topic to investigate the topological nature of the model. Recently, there are numerous articles published on the existence of Neimark-Sacker and Period doubling bifurcations (Din, 2018). Bifurcation occurs only when a certain parameter value called as a bifurcation parameter value passes through a critical value and the qualitative behavior undergoes changes. In this current section, Neimark-Sacker and Period doubling bifurcation behaviors

of the coexistence positive steady state S2 of system (7) are investigated. Standard bifurcation theory techniques are useful to examine the existence of Neimark-Sacker bifurcation (NSB) and Period doubling bifurcation (PDB) for the coexistence steady state S2 of system (7).

Neimark-Sacker Bifurcation of the Discretized Fractional-Order System

In order to discuss the NSB for the system (7) at the coexisting steady state S_2, choosing h as bifurcation parameter. From (13) it is easy to see that $P(\lambda)=0$ must have a complex conjugate root with modulus one (Elaydi, 2008). Clearly equation (13) will have two pure imaginary roots and one real root. Let $\tau_1,\tau_2=\tau_3$, for some values of h, say $h=h^*$, then equation (13) becomes $(\lambda^2+\tau_2)(\lambda+\tau_1)=0$ which has three roots $\lambda_{1,2}=\pm i\sqrt{\tau_2}$ and $\lambda_3=-\tau_1$. Hence the system (7) undergoes a NSB at the coexistence steady state S_2 if h varies in the small neighborhood of NS_{S_2}, where

$$NS_{S_2}=\left\{(q,h,\alpha,\beta,\mu,k):|\tau_2|=1,\tau_1\neq\pm1,\alpha,q,h,\beta,\mu,k>0\right\}.$$

Furthermore, the study of NSB in system (7), presents the following explicit condition of Hopf bifurcation (Wen, 2005).

Theorem 5: Consider a t dimensional discrete dynamical system $X_{t+1}=f_h\left(X_t\right)$, where $h\in\mathbb{R}$ is a bifurcation parameter. Let X^* be a steady state of f_h and the characteristic equation for Variation matrix $V\left(X^*\right)=\left(\delta_{\ell j}\right)_{t\times t}$ of t dimensional map $f_h(X_t)$ is given by

$$P_h\left(\lambda\right)=\lambda^t+\delta_1\lambda^{t-1}+\cdots+\delta_{t-1}\lambda+\delta_t \text{ where } \delta_\ell=\delta_\ell\left(h,w\right), \ell=1,2,3,...,t$$

and w is control parameter to be determined. Let

$$\Delta_0^{\pm}\left(h,w\right)=1,\Delta_1^{\pm}\left(h,w\right),...,\Delta_t^{\pm}\left(h,w\right)$$

be a sequence of determinates defined by $\Delta_\ell^{\pm}\left(h,w\right)=\det\left(\Lambda_1\pm\Lambda_2\right),\ \ell=1,2,...,t$, where

$$\Lambda_1 = \begin{bmatrix} 1 & \delta_1 & \delta_2 & ... & \delta_{\ell-1} \\ 0 & 1 & \delta_1 & ... & \delta_{\ell-2} \\ 0 & 0 & 1 & ... & \delta_{\ell-3} \\ ... & ... & ... & ... & ... \\ 0 & 0 & 0 & ... & 1 \end{bmatrix}, \Lambda_2 = \begin{bmatrix} \delta_{t-\ell+1} & \delta_{t-\ell+2} & ... & \delta_{t-1} & \delta_t \\ \delta_{t-\ell+2} & \delta_{t-\ell+3} & ... & \delta_t & 0 \\ ... & ... & ... & ... & ... \\ \delta_{t-1} & \delta_t & ... & 0 & 0 \\ \delta_t & 0 & 0 & ... & 0 \end{bmatrix}. \quad (16)$$

Furthermore, the following conditions hold:

(i) Eigenvalue assignment:

$$\Delta_{t-1}^{-}(h_0, w) = 0, \Delta_{t-1}^{+}(h_0, w) > 0, P_{h_0}(1) > 0, (-1)^t P_{h_0}(-1) > 0, = 0, \Delta_{\ell}^{\pm}(h_0, w) > 0,$$

for $\ell = t-3, t-5, \ldots, 1$ (or 2), when t is odd or even, respectively.

(ii) Transversality: $\left[\frac{d\left(\Delta_{t-1}^{-}(h, w)\right)}{dr}\right]_{h=h_0} \neq 0$.

(iii) (iii) Resonance or Non-resonance: $\cos(2\pi/m) = \psi$ or $\cos(2\pi/m) \neq \psi$ where $m = 3,4,\ldots$ and

$$\psi = -1 + 0.5 P_{h_0}(1) \Delta_{t-3}^{-}(h_0, w) / \Delta_{t-2}^{+}(h_0, w).$$

Then, NSB occurs at h_0.

According to the above theorem, for $t=3$, the cubic equation (13) of system (7) is evaluated at the coexistence steady state S_2. Thus the following theorem which presents the criteria for the system (7) to undergo NSB with h_0 as bifurcation parameter is presented.

Theorem 6: The coexistence steady state S_2 of the system (7) undergoes NSB for $\mu > \alpha$, if the following equalities and inequalities hold:

$$\Delta_2^{-}(h) = 1 - \tau_2 + \tau_3(\tau_1 - \tau_3) = 0, \Delta_2^{+}(h) = 1 + \tau_2 - \tau_3(\tau_1 + \tau_3) > 0, P_h(1) = 1 + \tau_1 + \tau_2 + \tau_3 > 0,$$

and $(-1)^3 P_h(-1) = 1 - \tau_1 + \tau_2 - \tau_3 > 0$, where τ_1, τ_2 and τ_3 are given in (14).

Period - Doubling Bifurcation of the Discretized Fractional-Order System

Period doubling bifurcation in a system (7) occurs when a mild change in the parametric value, which leads the system to a new behavior with twice the period of the original system. Moreover the system causes an eigenvalue to pass through -1. When this happens, steady state loses stability, and a stable cycle of period 2 appears. Continued parameter changes may result in a cascade of PDBs and onset of chaos. Furthermore, the study of PDB in system (7), presents the following explicit condition of Flip bifurcation (Wen et al., 2008).

Theorem 7: Consider a t dimensional discrete dynamical system $X_{t+1} = f_h(X_t)$, where $X_{t+1}, X_t \in \mathbb{R}^t$ are the state vectors, $h \in \mathbb{R}$ is a bifurcation parameter. Let X^* be a steady state of f_h and the characteristic equation for Variation matrix $V(X^*) = (\delta_{\ell j})_{t\times t}$ of t dimensional map $f_h(X_t)$ is given by

$$P_h(\lambda) = \lambda^t + \delta_1 \lambda^{t-1} + \cdots + \delta_{t-1}\lambda + \delta_t,$$

where $\delta_\ell = \delta_\ell(h), \ell$=1,2,3,...,t . Let $\Delta_0^{\pm}(h) = 1, \Delta_1^{\pm}(h), ..., \Delta_t^{\pm}(h)$ be a series of determinates defined by $\Delta_\ell^{\pm}(h) = \det(\Lambda_1 \pm \Lambda_2)$, $\ell = 1,2,...,t$, where Λ_1 and Λ_2 are given in (16).

The following conditions hold:

(i) Eigenvalue assignment:

$$P_{h_0}(-1) = 0, P_{h_0}(1) > 0, \Delta_{t-1}^{\pm}(h_0) > 0, \Delta_\ell^{\pm}(h_0) > 0,$$

ℓ=t–2, t–4,…,1 (or 2) when t is odd or even, respectively.

(ii) Transversality: $\dfrac{\sum_{\ell=1}^{t} \delta_\ell'(-1)^{t-\ell}}{\sum_{\ell=1}^{t}(t-\ell+1)(-1)^{t-\ell}\delta_{\ell-1}} \neq 0$, where δ_ℓ' stands for the first derivative of $\delta(h)$ with respect to h.

According to the above theorem, for t=3, the cubic equation (13) of system (7) is evaluated at the coexistence steady state S_2. Thus the following theorem shows that system (7) undergoes PDB if h_0 is taken as bifurcation parameter.

Theorem 8: The coexistence steady state S_2 of the system (7) undergoes PDB for $\mu>\alpha$, if the following equalities and inequalities hold:

$$\Delta_2^-(h)=1-\tau_2+\tau_3(\tau_1-\tau_3)>0, \Delta_2^+(h)=1+\tau_2-\tau_3(\tau_1+\tau_3)>0, \Delta_1^{\pm}(h)=1\pm\tau_2>0, P_h(1)=1+\tau_1+\tau_2+\tau_3>0,$$

and $P_h(-1)=-1+\tau_1-\tau_2+\tau_3=0$, where τ_1, τ_2 and τ_3 are given in (14).

CHAOS CONTROL

This section presents the control strategy in order to move the unstable fractional periodic orbits or the fractional chaotic orbits towards the stable one. In order to control the chaos in system (7), hybrid control feedback methodology (Ali et al., 2018) is introduced. Assume that system (7) undergoes NSB or PDB at unstable steady state (x^*,y^*,z^*), then corresponding fractional-order controlled system is expressed as

$$\begin{aligned}
x(t+1) &= \rho x(t)+\rho\frac{h^q}{q\Gamma(q)}\Big(x(t)\big(1-x(t)\big)-kx(t)y(t)\Big)+(1-\rho)x(t)\\
y(t+1) &= \rho y(t)+\rho\frac{h^q}{q\Gamma(q)}\left(kx(t)y(t)-\beta\left(\frac{y(t)z(t)}{y(t)+z(t)}\right)\right)+(1-\rho)y(t)\\
z(t+1) &= \rho z(t)+\rho\frac{h^q}{q\Gamma(q)}\left(\mu\left(\frac{y(t)z(t)}{y(t)+z(t)}\right)-\alpha z(t)\right)+(1-\rho)z(t).
\end{aligned} \tag{17}$$

where $0<\rho<1$ represents the control parameter. Controlling strategy in (17) is a combination of both parameter perturbation and feedback control. Moreover, by proper choice of controlled parameter ρ, the NSB of the steady state (x^*,y^*,z^*) of controlled system (17) can be advanced (delayed) or even completely eliminated. The Variation matrix of (17) evaluated at the coexistence steady state S_2=(x^*,y^*,z^*) is

$$V_1\left(x^*, y^*, z^*\right) = \begin{bmatrix} 1-\frac{h^q}{q\Gamma(q)}\rho x^* & -\frac{h^q}{q\Gamma(q)}\rho x^* k & 0 \\ \frac{h^q}{q\Gamma(q)}\rho\left(1-x^*\right) & 1+\frac{h^q}{q\Gamma(q)}\rho\xi_1 x^* k & -\frac{h^q}{q\Gamma(q)}\rho\xi_1^2\beta \\ 0 & \frac{h^q}{q\Gamma(q)}\rho x^* k\xi_2 & 1-\frac{h^q}{q\Gamma(q)}\rho x^* k\xi_3 \end{bmatrix}. \quad (18)$$

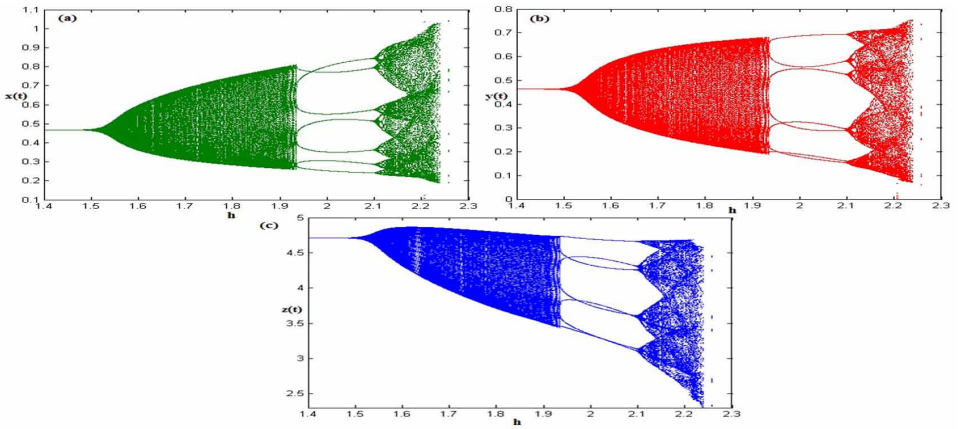
The expressions ξ_1, ξ_2, and ξ_3 have meaning as mentioned in (15). Then, the coexistence steady state S_2 of the controlled system (17) is locally asymptotically stable if roots of the characteristic equation of (18) lie in an open unit disk.

NUMERICAL EXPERIMENTS

In this section, the above theoretical analysis is verified and supported with appropriate examples by considering some special cases of system (7). Numerical simulations manifest clearly interesting rich complex dynamics behaviors. Moreover, hybrid control feedback methodology for chaos control is also demonstrated in this section.

Example 9: *Take* α=0.12, β=0.59, μ=1.34, k=1.15, q=0.97 *and* $1.4 \leq h \leq 2.3$ in system (7) with initial conditions $x(0)$=0.4, $y(0)$=0.3, $z(0)$=0.5. *In this case the system (7) undergoes a NSB*

Figure 1. Neimark-Sacker bifurcation diagram of system (7) in (h,x), (h,y) and (h,z) planes



emerges at the coexisting steady state $S_2=(x^*,y^*,z^*)=(0.4671, 0.4634, 4.7112)$ in a small neighborhood of the bifurcation parameter $h=1.54209$. The corresponding bifurcation diagram is shown in Figure 1. The characteristic polynomial evaluated at S_2 is

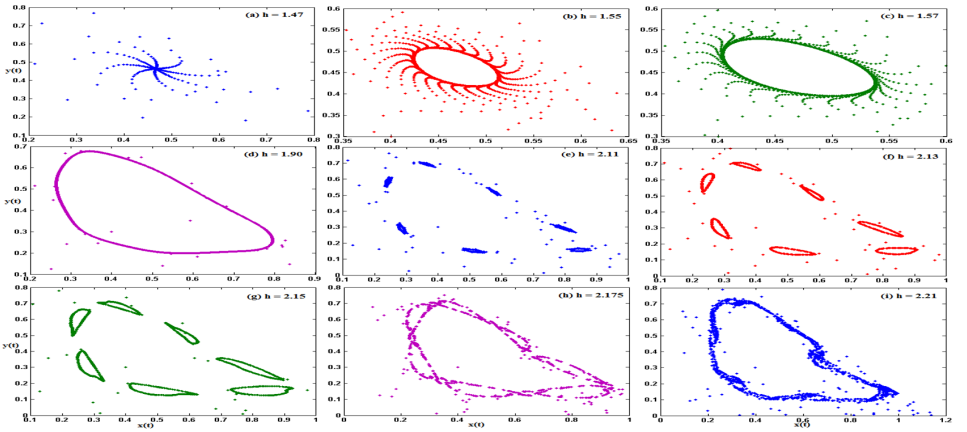
$$\lambda^3 - 2.1859\lambda^2 + 2.1195\lambda - 0.8192 = 0. \tag{19}$$

Furthermore, the roots of (19) are $\lambda_{1,2}=0.6833\pm i0.7301$ and $\lambda_3=0.8192$ with $|\lambda_{1,2}|=|\tau_2|=1$ and $\lambda_3=\tau_1=0.8192\neq\pm1$. Moreover,

$$\Delta_2^-(h)=1-\tau_2+\tau_3(\tau_1-\tau_3)=0,$$
$$\Delta_2^+(h)=1+\tau_2-\tau_3(\tau_1+\tau_3)=0.6579>0,$$
$$P_h(1)=1+\tau_1+\tau_2+\tau_3=0.1145>0,$$
$$(-1)^3P_h(-1)=1-\tau_1+\tau_2-\tau_3=6.1246>0,$$

By Theorem 6, conditions for Neimark-Sacker bifurcation are satisfied near the steady state S_2 at the bifurcation critical value $h=1.54209$. Figure 1(a), 1(b), 1(c) show Neimark-Sacker bifurcation diagrams in (h,x), (h,y) and (h,z) planes respectively.

Figure 2. Phase portraits of system (7) for various values of h in (x,y) planes

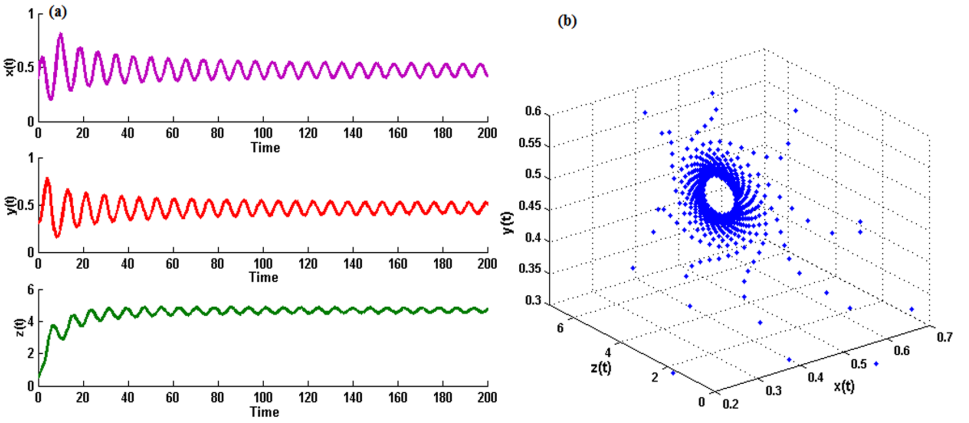


From Figure 2, it is observed that positive steady state S_2 of map (7) is stable for $h<1.54209$ and it loses stability through a Neimark - Sacker bifurcation for $h=1.54209$ and attracting different invariant circle appears for h in the range of

[1.54209, 1.90], see Figure 2(c) and Figure 2(d). The phase portraits with different values of h are plotted in Figure 2 corresponding the value of $h \in [1.4, 2.3]$ in Figure 1, to illustrate these observations. When h increases at certain values, for example $h=2.11$, the circles breakdown and the quasi-periodic orbits appear and it is also seen that seven-coexisting chaotic attractors at $h=2.13$ and $h=2.15$, chaotic attractor sets are plotted in figures 2(h) and 2(i) to illustrate these observations.

Example 10: *This example considers the set of parameter values* $\alpha=0.12$, $\beta=0.59$, $\mu=1.34$, $k=1.15$, $q=0.97$ *with initial conditions* $x(0)=0.4$, $y(0)=0.3$, $z(0)=0.5$. *Example 9 shows that for the system (7), Neimark-Sacker bifurcation occurs as* h varies in [1.4, 2.3]. Moreover, Figure 3 displays a closed invariant circle appearing at $h=1.547$ enclosing unstable positive steady state $S_2=(x^*,y^*,z^*)=(0.4671, 0.4634, 4.7112)$.

Figure 3. Time series and Phase portrait for the system (7)



For these parametric values, the controlled system (17) can be written as

$$\begin{aligned} x(t+1) &= x(t) + \rho \frac{h^q}{q\Gamma(q)}\Big(x(t)\big(1-x(t)\big) - kx(t)y(t)\Big) \\ y(t+1) &= y(t) + \rho \frac{h^q}{q\Gamma(q)}\left(kx(t)y(t) - \beta\left(\frac{y(t)z(t)}{y(t)+z(t)}\right)\right) \\ z(t+1) &= z(t) + \rho \frac{h^q}{q\Gamma(q)}\left(\mu\left(\frac{y(t)z(t)}{y(t)+z(t)}\right) - \alpha z(t)\right). \end{aligned} \tag{20}$$

with α=0.12, β=0.59, μ=1.34, k=1.15, q=0.97, h=1.547 *and* 0<ρ<1.

The Variation of the controlled system (20) evaluated at S_2 is

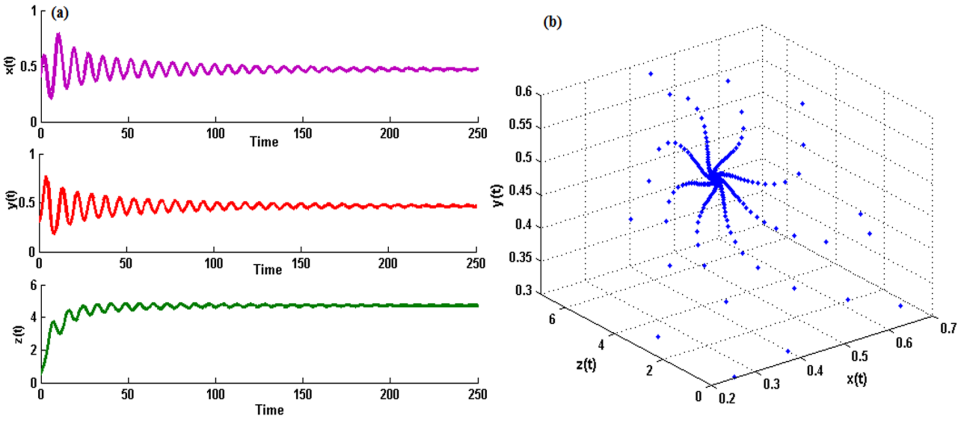
$$V_1\left(x^*, y^*, z^*\right) = \begin{pmatrix} 1-0.7221\rho & -0.8304\rho & 0 \\ 0.8238\rho & 1+0.0744\rho & -0.0073\rho \\ 0 & 1.7171\rho & 1-0.1689\rho \end{pmatrix} \tag{21}$$

The characteristic polynomial of (21) is given by

$$\lambda^3 + \left(0.8166\rho - 3\right)\lambda^2 + \left(0.7524\rho^2 - 1.6333\rho + 3\right)\lambda + \left(0.1155\rho^3 - 0.7524\rho^2 + 0.8166\rho - 1\right) = 0. \tag{22}$$

By Theorem 3, the roots of (22) lie in the unit open disk if and only if 0<ρ<0.9999. Moreover, the time plots for $x(t)$, $y(t)$, $z(t)$ and phase portrait of the controlled system (7) are shown in Figure 4 with ρ=0.96. From Figure 4(a) and 4(b), it is clear that the positive coexistence steady state S_2 is stable.

Figure 4. Time series and Phase portrait for the system (17)



CONCLUSION

This chapter examined the qualitative nature of a discrete counterpart of fractional order prey predator system involving infection in prey. Piecewise constant arguments method was implemented and the discrete fractional order system of the continuous fractional order model was obtained to study the rich dynamics of the proposed

system. A sufficient condition for existence of steady states of the discrete fractional order three species of system (7) is determined. Dynamical behavior of the model is investigated through the local stability analysis of the steady states with the help of Jury conditions. Neimark-Sacker bifurcation occurs for a small range of bifurcation parameter h for the system (7). The numerical simulations are shown for distinct parameter values and the time series diagrams are plotted with phase lines. Finally, hybrid control strategy is successfully implemented to control the chaos due to the occurrence of Neimark-Sacker bifurcation. Numerical examples justify the rich and chaotic dynamics in three species.

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APPENDIX

The following Matlab codes created for simulation of discrete fractional order prey - predator model involving infection in prey population. For illustration is presented an example 10 in Figure 3(a) and Figure 3(b) by typing command

```
set(0,'DefaultAxesFontsize',18)
time=200;
h=1.547;
q=0.97;
k=1.15;
alpha=0.12;
beta=0.59;
mu=1.34;
x(1)=0.4;
y(1)=0.3;
z(1)=0.5;
for t=1:time
x(t+1)=x(t)+(h^q)/(q*gamma(q))*[x(t)*(1-x(t))-k*x(t)*y(t)];
y(t+1)=y(t)+(h^q)/(q*gamma(q))*[k*x(t)*y(t)-(beta*y(t)*z(t)/(z(t)+y(t)))];
z(t+1)=z(t)+(h^q)/(q*gamma(q))*[(mu*y(t)*z(t)/(z(t)+y(t)))-alpha*z(t)];
end
```

(i) Plotting the solution of susceptible prey x(t) for time 0 – 200s in pink color:

```
>> subplot(3,1,1), hold on
>> plot([0:1:time],x,'m-','lineWidth',2);
>> xlabel('Time');
>> ylabel('x(t)');
```

(ii) Plotting the solution of infected prey y(t) for time 0 – 200s in red color:

```
>> subplot(3,1,2), hold on
>> plot([0:1:time],y,'r-','lineWidth',2);
>> xlabel('Time');
>> ylabel('y(t)');
```

(iii) Plotting the solution of predator z(t) for time 0 – 200s in green color:

```
>> subplot(3,1,3), hold on
>> plot([0:1:time],z,'g-','lineWidth',2);
>> xlabel('Time');
>> ylabel('z(t)');
```

(iv) Plotting the trajectory of susceptible prey, infected prey and predator x(t), y(t), z(t) in phase plane in blue color:

```
>> plot3(x,z,y);
>> xlabel('x(t)');
>> ylabel('z(t)');
>> zlabel('y(t)');
```

Chapter 6
Analysis of Discrete System Modelling Followed by Spread of Infectious Diseases Problem in Fuzzy Environments

Abdul Alamin
Maulana Abul Kalam Azad University of Technology, West Bengal, India

Sankar Prasad Mondal
Maulana Abul Kalam Azad University of Technology, West Bengal, India

Kunal Biswas
Maulana Abul Kalam Azad University of Technology, West Bengal, India

Shariful Alam
https://orcid.org/0000-0001-8263-117X
Indian Institute of Engineering Science and Technology, Shibpur, India

ABSTRACT

In this chapter, the authors discuss the solution of spread of infectious diseases in terms of SI model in fuzzy environment, which is modelled in a typical discrete system. As the system is discrete in nature, the concept of difference equation has been embarked. In order to understand the underlying uncertainty perspective, they explored the fuzzy difference equations to study the problem.

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INTRODUCTION

Fuzzy Set Theory

Due to inherent uncertainty of the preference expressions, real-world decision-making activities, management, storage and the extraction of various useful information is not always presented as crisp numbers. It is understood that fuzzy numbers are a significant player in evaluating information systems (Altman, D. 1994). Machine learning, un-certainty mining and associated information systems are established by Fuzzy set theory (Altman, D. 1994). Membership degree in the fuzzy set theory employes a single crisp value within 1\2 0;1. It is understood that classy fuzzy set experiences limitations while working with incomplete and uncertain information's. Due to such limitations, additional generalisations of fuzzy sets were developed (Kosiński, W. 2006).

Fuzzy Epidemic Modelling

The epidemic modelling of the disease model comprises several parameters which are uncertain and heterogeneous in the population mass (Mu, X., Zhang, Q., & Rong, L. 2019). Different dynamical systems in the epidemic models include the application to fuzzy theory approach (Verma, R., Tiwari, S. P., & Upadhyay, R. K. 2019). Several fuzzy parameters in the disease model include the interval value systems which is in a prey-predator model which thereby finds application in the computer network Mishra, B. K., & Pandey, S. K. (2010). Also, the dynamical behaviour of an epidemic modelling with fuzzy transmission has been explained by (Jafelice, R. M., Pereira, B. L., Bertone, A. M. A., & Barros, L. C. 2019). Human disease transmission in the fuzzy epidemic modelling has been studied by (Bufford, J. L., Hulme, P. E., Sikes, B. A., Cooper, J. A., Johnston, P. R., & Duncan, R. P. 2019).

Infectious Disease Modelling

Epidemic modelling is usually dominated by the prevalent human diseases and livestock disease modelling as the second category. Despite several economic impacts of the plant pests and diseases, plant diseases outbreak is less numerous in nature including the impact over the biodiversity Bufford, J. L., Hulme, P. E., Sikes, B. A., Cooper, J. A., Johnston, P. R., & Duncan, R. P. 2019), and the ecosystem services (Bussell, E. H., Dangerfield, C. E., Gilligan, C. A., & Cunniffe, N. J. 2019). Several epidemic journals have focussed on the modelling livestock diseases, plant disease modelling (Thompson, R. N., & Brooks-Pollock, E. 2019), Ebola epidemic (Alves, K. D. S., Moraes, W. B., Silva, W. B. D., & Ponte, E. M. D. 2019), marine diseases

modelling (Bufford, J. L., Hulme, P. E., Sikes, B. A., Cooper, J. A., Johnston, P. R., & Duncan, R. P. 2019), which showcases the significance of mathematical modelling over the disease management.

Also, several groups reported the human disease modelling which tend to bear significantly higher impact in societal behaviour. Due to the prevalence of diversified and detailed data, epidemiological modelling has seen great interest and wide acceptance. Previous models have shown the epidemiological dynamics of pathogens of humans, such as the susceptible-infected-removed (SIR) model and its approximation applied to them which is mostly applied to the plague in India (12). The basis of assumptions of the different features of the human disease modelling is the number of cases of death and birth in each time period along with the significant host interaction. Several other studies also relied on seroprevalence surveys (Cutts, F., Dansereau, E., Ferrari, M., Hanson, M., Mccarthy, K., Metcalf, C, Winter, A. 2020). It is understood that simple modelling is robust and powerful in nature whereas the complex modelling is rew\quired to be provided for different data gathering approach like the dynamical distribution of a variety of host species in the environment. The distribution of the different varieties of the pathogenic population in the provided landscape is also explained by the modelling attributes. The prevalence of multi-state data in the vicinity indicates the richness in the distribution profile of the real data sets (Zhang, B., Cai, Y., Wang, B., & Wang, W. 2020), which are used to understand the disease transmission dynamics in the population.

It is clear from the literature survey that, genomic data are widely accessible and available in human diseases (Omondi, F. H., Chandrarathna, S., Mujib, S., Brumme, C. J., Jin, S. W., Sudderuddin, H., Brumme, Z. L. 2019). The interconnected data sets of the population entities are measured for transmission dynamics in the geographical population space. One of the most significant experiments studied is by using the mobile phone transformation in understanding the epidemiological models (Chang, H.-H., Wesolowski, A., Sinha, I., Jacob, C. G., Mahmud, A., Uddin, D., … Buckee, C. 2019). The use of different data sets for the model development has been employed for the designing of the modelling of the imported pathogenic transmissions (Gottwald, T., Luo, W., Posny, D., Riley, T., & Louws, F. 2019). Also, the data being relied on the global travel parameter also explains the prevalence of large scale pandemic distribution in the modern-day (Thompson, R. N., Thompson, C. P., Pelerman, O., Gupta, S., & Obolski, U. 2019), animal movement data is being used for the transmission of pathogen spread (Chaters, G. L., Johnson, P. C. D., Cleaveland, S., Crispell, J., Glanville, W. A. D., Doherty, T., Kao, R. R. 2019). Chaters et al described the utilisation of sparsely routinely collected data for defining the high-dimensional network models. The prediction of environmental modelling could be utilised for the high-resolution climate data resolution and predicting environmental

changes on the outbreak dynamics in the fuzzy and uncertain environment (Shah, D. A., Paul, P. A., Wolf, E. D. D., & Madden, L. V. 2019).

Different Forms of the Modelling Approach

One of the most commonly used epidemiological modelling approaches is compartmental modelling (Hilton, J., & Keeling, M. J. 2019). Among the epidemiological modelling viewpoint, compartmental modelling can be classified into the deterministic or the stochastic. The individuals in the population density are primarily classified on the basis of the infection spread (Kleczkowski, A., Hoyle, A., & Mcmenemy, P. 2019) and the symptom status (Hart, W., Hochfilzer, L., Cunniffe, N., Lee, H., Nishiura, H., & Thompson, R. 2019). Also, Kleczkowski et al (Kleczkowski, A., Hoyle, A., & Mcmenemy, P. 2019) explained the overview of the compartmental modelling attributes which predominantly focusses on the standard SIR model. This model includes the different varieties of the epidemiological creatures like the transmission of the pathogenic entity between the hosts via the vector. The transmission of pathogens from host-host and into the environment is purely uncertain in its behaviour among different entities in the ecosystem comprising plants, animals and humans where pathogens spread from animals to humans in a special case of zoonotic transmission.

It is also understood from the work of Alonso et al. (Alonso, D., Dobson, A., & Pascual, M. (2019), that a complex compartmental modelling of malaria which includes the infection of malaria parasites from the mosquito to the human host systems. Also, Chowell et al. (Chowell, G., Mizumoto, K., Banda, J. M., Poccia, S., & Perrings, C. (2019) described the influence of parasitic transmission from the mosquitoes and an overall effect over the epidemic dynamics. It is also viewed from the disease dynamics that compartmental modelling comprises the common disease of the pathogens among the human, plants and animal species respectively signifying the common host entity in the disease spread. One of the significant examples of the plant transmission of the pathogenic spread includes the cryptic transmission in the plant species where the uncertainty is such a manner that the incidence of the infection is more prominent in the plant species much before the onset of the disease symptoms in the plant species (Rousseau, E., Bonneault, M., Fabre, F., Moury, B., Mailleret, L., & Grognard, F. (2019). It is also reported by several groups that the spread of the Ebola virus diseases in the human systems includes the incorporation of onset of the infectiousness following a non-infectious but symptomatic period (Thompson, R. N., Morgan, O. W., & Jalava, K. (2019). Owing to the intrinsic differences among the different species of the animal, plants and humans, several different approaches of the modelling approaches have been adopted by the researchers. The immobility in the host dynamical movement which

includes the plants to humans dictates the spread of the transmission of pathogens from one location to another. The nature of host governs the movement of the participating pathogens from host-host and to the surrounding interacted environment. The exceptionality of the movement of plant pathogens comprises the airborne spore movement over the longer distances (Severns, P. M., Sackett, K. E., Farber, D. H., & Mundt, C. C. (2019). The distribution in the pathogenic migration from one host to the another involves a spatial distributive profile in the environment. Animal or livestock disease models comprise the different forms of cattle movement (Chaters, G. L., Johnson, P. C. D., Cleaveland, S., Crispell, J., Glanville, W. A. D., Doherty, T.,Kao, R. R. (2019), the spatial mode of disease spread dynamics like the rabies virus, plague virus (Baker, L., Matthiopoulos, J., Müller, T., Freuling, C., & Hampson, K. 2019). Spatial models also involve huanglongbing diseases in citrus plants (Gottwald, T., Luo, W., Posny, D., Riley, T., & Louws, F. 2019). Human population systems also signify the geospatial distribution of the pathogens among the human to human species such as different forms of the contagious spread of infectious viruses among the humans etc. (Thompson, R. N., & Brooks-Pollock, E. (2019). Besides the compartmental modelling of the epidemiological approaches, several other approaches are also considered for the effective evaluation of the disease spread in the interacting environment. Different renewal forms of the models also are crucial modelling indicators for the prevalence of the pathogens and the forecasting of the number of such data enables the different cases of the disease surveillance (Roosa, K., & Chowell, G. 2019). In one of the research reports by Bourhis et al (Bourhis, Y., Gottwald, T., & Bosch, F. V. D. 2019), they have explained the prevalence of the pathogens in a typical outbreak effecting thereby the host species for the susceptible-infected compartmental modelling approach. The optimisation in such disease modelling involves the specific type of host-pathogen interaction and involving a different set of data repository comprises the variability in the modelling approaches in disease modelling in the environment. The dynamics of the epidemic models is also linked by the pathogen sequencing data source for designing an accurate disease modelling reflecting a transmission in the disease spread dynamics which highlights the phylodynamics (Blanquart, F. (2019).In one of the studies mentioned by Lycett et al. (Belser, J. A., Pulit-Penaloza, J. A., & Maines, T. R. 2019), the influenza infection route is explained and evaluated using a phylodynamic method. Such approaches enable to forecast and design several disease outbreaks probabilities in future for the different disease propagation, indicating warning and control mechanism in the prevention of crucial and critical disease system in the environment.

PRELIMINARIES

Definition 2.1: Fuzzy Set: A fuzzy set $\tilde{A}$ is defined as a set of ordered pair $\left(X, \mu_{\tilde{A}}(x)\right)$ where *X* is nonempty universal set and *x*∈*X, A* is the classical set. $\mu_{\tilde{A}}(x): X \to [0,1]$, membership function and $\mu_{\tilde{A}}(x)$ is the grade of membership of *x*∈*X* in $\tilde{A}$.

Definition 2.2: Triangular fuzzy number: A triangular fuzzy number (TFN) is defined as an ordered triplet $\tilde{A} = (a_1, a_2, a_3)$.and its membership function is given by

$$\mu_{\tilde{A}}(x) = \begin{cases} \dfrac{x - a_1}{a_2 - a_1}; & a_1 \le x \le a_2 \\ 1; & x = a_2 \\ \dfrac{a_3 - x}{a_3 - a_2}; & a_2 \le x \le a_3 \\ 0; & otherwise \end{cases}.$$

Definition2.3: Fuzzy function: Let the set of all real numbers and real-valued fuzzy numbers are denoted by $\mathcal{R}$.and $\mathcal{R}_{\mathcal{F}}$.respectively. The function $W: \mathcal{R} \to [0,1]$.s called a fuzzy number valued function if *w* satisfies the following properties

(1) *W* is upper semi continuous.

(2) *W* is fuzzy convex i.e.,

$$W\left(\lambda s_1 + (1-\lambda) s_2\right) \ge min\left\{W(s_1), W(s_2)\right\}.$$

for all $s_1, s_2 \in \mathcal{R}$ and λ∈(0,1).

(3) *W* is normal i.e., ∃ $s_0 \in \mathcal{R}$ such that $W(s_0)=1$

(4) Closure of *supp*(*W*) is compact, where $supp(W) = \left\{s \in \mathcal{R} \middle| W(s) \rangle 0\right\}$.

Definition 2.4: Hukuhara- difference on fuzzy function: Let *E** be the set of all fuzzy function and $\tilde{s}, \tilde{t} \in E^*$. If ∃ a fuzzy number $\tilde{w} \in E^*$ and $\tilde{w}$ satisfy the relation $\tilde{s} = \tilde{w} + \tilde{t}$ then $\tilde{w}$ is said to be the Hukuhara- difference of $\tilde{s}$ and $\tilde{t}$ denoted by $\tilde{w} = \tilde{s} \ominus \tilde{t}$.

Theorem 2.1: Characterization theorem: Let us consider the fuzzy difference equation problem

$$\tilde{x}_{n+1} = \tilde{f}\left(x_n, n\right), \tag{2.1}$$

With initial value

$$\tilde{x}_{n=0} = \tilde{x}_0 \tag{2.2}$$

where $f : E^* \times \mathbb{Z}_{\geq 0} \to E^*$ .such that

(1) The parametric form of the function is

$$\left[f\left(\left(x_n, n\right)\right)\right]_\alpha = \left[\underline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n, \alpha\right), \overline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n, \alpha\right)\right].$$

(2) The functions $\underline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n, \alpha\right)$ and $\overline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n, \alpha\right)$ re taken as continuous functions if for any $\in_1 > 0 \exists$, $\delta_1 > 0$ such that

$$\left|\underline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n\right) - \underline{f}\left(\underline{x}_{n_1}\left(\alpha\right), \overline{x}_{n_1}\left(\alpha\right), n_1\right)\right| < \in_1$$

or all $\alpha \in [0,1]$ with

$$\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n\right) - \left(\underline{x}_{n_1}\left(\alpha\right), \overline{x}_{n_1}\left(\alpha\right), n_1\right) < \delta_1$$

where $\in_2 > 0 \exists$ and $\delta_2 > 0$ such that

$$\left|\overline{f}\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n, \alpha\right) - \overline{f}\left(\underline{x}_{n_2}\left(\alpha\right), \overline{x}_{n_2}\left(\alpha\right), n_2\right)\right| < \in_2 .$$

or all $\alpha \in [0,1]$ with

$$\left(\underline{x}_n\left(\alpha\right), \overline{x}_n\left(\alpha\right), n\right) - \left(\underline{x}_{n_2}\left(\alpha\right), \overline{x}_{n_2}\left(\alpha\right), n_2\right) < \delta_2 .$$

Then the difference equation (2.1) reduces to a system of two difference equation as

$$\underline{x}_{n+1}(\alpha) = \underline{f}\left(\underline{x}_n(\alpha), \overline{x}_n(\alpha), n, \alpha\right), \overline{x}_{n+1}(\alpha) = \overline{f}\left(\underline{x}_n(\alpha), \overline{x}_n(\alpha), n, \alpha\right)$$

With initial conditions

$$\underline{x}_{n=0}(\alpha) = \underline{x}_0(\alpha), \overline{x}_{n=0}(\alpha) = \overline{x}_0(\alpha).$$

Note 2.1: By characterisation theorem, every single fuzzy difference equation is converted into a system of two crisp difference equations. In this paper, we have taken only a single fuzzy difference equation in a fuzzy environment. Hence, the difference equation converted into a pair of a crisp difference equation.

Definition 2.5: Strong and weak solution of fuzzy difference equation: The solutions of difference equation (2.1) with initial condition (2.2) to be regarded as

(1) A strong solution if $\underline{x}_n(\alpha) \le \overline{x}_n(\alpha)$ for every α∈[0,1] and

$$\frac{\partial}{\partial\alpha}\left[\underline{x}_n(\alpha)\right] > 0, \frac{\partial}{\partial\alpha}\left[\overline{x}_n(\alpha)\right] < 0$$

for every α∈[0,1].

(2) A weak solution if $\underline{x}_n(\alpha) \ge \overline{x}_n(\alpha)$.for every α∈[0,1] and

$$\frac{\partial}{\partial\alpha}\left[\underline{x}_n(\alpha)\right] < 0, \frac{\partial}{\partial\alpha}\left[\overline{x}_n(\alpha)\right] > 0$$

for every α∈[0,1].

Definition 2.6: Let *p,q* are fuzzy numbers and

$$[p]_\alpha = \left[\underline{p}(\alpha), \overline{p}(\alpha)\right], [q]_\alpha = \left[\underline{q}(\alpha), \overline{q}(\alpha)\right]$$

for all $\alpha \in (0,1]$. Then metric on fuzzy number space is defined as

$$d(p,q) = sup \max_{\alpha \in (0,1)} \left\{ \left| \underline{p}(\alpha) - \underline{q}(\alpha) \right|, \left| \overline{p}(\alpha) - \overline{q}(\alpha) \right| \right\}.$$

Note 2.2: Maybe somewhere strictly the strong and weak solution strategy may not occur. In this case for a particular time interval or particular interval of α the strong and weak solution both exist The scenario where the strong or weak both solutions not occur, then we will call them non recommended fuzzy solution. We recommended for taking strong solution cases.

DIFFERENCE EQUATION WITH FUZZY VARIABLE

Definition 3.1: A difference equation (sometimes called a recurrence relation) is an equation that relates consecutive terms of a sequence of numbers.
A q the order linear difference equation can be expressed in the form

$$x_{n+q} = d_1 x_{n+q-1} + d_2 x_{n+q-2} + \ldots + d_q x_n + b_n . \tag{3.1}$$

where $d_1, d_2, \ldots, d_q$ and b_n are known constant.
If $b_n = 0$, or all n the equation (3.1) is homogeneous difference equation and non-homogeneous difference equation if $b_n \neq 0$.
b_n is called the forcing factor.

Theorem 3.1: Let $m \in \mathbb{N}, m \geq 2$ linear inhomogeneous system of m first-order difference equations are given by in matrix form as

$$X_{n+1} = AX_n + B . \tag{3.2}$$

where,

$$X_n = \left(X_n^1, X_n^2, \ldots, X_n^m \right)^T, A = \left(a_{ij} \right)_{m \times m}, i, j = 1, 2, \ldots, m$$

and $B = (b_1, b_2, \ldots, b_n)^T$. Then the solution of equation (3.3) can be written as

$$X_n = A^n X_0 + \sum_{j=0}^{n-1} A^j B, n \in \mathbb{N} \tag{3.3}$$

The above difference equation (3.1) is called fuzzy difference equation if

(i) The initial condition or conditions are fuzzy number
(ii) The coefficient or coefficients are fuzzy numbers
(iii) The initial conditions and coefficient or coefficients are fuzzy numbers

DISCRETE SUSCEPTIBLE INFECTED (SI) MODEL

Let us assume the following definition for two variables as:

S_n is the number in the population susceptible after period n.
I_n is the number of infected after period n.

Here I_n is increased by the number of susceptible people who come into contact with infected people and catch the disease: aS_nI_n and S_n decreased at the same rate a. We define a as the rate at which the disease is spread or the transmission n coefficient. We realize this is a probabilistic coefficient. We will assume initially that this rate is a constant value that can be found from the initial conditions.

Let's illustrate as follows: Assume we have a population of N student residing in the dorms. Our nurse found five students reporting to the infirmary initially: $I_{n=0} = I_0$ and $S_{n=0} = S_0$. After one week, the total number infected with the flu is $I_{n=1} = I_1$. We compute a as follows:

$$I_{n=0} = I_0,\ I_{n=1} = I_{n=0} + aI_{n=0}S_{n=0}.$$

From the above equation, we can easily find the value of a.

Let's consider S_n which is decreased only by the number that becomes infected. We may use the same rate a as before to obtain the model:

$$S_{n+1} = S_n - aS_nI_n.$$

So the system of difference equation of the model is

$$S_{n+1} = S_n - aS_nI_n,\ I_{n+1} = I_n + aI_nS_n.$$

With initial condition $I_{n=0} = I_0$ and $S_{n=0} = S_0$.

Table 1. Solution of different case

	Number of Boarders	Infected people	Infected people after the first week	a the rate at which the disease is spread	Collection of different values of a
Hostel A	250	15	25	0.0028	(0.0007, 0.0028, 0.0207)
Hostel B	1500	50	100	0.0007	
Hostel C	150	5	20	0.0207	

Finding the Value of *a* (the Rate at Which the Disease is Spread) From Some Observations

Suppose in an institute campus there is four Hostel: Hostel A, Hostel B, Hostel C and Hostel D. The numbers of boarders of the above-mentioned hostel are 250, 1500, 150respectively. An infectious disease affected the first three hostels and the number of infected people is as follows: 15, 50, 5. Suppose some week later it is found that the infected people in hostel D is 20. Now we also predict the number of susceptible and infected people just in the next week when found the infected people in hostel D.

Now we wish to predict the number of an infected person in hostel D if the infectious disease spread continuously in the campus. The total scenario is shown in the following table:

FUZZIFICATION OF DISCRETE SI MODEL

Considering the above SI model for the discrete system the above observation if we can take a as a fuzzy number in the model (since the above hostel problem we see that the value of a varies)

$$S_{n+1} = S_n - \tilde{a}S_nI_n, I_{n+1} = I_n + \tilde{a}I_nS_n.$$

With initial condition $I_{n=0} = I_0$ and $S_{n=0} = S_0$.

Solution: Using the concept of Characterisation theorem we get a system in the crisp system as follows:

$$S_{L,n+1}(\alpha) = S_{L,n}(\alpha) - a_R(\alpha) S_{R,n}(\alpha) I_{R,n}(\alpha)$$

$$S_{R,n+1}(\alpha) = S_{R,n}(\alpha) - a_L(\alpha) S_{L,n}(\alpha) I_{L,n}(\alpha)$$

$$I_{L,n+1}(\alpha) = I_{L,n}(\alpha) + a_L(\alpha) I_{L,n}(\alpha) S_{L,n}(\alpha)$$

$$I_{R,n+1}(\alpha) = I_{R,n}(\alpha) + a_R(\alpha) I_{R,n}(\alpha) S_{R,n}(\alpha).$$

With initial condition

$$I_{L,n=0}(\alpha) = I_0, I_{R,n=0}(\alpha) = I_0, S_{L,n=0}(\alpha) = S_0,$$

and $S_{R,n=0}(\alpha) = S_0$. Here

$$\left(S_{L,n+1}(\alpha), S_{R,n+1}(\alpha)\right), \left(I_{L,n+1}(\alpha), I_{R,n+1}(\alpha)\right)$$

is the α cut of the fuzzy solution $\tilde{S}_{n+1}$ and $\tilde{I}_{n+1}$ respectively.

NUMERICAL SIMULATION

Now we wish to predict the number of susceptible and infected population of Hostel D, where the number of susceptible initially is $S_{n=0} = 480$ and infected is $I_{n=0} = 20$.

For predicting the solution we have to use the parameter value of *a*. From the three hostel cases, we see that the value differs from each case. So we cannot take any of one values. From three data set of *a* we construct a fuzzy number $\tilde{a} = (0.0007, 0.0028, 0.0207)$ whose α cut is (0.0007+0.0021α, 0.0207–0.0179α).

Now from section 5 concepts the corresponding crisp system of difference equation are as follows:

$$S_{L,n+1}(\alpha) = S_{L,n}(\alpha) - (0.0207 - 0.0179\alpha) S_{R,n}(\alpha) I_{R,n}(\alpha)$$

$$S_{R,n+1}(\alpha) = S_{R,n}(\alpha) - (0.0007 + 0.0021\alpha) S_{L,n}(\alpha) I_{L,n}(\alpha)$$

Table 2. Solution for different α

n=0	$S_{L,n+1}(\pm)$	$S_{A,L,n+1}(\pm)$	$S_{R,n+1}(\pm)$	$S_{A,R,n+1}(\pm)$	$I_{L,n+1}(\pm)$	$I_{A,L,n+1}(\pm)$	$I_{R,n+1}(\pm)$	$I_{A,R,n+1}(\pm)$
α=0	281.28	281	473.28	473	26.72	27	218.72	219
α=0.1	298.46	298	471.26	471	28.73	29	201.53	202
α=0.2	315.64	315	469.24	479	30.75	31	184.35	185
α=0.3	332.83	332	467.23	467	32.76	33	167.16	168
α=0.4	350.01	350	465.21	465	34.78	35	149.98	150
α=0.5	367.20	367	463.20	463	36.79	37	132.80	133
α=0.6	384.38	384	461.18	461	38.81	39	115.61	116
α=0.7	401.56	401	459.16	459	40.83	41	98.43	99
α=0.8	418.75	418	457.15	457	42.84	43	81.24	82
α=0.9	435.93	435	455.13	455	44.86	45	64.06	65
α=1	453.12	453	453.12	453	46.88	47	46.88	47

$$I_{L,n+1}(\alpha) = I_{L,n}(\alpha) + (0.0007 + 0.0021\alpha) I_{L,n}(\alpha) S_{L,n}(\alpha)$$

$$I_{R,n+1}(\alpha) = I_{R,n}(\alpha) + (0.0207 - 0.0179\alpha) I_{R,n}(\alpha) S_{R,n}(\alpha)$$

With initial condition

$$I_{L,n=0}(\alpha) = 20, I_{R,n=0}(\alpha) = 20, S_{L,n=0}(\alpha) = 480$$

and $S_{R,n=0}(\alpha) = 480$.

In the above table we consider

$$\left(S_{A,L,n+1}(\alpha), S_{A,R,n+1}(\alpha)\right) \text{and } \left(I_{A,L,n+1}(\alpha), I_{A,R,n+1}(\alpha)\right)$$

as corresponding approximate integer fuzzy interval of different α of the fuzzy solution of $\tilde{S}_n$ and $\tilde{I}_n$ respectively.

Remarks 6.1: From the figure and table we see that $S_{A,L,n+1}(\alpha)$, $I_{A,L,n+1}(\alpha)$ are increasing function and $S_{A,R,n+1}(\alpha)$, $I_{A,R,n+1}(\alpha)$ decreasing function. So in both of the cases, the strong solution exists.

Figure 1. Fuzzy solution of $\tilde{S}_n$

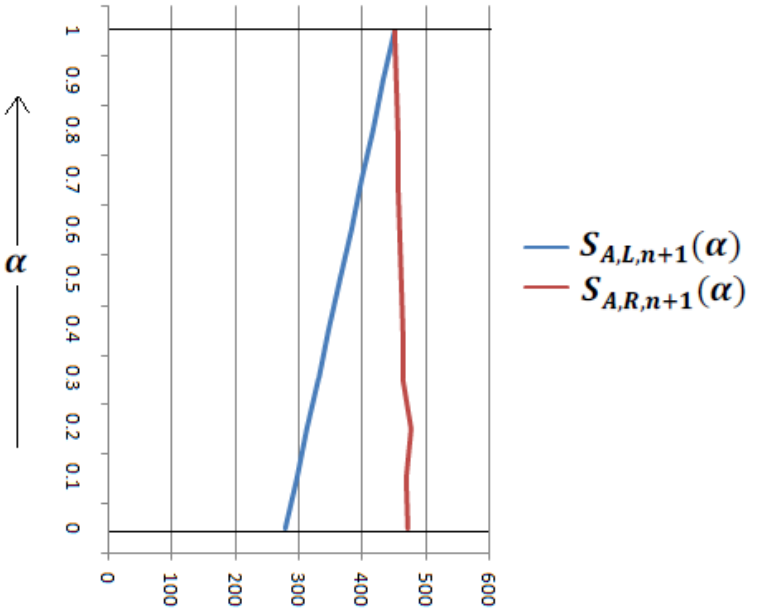
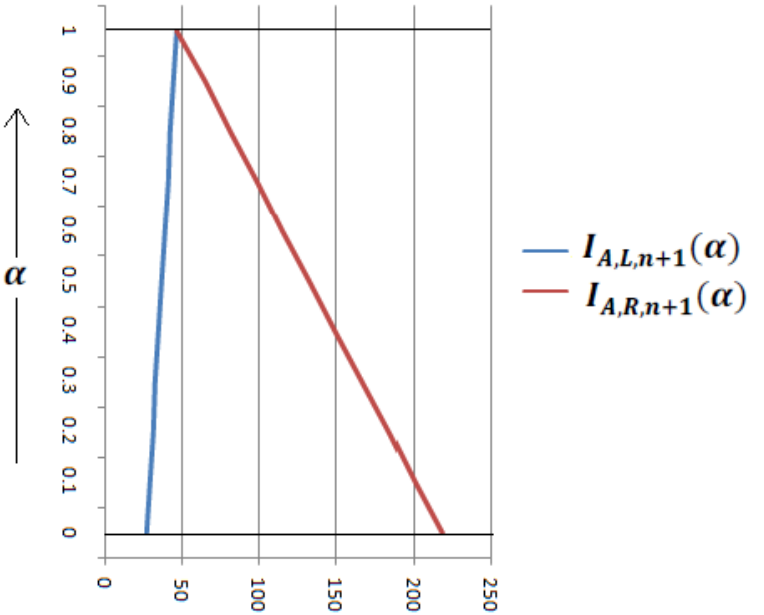


Figure 2. Fuzzy solution of $\tilde{I}_n$



CONCLUSION

In this chapter, we have concentrated on the solution of the discrete model of the spread of infectious disease modelling, which is subsequently followed by a characteristic SI model in a fuzzy environment. Due to system behaviour of the model in a discrete system, the concept of the difference equation arrives. We have also introduced the fuzzy discrete SI model for the first time. With a real-life example, we have shown the importance of fuzziness in modelling the spread of infectious diseases. Moreover, it is concluded that the idea is very helpful for those who deal with uncertainty modelling in the discrete system, thereby having a close and accurate predictive analysis of the incidence of disease prevalence in the environment.

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Chapter 7
Stability Analysis of Co-Infection of Malaria-Dengue

Nisha Sheoran
Department of Mathematics, Gujarat University, Ahmedabad, India

Moksha H. Satia
Department of Mathematics, Gujarat University, Ahmedabad, India

ABSTRACT

Dengue and malaria most commonly occur in tropical and sub-tropical areas. Dengue is a viral infection in a human being caused by a bite of a female aedes mosquito whereas malaria is caused by plasmodium parasite transmitted by a bite of infected mosquito. In this chapter, a mathematical model of co-infection of malaria and dengue is described by deterministic system of non-linear ordinary differential equations. This system considers the force of infection which is applied to dengue susceptible individuals. Moreover, two sub-models, namely malaria-only and dengue-only, are also constructed to study the transmission dynamics. Basic reproduction number is calculated for these models to investigate the existence of the models. The system is proved to be locally and globally stable at its equilibrium points. Stability of these models is also shown through numerical simulation.

INTRODUCTION

Now-a-days, due to environmental conditions and human behavior, population is prone to many viral infectious diseases. Most commonly occurring diseases in tropical and sub-tropical areas are due to mosquito bites. Some of the vector borne diseases are malaria, dengue, chikungunya, zika fever, filariasis etc. Among them malaria and

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dengue are most commonly prevailing diseases which leads to death also. Malaria is an infectious parasitic disease transmitted by *Anopheles* mosquito. There are five parasite species that causes malaria in humans, two of its major threating species are P. falciparum and P. vivax. Some of its symptoms are pain in abdomen, muscles, fatigue, sweating, shivering, vomiting etc. According to world malaria report 2013 published by WHO, malaria is a leading cause of premature death, particularly in children under the age of five, with an estimate of 207 million cases and more than half a million deaths in 2012. Also, death toll reached to one million as of 2018 according to the American mosquito control association. The other tropical disease Dengue which is a viral disease transmitted by the mosquitoes *Aedes aegypti* and *Aedes albopictus*, which are found throughout the world. Dengue was first recognised in 1950's. Around 2.5 billion people, or 40% of the world's population, live in areas where there is a risk of dengue transmission. Some of the dengue symptoms include pain in abdomen and back of the eyes, nausea, skin rashes, vomiting, mild bleeding etc. Symptoms usually appear 4 to 7 days after the mosquito bite and typically last 3 to 10 days. Both the disease starts with some common symptoms for example headache, intense muscle pain, weakness. Which makes it difficult to identify the disease. Therefore, one should go for test of both the diseases.

Mathematical modeling is among the best ways to study dynamics of transmission of many problems. Kermack and McKendrick established basic foundation of mathematical modeling by developing *SIR* model in 1927. Martcheva in her book "mathematical modeling in epidemiology" described various methods and strategies to solve infectious disease models. The basic Mathematical model *SIS* of malaria transmission dynamics consisting of two compartments (Ross, 1911) and its modified model by Macdonald, (1957) which together known as Ross-Macdonald model of malaria transmission was developed which laid down the base for constructing future models. A review related to malaria infection due to vectors were discussed containing different models covering every criterion such as age, environment, immunity, socio-economic etc. by Mandal *et al*., (2011). Various dengue transmission models also exist. One of dengue transmission model considering severe DHF compartment was studied by Nuraini *et al.*, (2007). Global stability of dengue model with the help of saturation and bilinear incidence was discussed by Cai *et al*., (2009). Rodrigues *et al*. considered both human and mosquitos' population and applied control parameter (insecticide) in order to fight against mosquitoes and they have also concluded usage of insecticide to be done in night. Co-infection model has also been studied by some of the researchers. for example, Stability Analysis of Zika – Malaria Co-infection Model with its sub-models zika only, malaria-only for Malaria Endemic Region was studied (Mensah *et al*.,2018). Mathematical model considering both human population and mosquitos population in dengue-chikungunya co-infection is studied by Aldila and Agustin, 2018. Mathematical analysis for co-infection of

HIV-Malaria which showed their co-existence when their reproduction number exceeds unity was contributed by Mukandavire *et al*., (2009) Salam *et al.* have reviewed the literature associated with the co-infection of three vector borne diseases malaria-dengue-chikungunya. One of the co-infection malaria-dengue studied by them showed that all possible combination of co-infection of these three diseases were seen in only India and Nigeria.

Construction of this chapter is as follows section 2 describes mathematical model of malaria-dengue co-infection with notations and parametric values and formation of system of malaria only, dengue only sub models with their equilibrium points. section 3 provides basic reproduction number for all the models. In section 4, stability analysis is worked out. In section 5 the observations are exhibited from the numerical analysis. Section 6 concludes the chapter.

Mathematical Modeling

Mathematical models can be considered to be very useful in studying disease dynamics. A mathematical model of malaria-dengue co-infection is built along with two of its sub model malaria-only and dengue-only. Co-infection model has 7 epidemiological stages, class of susceptible denoted by S, next is Exposed class also read as latent period which is the duration for which infected individuals stays before becoming infectious, this model has two exposed classes, E_M number of individuals exposed to malaria and E_D consisting of individuals exposed to dengue. Similarly two infectious classes, namely I_M, individual infectious to malaria only are in this class and I_D consist of individual infectious to dengue only. Next is co-infectious stage denoted by I_{MD}, individual infected by malaria-dengue belong to this stage. Recovered individuals are in the last stage R. The chapter also considers force of infection λ_D defined as rate at which susceptible acquire dengue.

Notations and parametric values used in the formulation of dynamical system of malaria-dengue co-infectious model are given in the following Table 1.

Assumptions: for simplicity the following assumptions are made

(1) vectors (mosquitoes) are responsible for spread of both diseases, though vector population is not taken in account and constant vector population is considered.
(2) Disease spread is considered only in Human population.
(3) New recruitment is considered as infected individual's contact with susceptible individual through mosquito bite.

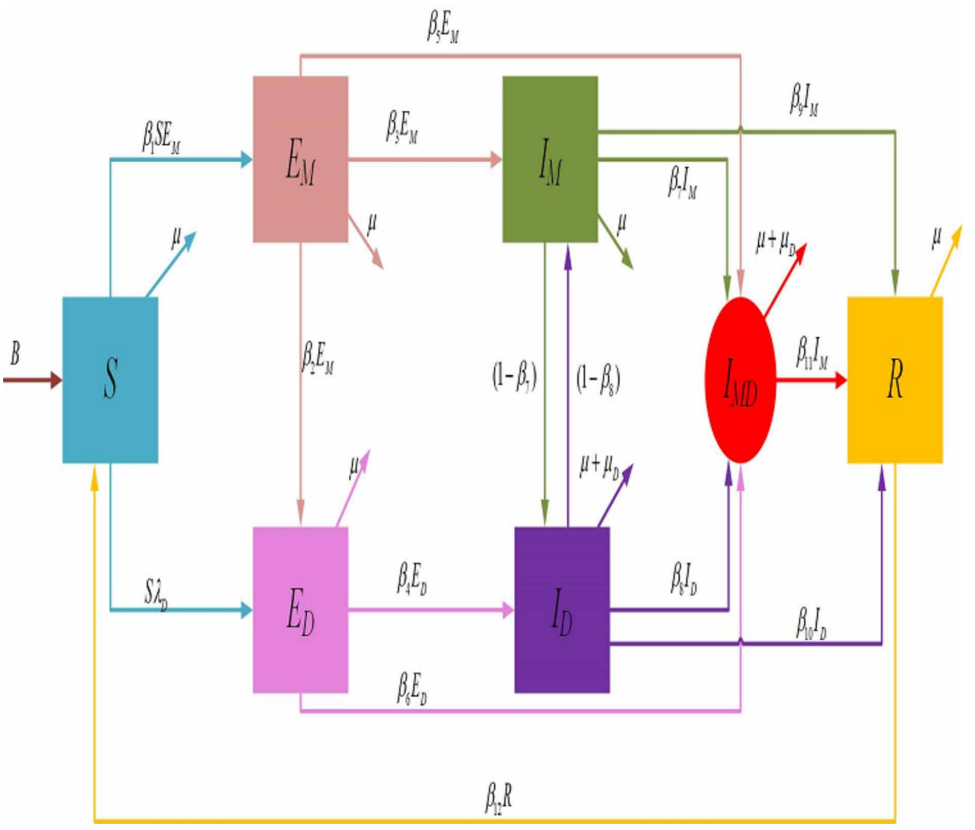
Table 1. Parametric definitions and its values

Notations	Description	Parametric values
$N(t)$	Number of Individual at any instant of time	100
B	Birth rate	0.10
β_1	Transmission rate from susceptible to malaria exposed individual	0.20
β_2	Rate with which individual exposed to malaria joins exposed dengue class	0.20
β_3	Rate at which individuals exposed to malaria becomes infectious	0.50
β_4	Rate at which individuals exposed to dengue becomes infectious	0.2
β_5	Rate at which individual exposed to malaria moves to co-infectious class	0.34
β_6	Rate at which individual exposed to dengue moves to co-infectious class	0.23
β_7	Rate at which individual infectious to malaria moves to co-infectious class	0.2
β_8	Rate at which individual infectious to dengue moves to co-infectious class	0.40
β_9	Rate at which individual infectious to malaria gets recovered	0.95
β_{10}	Rate at which individual infectious to dengue gets recovered	0.9
β_{11}	Rate at which co-infectious individual gets recovered	0.70
β_{12}	Rate at which recovered individual joins susceptible class.	0.20
β_D	Rate of dengue susceptible individual	0.10
η_1	Probability of new births exposed to malaria	0.10
η_2	Probability of new births infectious to malaria	0.15
η_3	Probability of new births infectious to both disease	0.30
μ	Natural Death rate	0.10
μ_D	Rate at which dengue infected deaths occur	0.05
γ	Transmission rate from susceptible to dengue exposed individual	0.15

Co-Infectious Model

The Figure 1 gives rise to system of non-linear ordinary differential equations of Malaria-Dengue co-infectious model.

Figure 1. Describes movement of individuals in different compartments



$$\frac{dS}{dt} = B + \beta_{12}R - \beta_1 SE_M - S\lambda_D - \mu S$$

$$\frac{dE_M}{dt} = \beta_1 SE_M - (\mu + \beta_2 + \beta_3 + \beta_5)E_M$$

$$\frac{dE_D}{dt} = S\lambda_D + \beta_2 E_M - (\mu + \beta_4 + \beta_6)E_D$$

$$\frac{\mathrm{dI}_M}{dt} = \beta_3 E_M + (1-\beta_8)\,\mathrm{I}_D - (1+\mu+\beta_9)I_M$$

$$\frac{dI_D}{dt} = \beta_4 E_D + (1-\beta_7) I_M - (1+\mu+\mu_D+\beta_{10}) I_D$$
$$\frac{dI_{MD}}{dt} = \beta_5 E_M + \beta_6 E_D + \beta_7 I_M + \beta_8 I_D - (\mu+\mu_D+\beta_{11}) I_{MD} \quad (1)$$
$$\frac{dR}{dt} = \beta_9 I_M + \beta_{10} I_D - (\mu+\beta_{12}) R + \beta_{11} I_{MD}$$

where,

$$N(t) = S(t) + \mathrm{E}_M(t) + E_D(t) + I_M(t) + I_D(\mathrm{t}) + \mathrm{I}_{MD}(\mathrm{t}) + \mathrm{R(t)}$$

and force of infection $\lambda_D = \dfrac{\beta_D(\eta_1 E_M + \eta_2 I_M + \eta_3 I_{MD})}{N}$

Adding all the differential equations of co-infectious model (1), we get,

$$\frac{dN}{dt} \le B - \mu(\mathrm{S} + \mathrm{E}_M + E_D + I_M + I_D + I_{MD} + R) \ge 0$$

Hence, $\dfrac{dN}{dt} \le B - \mu N$, So that $\lim_{t\to\infty} \sup \mathrm{N} \le \dfrac{B}{\mu}$. Then, Feasible Region for the system is defined as

$$\Lambda_{MD} = \left\{ \begin{array}{l} (S, E_M, E_D, I_M, I_D, I_{MD}, R); S + E_M + E_D + I_M + I_D + I_{MD} + R \le \dfrac{B}{\mu}, \\ S \ge 0, E_M \ge 0, E_D \ge 0, I_M \ge 0, I_D \ge 0, I_{MD} \ge 0, R \ge 0 \end{array} \right\}$$

Assume,

$$A_1 = \mu + \beta_2 + \beta_3 + \beta_5, A_2 = \mu + \beta_4 + \beta_6, A_3 = 1 + \mu + \beta_9,$$
$$A_4 = 1 + \mu + \mu_D + \beta_{10}, A_5 = \mu + \mu_D + \beta_{12}, A_6 = \mu + \beta_{12}$$

Now, modified system is

$$\frac{dS}{dt} = B + \beta_{12}R - \beta_1 SE_M - S\lambda_D - \mu S$$
$$\frac{dE_M}{dt} = \beta_1 SE_M - A_1 E_M$$
$$\frac{dE_D}{dt} = S\lambda_D + \beta_2 E_M - A_2 E_D$$

$$\frac{dI_M}{dt} = \beta_3 E_M + (1-\beta_8) I_D - A_3 I_M$$
$$\frac{dI_D}{dt} = \beta_4 E_D + (1-\beta_7) I_M - A_4 I_D$$
$$\frac{dI_{MD}}{dt} = \beta_5 E_M + \beta_6 E_D + \beta_7 I_M + \beta_8 I_D - A_5 I_{MD} \quad (2)$$
$$\frac{dR}{dt} = \beta_9 I_M + \beta_{10} I_D + \beta_{11} I_{MD} - A_6 R$$

System (1) and (2) are equivalent. Hence Λ_{MD} is the feasible region for the system (2).

Solving system (2) we get three sets of equilibrium points as follows:

i. Disease-free equilibrium point $E^0\left(\frac{B}{\mu}, 0, 0, 0, 0, 0, 0\right)$

ii. ii. Malaria free equilibrium point $E^1\left(S^1, E_M^1, E_D^1, I_M^1, I_D^1, I_{MD}^1, R^1\right)$ where,

$$S^1 = A_2 A_5 N(A_3 A_4 - (1-\beta_7)(1-\beta_8))/\beta_D (A_5\beta_4(1-\beta_8)\eta_2$$
$$+(\beta_6(A_3 A_4 - (1-\beta_7)(1-\beta_8)) + \beta_4(A_3\beta_8 - \beta_7\beta_8 + \beta_7))\eta_3)$$

$$E_M^1 = 0$$

$$E_D^1 = \{A_5 A_6(A_3 A_4 - (1-\beta_7)(1-\beta_8))\{\beta_D B\beta_4(A_5\eta_2(1-\beta_8) + \eta_3(A_3\beta_8 - \beta_7\beta_8 + \beta_7)) + (A_3 A_4 - (1-\beta_7)(1$$
$$-\beta_8))(\beta_6 B\beta_D\eta_3 - A_2 A_5 N\mu)\}\}/\beta_D\{[(A_3 A_4 - (1-\beta_7)(1-\beta_8))(A_2 A_5 A_6 - \beta_6\beta_{11}\beta_{12}) + \beta_4\beta_{12}(\beta_9 A_5$$
$$+\beta_7\beta_{11})(\beta_8 - 1) - \beta_4\beta_{12}\,\mathrm{A_3}(\mathrm{A_5}\,\beta_{10} + \beta_8\beta_{11})]\beta_4 A_5(1-\beta_8)\eta_2 + [(A_3 A_4 - (1-\beta_7)(1-\beta_8))\{(A_3 A_4$$
$$-(1-\beta_7)(1-\beta_8))\beta_6(A_2 A_5 A_6 - \beta_6\beta_{11}\beta_{12}) + (A_3\beta_8 - \beta_7\beta_8 + \beta_7)\beta_4(A_2 A_5 A_6 - 2\beta_6\beta_{11}\beta_{12})$$
$$+\beta_4\beta_6\beta_{12}A_5(\beta_8\beta_9 - A_3\beta_{10} - \beta_9)\} + \beta_4^2\beta_{12}[(\beta_8 - 1)((\beta_{11}\beta_7 + \beta_9 A_5)((1-\beta_8)\beta_7 + \beta_8 A_3)) + (\beta_7\beta_8$$
$$-\beta_7 - A_3\beta_8)A_3(\beta_{10}A_5 + \beta_8\beta_{11})]]\eta_3\}$$

$$I_M^1 = A_5A_6\beta_4(1-\beta_8)\{(\beta_D B\beta_4(A_5\eta_2(1-\beta_8)+\eta_3(A_3\beta_8-\beta_7\beta_8+\beta_7))+(A_3A_4-(1-\beta_7)(1-\beta_8))(\beta_6B\beta_D\eta_3 -A_2A_5N\mu))\}/\beta_D\{[(A_3A_4-(1-\beta_7)(1-\beta_8))(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+\beta_4\beta_{12}(\beta_9A_5+\beta_7\beta_{11})(\beta_8-1) -\beta_4\beta_{12}\,\mathrm{A}_3(\mathrm{A}_5\,\beta_{10}+\beta_8\beta_{11})]\beta_4A_5(1-\beta_8)\eta_2+[(A_3A_4-(1-\beta_7)(1-\beta_8))\{(A_3A_4-(1-\beta_7)(1 -\beta_8))\beta_6(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+(A_3\beta_8-\beta_7\beta_8+\beta_7)\beta_4(A_2A_5A_6-2\beta_6\beta_{11}\beta_{12})+\beta_4\beta_6\beta_{12}A_5(\beta_8\beta_9-A_3\beta_{10} -\beta_9)\}+\beta_4^2\beta_{12}[(\beta_8-1)((\beta_{11}\beta_7+\beta_9A_5)((1-\beta_8)\beta_7+\beta_8A_3))+(\beta_7\beta_8-\beta_7-A_3\beta_8)A_3(\beta_{10}A_5+\beta_8\beta_{11})]]\eta_3\}$$

$$I_D^1 = A_3A_5A_6\beta_4\{(\beta_D B\beta_4(A_5\eta_2(1-\beta_8)+\eta_3(A_3\beta_8-\beta_7\beta_8+\beta_7))+(A_3A_4-(1-\beta_7)(1-\beta_8))(\beta_6B\beta_D\eta_3 -A_2A_5N\mu))\}/\beta_D\{[(A_3A_4-(1-\beta_7)(1-\beta_8))(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+\beta_4\beta_{12}(\beta_9A_5+\beta_7\beta_{11})(\beta_8-1) -\beta_4\beta_{12}\,\mathrm{A}_3(\mathrm{A}_5\,\beta_{10}+\beta_8\beta_{11})]\beta_4A_5(1-\beta_8)\eta_2+[(A_3A_4-(1-\beta_7)(1-\beta_8))\{(A_3A_4-(1-\beta_7)(1 -\beta_8))\beta_6(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+(A_3\beta_8-\beta_7\beta_8+\beta_7)\beta_4(A_2A_5A_6-2\beta_6\beta_{11}\beta_{12})+\beta_4\beta_6\beta_{12}A_5(\beta_8\beta_9-A_3\beta_{10} -\beta_9)\}+\beta_4^2\beta_{12}[(\beta_8-1)((\beta_{11}\beta_7+\beta_9A_5)((1-\beta_8)\beta_7+\beta_8A_3))+(\beta_7\beta_8-\beta_7-A_3\beta_8)\mathrm{A}_3(\beta_{10}A_5+\beta_8\beta_{11})]]\eta_3\}$$

$$I_{MD}^1 = A_6(\beta_6(A_3A_4-(1-\beta_7)(1-\beta_8))+\beta_4(A_3\beta_8-\beta_7\beta_8+\beta_7))\{(\beta_D B\beta_4(A_5\eta_2(1-\beta_8)+\eta_3(A_3\beta_8-\beta_7\beta_8+\beta_7)) +(A_3A_4-(1-\beta_7)(1-\beta_8))(\beta_6B\beta_D\eta_3-A_2A_5N\mu))\}/\beta_D\{[(A_3A_4-(1-\beta_7)(1-\beta_8))(A_2A_5A_6-\beta_6\beta_{11}\beta_{12}) +\beta_4\beta_{12}(\beta_9A_5+\beta_7\beta_{11})(\beta_8-1)-\beta_4\beta_{12}\,\mathrm{A}_3(\mathrm{A}_5\,\beta_{10}+\beta_8\beta_{11})]\beta_4A_5(1-\beta_8)\eta_2+[(A_3A_4 -(1-\beta_7)(1-\beta_8))\{(A_3A_4-(1-\beta_7)(1-\beta_8))\beta_6(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+(A_3\beta_8-\beta_7\beta_8+\beta_7)\beta_4(A_2A_5A_6 -2\beta_6\beta_{11}\beta_{12})+\beta_4\beta_6\beta_{12}A_5(\beta_8\beta_9-A_3\beta_{10}-\beta_9)\}+\beta_4^2\beta_{12}[(\beta_8-1)((\beta_{11}\beta_7+\beta_9A_5)((1-\beta_8)\beta_7+\beta_8A_3)) +(\beta_7\beta_8-\beta_7-A_3\beta_8)\mathrm{A}_3(\beta_{10}A_5+\beta_8\beta_{11})]]\eta_3\}$$

$$R^1 = (\beta_6\beta_{11}(A_3A_4-(1-\beta_7)(1-\beta_8))+\beta_4\,\mathrm{A}_5(\beta_{10}\,\mathrm{A}_3-\beta_8\beta_9+\beta_9)+\beta_4\beta_{11}(\mathrm{A}_3\,\beta_8-\beta_7\beta_8 +\beta_7))\{(\beta_D B\beta_4(A_5\eta_2(1-\beta_8)+\eta_3(A_3\beta_8-\beta_7\beta_8+\beta_7))+(A_3A_4-(1-\beta_7)(1-\beta_8))(\beta_6B\beta_D\eta_3 -A_2A_5N\mu))\}/\beta_D\{[(A_3A_4-(1-\beta_7)(1-\beta_8))(\mathrm{A}_2\mathrm{A}_5\mathrm{A}_6-\beta_6\beta_{11}\beta_{12})+\beta_4\beta_{12}(\beta_9A_5+\beta_7\beta_{11})(\beta_8-1) -\beta_4\beta_{12}\,\mathrm{A}_3(\mathrm{A}_5\,\beta_{10}+\beta_8\beta_{11})]\beta_4\,\mathrm{A}_5(1-\beta_8)\eta_2+[(A_3A_4-(1-\beta_7)(1-\beta_8))\{(A_3A_4-(1-\beta_7)(1 -\beta_8))\beta_6(A_2A_5A_6-\beta_6\beta_{11}\beta_{12})+(A_3\beta_8-\beta_7\beta_8+\beta_7)\beta_4(A_2A_5A_6-2\beta_6\beta_{11}\beta_{12})+\beta_4\beta_6\beta_{12}A_5(\beta_8\beta_9-A_3\beta_{10} -\beta_9)\}+\beta_4^2\beta_{12}[(\beta_8-1)((\beta_{11}\beta_7+\beta_9A_5)((1-\beta_8)\beta_7+\beta_8A_3))+(\beta_7\beta_8-\beta_7-A_3\beta_8)\mathrm{A}_3(\beta_{10}A_5+\beta_8\beta_{11})]]\eta_3\}$$

iii. iii. Endemicequilibriumpoint $E^*\left(S^*,E_M^*,E_D^*,I_M^*,I_D^*,I_{MD}^*,R^*\right)$ where, $S^*=A_1/\beta_1$

$$E_M^* = A_6\{(A_1\mu - B\beta_1)[(A_3A_4 - (1-\beta_7)(1-\beta_8))(A_2A_5N\beta_1 - \beta_D\beta_6A_1) - (\beta_D\beta_4A_1((1-\beta_8)\beta_4A_5\eta_2 + (\beta_8A_3 - \beta_7\beta_8 + \beta_7)\eta_3))]\}/\beta_1\{\{\beta_DA_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1+\eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1-\beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5 + \beta_8\beta_{11})((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)]\}$$

$$E_D^* = A_6((A_1\mu - B\beta_1)(\beta_DA_1\{((A_3A_4 - (1-\beta_7)(1-\beta_8))(A_5\eta_1 + A_5A_4\beta_3\eta_2 + \beta_5\eta_3)) + \beta_3(\beta_7A_4 - \beta_7\beta_8 + \beta_7)\}) + \beta_1\beta_2A_5N)/\beta_1\{\{\beta_DA_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1+\eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1-\beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5$$

$$I_M^* = A_6(A_1\mu - B\beta_1)\{(1-\beta_8)(\beta_D\beta_4A_1(A_5\eta_1 + \beta_5\eta_3) + \beta_2\beta_4A_5N\beta_1) - \beta_DA_1\eta_3\beta_3(\beta_6A_4 + \beta_4\beta_8) + A_5N\beta_1\beta_3A_2A_4\}/\beta_1\{\{\beta_DA_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1+\eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1-\beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5 + \beta_8\beta_{11})((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)]\}$$

$$I_D^* = A_6(A_1\mu - B\beta_1)\{\beta_DA_1(A_3\beta_4(A_5\eta_1 + \beta_5\eta_3) + \beta_3\beta_4(\beta_7\eta_3 + A_5\eta_2) + \beta_3\beta_6\eta_3(\beta_7 - 1)) + N\beta_1A_5(A_2\beta_3(1-\beta_7) + \beta_2\beta_4A_3)\}/\beta_1\{\{\beta_DA_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1+\eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1-\beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5 + \beta_8\beta_{11})((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)]\}$$

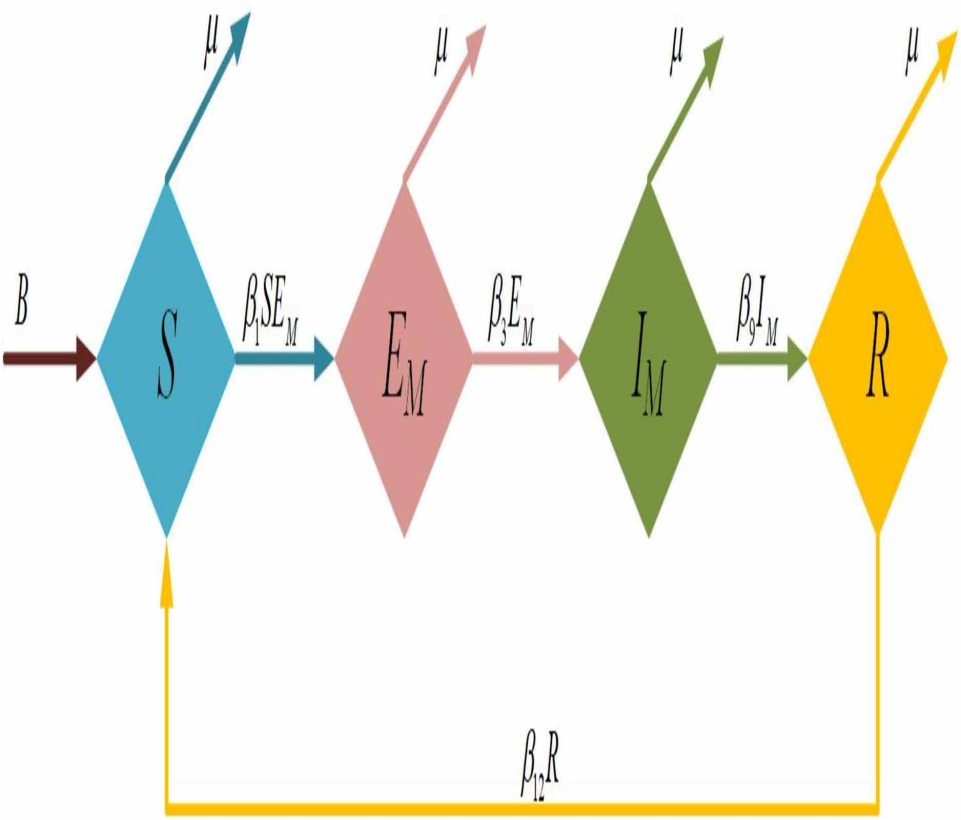
$$I^*_{MD} = A_6(A_1\mu - B\beta_1)\{(A_3A_4 - (1-\beta_7)(1-\beta_8)(N\beta_1(A_2A_5 + \beta_2\beta_6) + \beta_D A_1\eta_1\beta_6) + (\beta_8A_3 - \beta_7\beta_8 + \beta_7)(\beta_D\beta_4A_1\eta_1 + N\beta_1\beta_2\beta_4) + (\beta_3(\beta_6A_4 + \beta_4\beta_8) - \beta_4\beta_5(1-\beta_8))\eta_2 + N\beta_1\beta_3A_2(\beta_7A_4 - \beta_7\beta_8 + \beta_7)\}/\beta_1\{\{\beta_D A_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1 + \eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1 - \beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5 + \beta_8\beta_{11})((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)]\}$$

$$R^* = (A_1\mu - B\beta_1)\{\beta_D A_1[(\beta_3\beta_4(\beta_{10}A_5 + \beta_8\beta_{11}) + \beta_{11}(\beta_3\beta_6A_4 + \beta_4\beta_5\beta_8 - \beta_4\beta_5))\eta_2 + \beta_4(\beta_{10}A_3 - \beta_8\beta_9 + \beta_9)(A_5\eta_1 + \beta_5\eta_3) + \beta_3(\beta_4(\beta_7\beta_{10} - \beta_8\beta_9) + \beta_6(\beta_7\beta_{10} - \beta_{10} - A_4\beta_9))\eta_3 + \beta_4\beta_{11}(\beta_8A_3 - \beta_7\beta_8 + \beta_7)\eta_1] + (A_3A_4 - (1-\beta_7)(1-\beta_8))\beta_{11}(\beta_D\beta_6A_1 + N\beta_1(A_2A_5 + \beta_2\beta_6)) + N\beta_1[((1-\beta_8)\beta_2\beta_4 + \beta_3A_2A_4)(\beta_9A_5 + \beta_7\beta_{11}) + ((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)(\beta_{10}A_5 + \beta_8\beta_{11})]\}/\beta_1\{\{\beta_D A_1[(A_3A_4 - (1-\beta_7)(1-\beta_8))[(\beta_6\beta_{11}\beta_{12} - A_2A_5A_6)\eta_1 + A_6(\beta_6A_1 - A_2(\beta_5 + \beta_6))\eta_3] + (1-\beta_8)\beta_4(\beta_9\beta_{12}A_5(\eta_1 + \eta_3) + \beta_7\beta_{11}\beta_{12}\eta_1 - \beta_2\beta_7A_6\eta_3 + (A_5A_6(A_1 - \beta_2) - \beta_5\beta_{11}\beta_{12})\eta_2) + \beta_4\beta_{12}A_3\eta_1(\beta_{10}A_5 + \beta_8\beta_{11}) - (1-\beta_7)\beta_3(\beta_8A_2A_6 + \beta_6\beta_{10}\beta_{12})\eta_3 - (A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})\beta_3(A_5\eta_2 + \beta_7\eta_3) + \beta_3\beta_{12}(\beta_6A_4 + \beta_4\beta_8)(\beta_{11}\eta_2 - \beta_9\eta_3) + A_3\beta_4(\beta_5\beta_{10}\beta_{12} - \beta_2\beta_8A_6)\eta_3]\} + (A_3A_4 - (1-\beta_7)(1-\beta_8))N\beta_1((\beta_2\beta_6 + \beta_5A_2)(\beta_{11}\beta_{12}) - A_1A_2A_5A_6) + N\beta_1\beta_{12}[(\beta_9A_5 + \beta_7\beta_{11})((1-\beta_8)\beta_4\beta_2 + \beta_3A_2A_4) + (\beta_{10}A_5 + \beta_8\beta_{11})((1-\beta_7)\beta_3A_2 + \beta_2\beta_4A_3)]\}$$

Malaria-Only Sub-Model

The system of non-linear differential equation for the malaria spread described in Figure 2 is

Figure 2. Malaria-only sub-model



$$\begin{aligned}
\frac{dS}{dt} &= B + \beta_{12}R - \beta_1 SE_M - \mu S \\
\frac{dE_M}{dt} &= \beta_1 SE_M - (\mu + \beta_3)E_M \\
\frac{\mathrm{dI}_M}{dt} &= \beta_3 E_M - (\mu + \beta_9)I_M \\
\frac{dR}{dt} &= \beta_9 I_M - (\mu + \beta_{12})R
\end{aligned} \tag{3}$$

Adding these equations, we have

$$\frac{d}{dt}(S + E_M + I_M + R) = B - \mu(S + E_M + I_M + R) \geq 0$$

Hence, $\limsup_{t\to\infty}(S+E_M+I_M+R)(t)\le\frac{B}{\mu}$. Feasible Region for the spread of malaria only model is,

$$\Lambda_M=\left\{(S,E_M,I_M,R):S+E_M+I_M+R\le\frac{B}{\mu},S\ge 0,E_M\ge 0,I_M\ge 0,R\ge 0\right\}.$$

On equating system of equations (3) equal to zero, we get two Equilibrium points i.e.

i. Disease-free equilibrium point $E_M^0\left(\frac{B}{\mu},0,0,0\right)$

ii. ii. Endemic equilibrium point

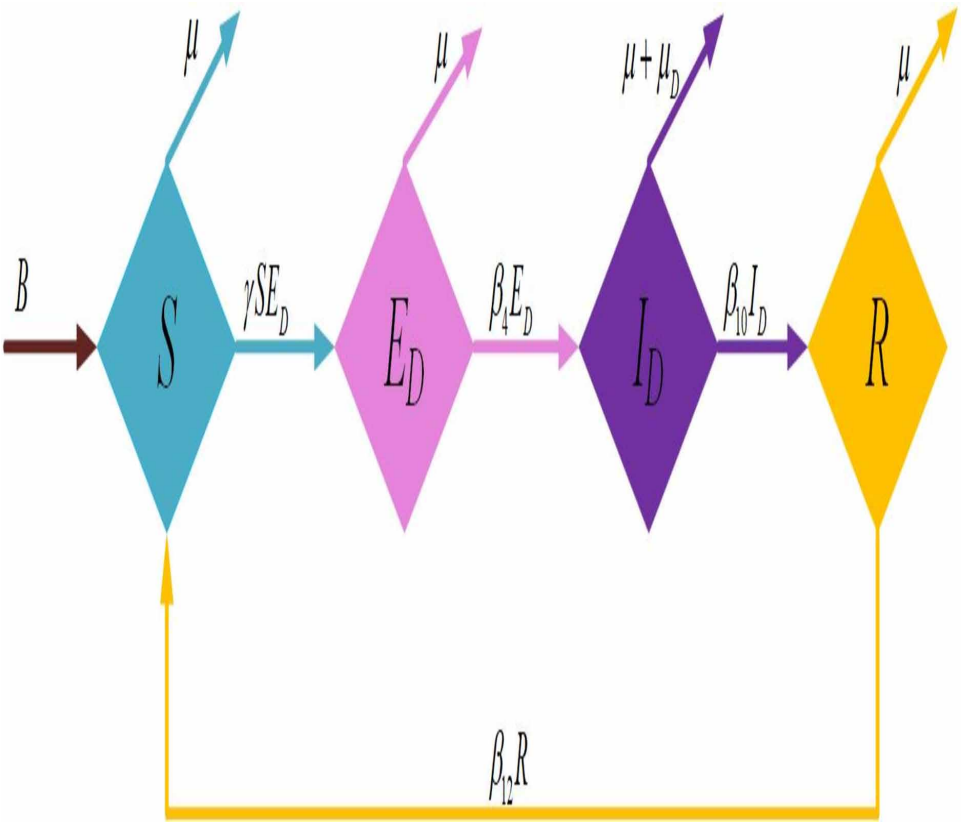
$$E_M^*\left(\frac{\mu+\beta_3}{\beta_1},\frac{(B\beta_1-\mu\beta_3-\mu^2)(\mu+\beta_{12})(\mu+\beta_9)}{\beta_1\mu((\mu+\beta_{12})(\mu+\beta_3+\beta_9)+\beta_3\beta_9)},\right.$$
$$\left.\frac{\beta_3(\mu+\beta_{12})(B\beta_1-\mu\beta_3-\mu^2)}{\beta_1\mu((\mu+\beta_{12})(\mu+\beta_3+\beta_9)+\beta_3\beta_9)},\frac{\beta_3\beta_9(B\beta_1-\mu\beta_3-\mu^2)}{\beta_1\mu((\mu+\beta_{12})(\mu+\beta_3+\beta_9)+\beta_3\beta_9)}\right)$$

Dengue-Only Sub-Model

Transmission of Dengue is given in Figure 3 which can be represented by the following non-linear differential equations

$$\begin{aligned}\frac{dS}{dt}&=B+\beta_{12}R-\mu S-SE_D\gamma\\ \frac{dE_D}{dt}&=SE_D\gamma-\mu E_D-\beta_4E_D\\ \frac{dI_D}{dt}&=\beta_4E_D-(\mu+\mu_D)I_D-\beta_{10}I_D\\ \frac{dR}{dt}&=\beta_{10}I_D-(\mu+\beta_{12})R\end{aligned}\tag{4}$$

Figure 3. Dengue-only sub-model



Adding above equations, we get,

$$\frac{d}{dt}(S+E_D+I_D+R)=B-\mu(S+E_D+I_D+R)-\mu_D I_D \le B-\mu(S+E_D+I_D+R)\ge 0$$

Hence, $\lim_{t\to\infty}\sup(S+E_D+I_D+R)\le\frac{B}{\mu}$. Feasible region for dengue only model is

$$\Lambda_D=\left\{(S,E_D,I_D,R):S+E_D+I_D+R\le\frac{B}{\mu},S\ge 0,E_D\ge 0,I_D\ge 0,R\ge 0\right\}.$$

Now, on equating we get two equilibrium points

i. Disease-free equilibrium point $E_D^0\left(\frac{B}{\mu},0,0,0\right)$

ii. ii. Endemic equilibrium point

$$E_D^*\left(\frac{\mu+\beta_4}{\gamma},\frac{(B\gamma-\mu\beta_4-\mu^2)(\mu+\beta_{12})(\mu+\mu_D+\beta_{10})}{\gamma((\mu_D\beta_4+\mu\beta_4+\mu(\mu+\mu_D+\beta_{10}))(\mu+\beta_{12})+\mu\beta_4\beta_{10})},\right.$$
$$\frac{(\mathrm{B}\gamma-\mu\beta_4-\mu^2)\beta_4(\mu+\beta_{12})}{\gamma((\mu_D\beta_4+\mu\beta_4+\mu(\mu+\mu_D+\beta_{10}))(\mu+\beta_{12})+\mu\beta_4\beta_{10})},$$
$$\left.\frac{(\mathrm{B}\gamma-\mu\beta_4-\mu^2)\beta_4\beta_{10}}{\gamma((\mu_D\beta_4+\mu\beta_4+\mu(\mu+\mu_D+\beta_{10}))(\mu+\beta_{12})+\mu\beta_4\beta_{10})}\right)$$

In the next section, we discuss basic reproduction number for each model formulated in section 2 using next generation matrix method (Diekmann *et al.*, 2009).

BASIC REPRODUCTION NUMBER

Basic reproduction number also known as threshold value is defined as the number of infections generated due to single infected individual in a susceptible population. It is helpful in developing control strategies for an epidemic.

Reproduction Number R_0 for Co-Infectious Model

Reproduction number R_0 is the value of number of individuals co-infected by malaria-dengue caused by single infected individual in susceptible population.

We have,

$$F(E^0)=\begin{bmatrix} \frac{B\beta_1}{\mu} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{B\beta_D\eta_1}{\mu N} & 0 & \frac{B\beta_D\eta_2}{\mu N} & 0 & \frac{B\beta_D\eta_3}{\mu N} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{B\beta_1}{\mu}+\frac{B\beta_D\eta_1}{\mu N} & 0 & \frac{B\beta_D\eta_2}{\mu N} & 0 & \frac{B\beta_D\eta_3}{\mu N} & 0 & 0 \end{bmatrix}$$

$$V(E^0)=\begin{bmatrix} A_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\beta_2 & A_2 & 0 & 0 & 0 & 0 & 0 \\ -\beta_3 & 0 & A_3 & -(1-\beta_8) & 0 & 0 & 0 \\ 0 & -\beta_4 & -(1-\beta_7) & A_4 & 0 & 0 & 0 \\ -\beta_5 & -\beta_6 & -\beta_7 & -\beta_8 & A_5 & 0 & 0 \\ 0 & 0 & -\beta_9 & -\beta_{10} & -\beta_{11} & A_6 & 0 \\ \frac{B\beta_1}{\mu}+\frac{B\beta_D\eta_1}{\mu N} & 0 & \frac{B\beta_D\eta_2}{\mu N} & 0 & \frac{B\beta_D\eta_3}{\mu N} & -\beta_{12} & \mu \end{bmatrix}$$

Reproduction number R_0 is spectral radius of the matrix FV^{-1}.

$$R_0=\frac{B\beta_1}{\mu A_1}+\frac{B\beta_D}{\mu N A_2\left(\begin{matrix}A_3A_4-1-\beta_7\beta_8\\+\beta_7+\beta_8\end{matrix}\right)}\left[\frac{\eta_3\left(\begin{matrix}A_6(A_3A_4-1-\beta_7\beta_8\\+\beta_7+\beta_8)+A_3\beta_4\beta_8\\-\beta_4\beta_7-\beta_4\beta_7\beta_8\end{matrix}\right)}{A_5}-\eta_2\beta_4(\beta_8-1)\right] \quad (5)$$

Basic Reproduction Number for Malaria-Only Model

Basic Reproduction number for malaria infected individuals gives us the number of infected individuals with malaria due to one infection.

$$F_M(E_M^0)=\begin{bmatrix} \frac{B\beta_1}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},\ V_M(E_M^0)=\begin{bmatrix} \mu+\beta_3 & 0 & 0 & 0 \\ -\beta_3 & \mu+\beta_9 & 0 & 0 \\ 0 & -\beta_9 & \mu+\beta_{12} & 0 \\ \frac{B\beta_1}{\mu} & 0 & -\beta_{12} & \mu \end{bmatrix},$$

R_{0_M} is the spectral radius of the matrix $F_M V_M^{-1}$.

$$R_{0_M}=\frac{B\beta_1}{\mu(\mu+\beta_3)} \tag{6}$$

R_{0_D} for Dengue-Only Model

Basic reproduction number defines the number of dengue infected individuals due to one infected individual. It is calculated as follow

$$F_D(E_D^0)=\begin{bmatrix} \frac{\gamma B}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},\ V_D(E_D^0)=\begin{bmatrix} \mu+\beta_4 & 0 & 0 & 0 \\ -\beta_4 & \mu+\mu_D+\beta_{10} & 0 & 0 \\ 0 & -\beta_{10} & \mu+\beta_{12} & 0 \\ \frac{\gamma B}{\mu} & 0 & -\beta_{12} & \mu \end{bmatrix}$$

Reproduction number for only dengue infection is the spectral radius of the matrix $F_D V_D^{-1}$.

Table 2. Basic Reproduction Number

Model	Basic Reproduction Number
Malaria-only	0.3333
Dengue-only	0.5000
Co-infection	0.1754

$$R_0(D) = \frac{\gamma B}{\mu(\mu + \beta_4)} \tag{7}$$

STABILITY ANALYSIS

In this section local and global stability of the co-infectious model and its sub-models' malaria-only, dengue-only will be studied.

Stability of Co-Infectious Model

Local Stability

Local stability is obtained for co-infectious model for three of its equilibrium points E^0, E^1, E^* by using Routh-Hurwitz criteria. Jacobian matrix for the system of non -linear differential equations given in (2) is

$$J=\begin{bmatrix} \begin{pmatrix} -\beta_1 E_M-\mu \\ -\beta_D\dfrac{(\mathrm{E}_M\eta_1+\mathrm{I}_M\eta_2+\mathrm{I}_{MD}\eta_3)}{N}\end{pmatrix} & -S\beta_1-\dfrac{S\beta_D\eta_1}{N} & 0 & -\dfrac{S\beta_D\eta_2}{N} & 0 & -\dfrac{S\beta_D\eta_3}{N} & \beta_{12} \\ \beta_1 E_M & S\beta_1-A_1 & 0 & 0 & 0 & 0 & 0 \\ \beta_D\dfrac{(\mathrm{E}_M\eta_1+\mathrm{I}_M\eta_2+\mathrm{I}_{MD}\eta_3)}{N} & \beta_2+\dfrac{S\beta_D\eta_1}{N} & -A_2 & \dfrac{S\beta_D\eta_2}{N} & 0 & \dfrac{S\beta_D\eta_3}{N} & 0 \\ 0 & \beta_3 & 0 & -A_3 & 1-\beta_8 & 0 & 0 \\ 0 & 0 & \beta_4 & 1-\beta_7 & -A_4 & 0 & 0 \\ 0 & \beta_5 & \beta_6 & \beta_7 & \beta_8 & -A_5 & 0 \\ 0 & 0 & 0 & \beta_9 & \beta_{10} & \beta_{11} & -A_6 \end{bmatrix} \tag{8}$$

Theorem 1: Disease free equilibrium point E^0 is said to be asymptotically stable if following conditions are satisfied

1. $x_2<0$
2. $A_2A_5 > \max\left\{x_5\beta_6, \frac{x_4\beta_4}{A_3}\right\}$
3. $(A_3A_4 - (1-\beta_7)(1-\beta_8)) > 0$
4. $\beta_7\beta_8 > \max\left\{\frac{x_5\beta_7(A_3\beta_6+\beta_4\beta_8)}{x_4\beta_4}, \frac{A_3x_5\beta_8 + x_4A_5}{x_5}\right\}$
5. $A_5x_4\beta_8 - x_5\beta_7 > 0$

Proof: The Jacobian matrix for disease free equilibrium is

$$J^0 = \begin{bmatrix} -\mu & -x_1 & 0 & -x_4 & 0 & -x_5 & \beta_{12} \\ 0 & x_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & x_3 & -A_2 & x_4 & 0 & x_5 & 0 \\ 0 & \beta_3 & 0 & -A_3 & 1-\beta_8 & 0 & 0 \\ 0 & 0 & \beta_4 & 1-\beta_7 & -A_4 & 0 & 0 \\ 0 & \beta_5 & \beta_6 & \beta_7 & \beta_8 & -A_5 & 0 \\ 0 & 0 & 0 & \beta_9 & \beta_{10} & \beta_{11} & -A_6 \end{bmatrix} \tag{9}$$

where,

$$x_1 = \frac{B\beta_1}{\mu} + \frac{B\beta_D\eta_1}{\mu N}, x_2 = \frac{B\beta_1}{\mu} - A_1, x_3 = \beta_2 + \frac{B\beta_D\eta_1}{\mu N}, x_4 = \frac{B\beta_D\eta_2}{\mu N}, x_5 = \frac{B\beta_D\eta_3}{\mu N}$$

The characteristic polynomial for Jacobian J^0 is

$$\lambda^7 + a_1\lambda^6 + a_2\lambda^5 + a_3\lambda^4 + a_4\lambda^3 + a_5\lambda^2 + a_6\lambda + a_7$$

Here,

$$a_1 = A_2 + A_3 + A_4 + A_5 + A_6 + \mu - x_2$$

$$a_2 = A_2(A_3 + A_4 + A_6 + \mu) - x_2(A_2 + A_3 + A_4 + A_5 + A_6 + \mu) + (A_3 + A_4)(A_5 + A_6 + \mu) + A_5(A_6 + \mu) + A_2A_5 - x_5\beta_6 + A_3A_4 - (1-\beta_7)(1-\beta_8) + A_6\mu$$

$$a_3 = (A_2 + A_5 + A_6 + \mu - x_2)(A_3A_4 - (1-\beta_7)(1-\beta_8)) + (A_4 + A_6 - x_2 + \mu)(A_2A_5 - x_5\beta_6) + (A_2 - x_2)((A_3 + A_4)(A_6 + \mu) + A_6\mu) - x_2(A_2 + A_5)(A_3 + A_4 + A_6 + \mu) + (A_3 + A_4)(A_5A_6 + A_5\mu + A_6\mu) + A_5A_6\mu + (A_2A_3A_5 - x_4\beta_4) + (x_4\beta_4\beta_8 - A_3x_5\beta_6 - x_5\beta_4\beta_8)$$

$$a_4 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{(A_2A_5 - x_5\beta_6) + ((A_2 + A_5)(A_6 + \mu - x_2) + A_6\mu - x_2(A_6 + \mu))\} + (A_4(A_6 + \mu) + A_6\mu - x_2(A_4 + A_6 + \mu))(A_2A_5 - x_5\beta_6) + (A_6 + \mu - x_2)(A_2A_3A_5 - \beta_4x_4) + (A_6 + \mu - x_2)(x_4\beta_4\beta_8 - A_3x_5\beta_6 - x_5\beta_4\beta_8) + \beta_4(x_5\beta_7\beta_8 - A_3x_5\beta_8 - A_5x_4) + \beta_4(A_5x_4\beta_8 - x_5\beta_7) - x_2((A_2 + A_5)(A_3 + A_4)(A_6 + \mu) + A_6\mu(A_2 + A_3 + A_4 + A_5)) + A_6\mu(A_2 + A_5)(A_3 + A_4)$$

$$a_5 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{(A_2A_5 - x_5\beta_6)(A_6 + \mu - x_2) + A_6\mu(A_2 + A_5) - x_2((A_2 + A_5)(A_6 + \mu) + A_6\mu) + (A_2A_5 - x_5\beta_6)(A_6\mu A_4 - x_2(A_3 + A_4 + A_6\mu)) + (A_6\mu - x_2(A_6 + \mu))(A_2A_3A_5 - \beta_4x_4) + (A_6\mu - x_2(A_6 + \mu))(x_4\beta_4\beta_8 - A_3x_5\beta_6 - x_5\beta_4\beta_8) + \beta_4(x_5\beta_7\beta_8 - A_5x_4 - A_3x_5\beta_8)(A_6 + \mu - x_2) + \beta_4(A_5x_4\beta_8 - x_5\beta_7)(A_6 + \mu - x_2) - x_2A_6\mu(A_3 + A_4)(A_2 + A_5)$$

$$a_6 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{(A_2A_5 - x_5\beta_6)(A_6\mu - x_2(A_6 + \mu)) - x_2A_6\mu(A_2 + A_5)\} - x_2A_6\mu(A_2A_5 - x_5\beta_6)(A_3 + A_4) - x_2A_6\mu\beta_4(x_4\beta_8 - x_5\beta_8 - x_4) + \beta_4(A_5x_4\beta_8 - x_5\beta_7)(A_6\mu - x_2(A_6 + \mu)) + \beta_4(x_5\beta_7\beta_8 - A_5x_4 - A_3x_5\beta_8)(A_6\mu - x_2(A_6 + \mu))$$

$$a_7 = -x_2A_6\mu\{(A_3A_4 - (1-\beta_7)(1-\beta_8))(A_2A_5 - x_5\beta_6) + \beta_4(x_5\beta_7\beta_8 - A_5x_4 - A_3x_5\beta_8) + \beta_4(A_5x_4\beta_8 - x_5\beta_7)\}$$

with, a_1, a_2, a_3, a_4, a_5, a_6, a_7>0 if

$$x_2 < 0, A_2A_3A_5 > \max\{x_5A_3\beta_6, x_4\beta_4\},$$

$$(A_3A_4 - (1-\beta_7)(1-\beta_8)) > 0, \beta_7\beta_8 > \max\left\{\frac{x_5\beta_7(A_3\beta_6 + \beta_4\beta_8)}{x_4\beta_4}, \frac{A_3x_5\beta_8 + x_4A_5}{x_5}\right\}, A_5x_4\beta_8 - x_5\beta_7 > 0 \cdot$$

Then, by Routh Hurwitz criteria, disease free equilibrium point is said to be locally asymptotically stable.

Theorem 2: Malaria free equilibrium point is asymptotically stable if it satisfies following conditions

1. $y_2<0$
2. $A_1-S\beta_1>0$
3. $(A_3A_4-(1-\beta_7)(1-\beta_8))>0$
4. $$\beta_7\beta_8>\left\{\frac{y_5\beta_8A_3+y_4A_5}{y_5},\frac{(A_3\beta_6+\beta_4\beta_8)\,\mathrm{y}_5\,\beta_7}{y_4\beta_4},\frac{\beta_{10}A_3A_5+A_5\beta_9}{\beta_{11}},\frac{\beta_7(\beta_3\beta_4\beta_{10}+A_3}{\beta_4\beta_9}\quad\beta\right.$$
5. $$A_2A_5>\max\left\{y_5\beta_6,\frac{y_5\beta_4}{A_3},\frac{\beta_6\beta_{11}\beta_{12}}{A_6},\frac{\beta_4\beta_6\beta_{11}\beta_{12}+A_5\beta_4\beta_{10}\beta_{12}}{A_4A_6},\frac{\beta_4\beta_{12}(\beta_8\beta_{11}+\beta_9)}{A_3A_6},\frac{\beta_{11}(\beta_8A_3+}{\beta_8\beta_9}\right.\quad)$$

Proof: The Jacobian matrix for Malaria free equilibrium is

$$J^1=\begin{bmatrix}-\mu-y_1 & y_2 & 0 & -y_4 & 0 & -y_5 & \beta_{12}\\ 0 & S\beta_1-A_1 & 0 & 0 & 0 & 0 & 0\\ y_1 & y_3 & -A_2 & y_4 & 0 & y_5 & 0\\ 0 & \beta_3 & 0 & -A_3 & 1-\beta_8 & 0 & 0\\ 0 & 0 & \beta_4 & 1-\beta_7 & -A_4 & 0 & 0\\ 0 & \beta_5 & \beta_6 & \beta_7 & \beta_8 & -A_5 & 0\\ 0 & 0 & 0 & \beta_9 & \beta_{10} & \beta_{11} & -A_6\end{bmatrix} \tag{10}$$

where,

$$y_1=\frac{\beta_D(I_M\eta_2+I_{MD}\eta_3)}{N},\mathrm{y}_2=-S\beta_1-\frac{S\beta_D\eta_1}{N},\mathrm{y}_3=\beta_2+\frac{S\beta_D\eta_1}{N},\mathrm{y}_4=\frac{S\beta_D\eta_2}{N},\mathrm{y}_5=\frac{S\beta_D\eta_3}{N}$$

The characteristic polynomial for Jacobian J^1 is

$$\lambda^7+b_1\lambda^6+b_2\lambda^5+b_3\lambda^4+b_4\lambda^3+b_5\lambda^2+b_6\lambda+b_7$$

Here,

$$b_1 = A_2 + A_3 + A_4 + A_5 + A_6 + y_1 + \mu + A_1 - S\beta_1$$

$$b_2 = (A_3A_4 - (1-\beta_7)(1-\beta_8)) + (A_2A_5 - y_5\beta_6) + (A_1 - S\beta_1)(A_2 + A_3 + A_4 + A_5 + A_6 + y_1 + \mu) + (y_1 + \mu)(A_2 + A_3 + A_4 + A_5 + A_6) + (A_3 + A_4)(A_2 + A_5 + A_6) + A_6(A_2 + A_5)$$

$$b_3 = (A_3A_4 - (1-\beta_7)(1-\beta_8))(A_1 - S\beta_1 + A_2 + A_5 + A_6 + y_1 + \mu) + (A_1 - S\beta_1)\{(A_2 + A_3 + A_4 + A_5 + A_6)(y_1 + \mu) + (A_2 + A_5 + A_6)(A_3 + A_4) + A_6(A_2 + A_5)\} + (\mathrm{A_2A_5} - \mathrm{y_5}\,\beta_6)(A_1 - S\beta_1 + A_4 + A_6 + \mu) + A_6(\mathrm{y_1} + \mu)(A_2 + A_3 + A_4 + A_5) + (A_6 + y_1 + \mu)(A_2 + A_5)(A_3 + A_4) + \mathrm{A_2}\,A_5y_1 + ((A_2A_3A_5 - y_4\beta_4) + (y_4\beta_4\beta_8 - A_3y_5\beta_6 - y_5\beta_4\beta_8))$$

$$b_4 = (A_2A_5 - y_5\beta_6)((A_1 - S\beta_1)(A_4 + A_6 + \mu) + A_6A_4 + \mu(A_4 + A_6)) + (A_3A_4 - (1-\beta_7)(1-\beta_8))\{(y_1 + \mu)(A_2 + A_5 + A_6) + A_6(A_2 + A_5) + A_2A_5 - y_5\beta_6 + (A_1 - S\beta_1)(A_2 + A_5 + A_6 + y_1 + \mu)\} + (A_1 - S\beta_1)\{(y_1 + \mu)((A_2 + A_5 + A_6)(A_3 + A_4) + A_6(A_2 + A_5)) + A_6(A_2 + A_5)(A_3 + A_4) + A_2A_5y_1\} + (A_1 - S\beta_1 + \mu + A_6)((A_2A_3A_5 - y_4\beta_4) + (y_4\beta_4\beta_8 - A_3y_5\beta_6 - y_5\beta_4\beta_8)) + \beta_4((\beta_8A_5y_4 - y_5\beta_7) + (y_5\beta_7\beta_8 - \beta_8A_3y_5 - y_4A_5)) + y_1((A_2A_4A_5 - \beta_4\beta_{10}\beta_{12}) + (A_2A_5A_6 - \beta_6\beta_{11}\beta_{12})) + y_1A_2A_3A_5 + (y_1 + \mu)A_6(A_2 + A_5)(A_3 + A_4)$$

$$b_5 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{(A_1 - S\beta_1 + A_6 + \mu)(A_2A_5 - y_5\beta_6) + (A_1 - S\beta_1)[(y_1 + \mu)(A_2 + A_5 + A_6) + A_6(A_2 + A_5)] + A_2A_5y_1 + A_6(A_2 + A_5)(y_1 + \mu)\} + (A_2A_5 - y_5\beta_6)\{A_6\mu A_4 + (A_1 - S\beta_1)((A_6 + \mu)A_4 + A_6\mu)\} + \{(A_1 - S\beta_1)(\mu + A_6) + A_6\mu\}((A_2A_3A_5 - y_4\beta_4) + (y_4\beta_4\beta_8 - A_3y_5\beta_6 - y_5\beta_4\beta_8)) + (A_1 - S\beta_1)\{A_2A_3A_5y_1 + y_1((A_2A_4A_5 - \beta_4\beta_{10}\beta_{12}) + A_2A_5A_6 - \beta_6\beta_{11}\beta_{12}) + (y_1 + \mu)A_6(A_2 + A_5)(A_3 + A_4)\} + (A_1 - S\beta_1 + \mu + A_6)\beta_4((\beta_8A_5y_4 - y_5\beta_7) + (y_5\beta_7\beta_8 - \beta_8A_3y_5 - y_4A_5)) + y_1(A_2A_4A_5A_6 - \beta_6\beta_{11}\beta_{12}A_4 - \beta_4A_5\beta_{10}\beta_{12})) + y_1\{\beta_{12}(\beta_4\beta_8\beta_9 - \beta_4\beta_{10}A_3 - \beta_6\beta_{11}A_3) + (A_2A_3A_5A_6 - \beta_4\beta_{12}\beta_8\beta_{11} - \beta_4\beta_{12}\beta_9)\}$$

$$b_6 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{((A_1 - S\beta_1)(A_6 + \mu) + \mu A_6)(A_2A_5 - y_5\beta_6) + (A_1 - S\beta_1)(A_6(A_2 + A_5) + y_1 + \mu + y_1A_2A_5) + y_1(A_2A_5A_6 - \beta_6\beta_{11}\beta_{12})\} + \mu A_6(A_1 - S\beta_1)(A_2A_5 - y_5\beta_6)A_4 + (A_1 - S\beta_1)y_1(A_2A_4A_5A_6 - \beta_6\beta_{11}\beta_{12}A_4 - \beta_4\beta_{10}\beta_{12}A_5)\} + \beta_4((A_1 - S\beta_1)(A_6 + \mu) + A_6\mu)((y_5\beta_7\beta_8 - y_5A_3\beta_8 - y_4A_5) + (\beta_8y_4A_5 - y_5\beta_7)) + \mu A_6(A_1 - S\beta_1)((A_2A_3A_5 - y_4\beta_4) + (y_4\beta_4\beta_8 - A_3y_5\beta_6 - y_5\beta_4\beta_8)) + y_1(A_1 - S\beta_1)\{\beta_{12}(\beta_4\beta_8\beta_9 - \beta_4\beta_{10}A_3 - \beta_6\beta_{11}A_3) + (A_2A_3A_5A_6 - \beta_4\beta_{12}\beta_8\beta_{11} - \beta_4\beta_{12}\beta_9)\} + y_1\beta_4\beta_{12}\{(\beta_7\beta_8\beta_{11} - \beta_{10}A_3A_5 - \beta_9A_5) + (A_5\beta_8\beta_9 - \beta_8\beta_{11}A_3 - \beta_7\beta_{11})\}$$

$$b_7 = (A_3A_4 - (1-\beta_7)(1-\beta_8))(A_1 - S\beta_1)\{y_1(A_2A_5A_6 + \beta_6\beta_{11}\beta_{12}) + A_6\mu(A_2A_5 - y_5\beta_6)\} + \mu A_6(A_1 - S\beta_1)\beta_4((y_5\beta_7\beta_8 - \beta_8y_5A_3 - y_4A_5) + (\beta_8y_4A_5 - y_5\beta_7)) + y_1\beta_4\beta_{12}(A_1 - S\beta_1)\{(\beta_7\beta_8\beta_{11} - \beta_{10}A_3A_5 - \beta_9A_5) + (A_5\beta_8\beta_9 - \beta_8\beta_{11}A_3 - \beta_7\beta_{11})\}$$

with b_1, b_2, b_3, b_4, b_5, b_6, b_7>0, if

$y_2 < 0, A_1 - S\beta_1 > 0, (A_3A_4 - (1-\beta_7)(1-\beta_8)) > 0$,

$$\beta_7\beta_8 > \left\{ \frac{y_5\beta_8A_3 + y_4A_5}{y_5}, \frac{(A_3\beta_6 + \beta_4\beta_8)\,y_5\,\beta_7}{y_4\beta_4}, \frac{\beta_{10}A_3A_5 + A_5\beta_9}{\beta_{11}}, \frac{\beta_7(\beta_3\beta_4\beta_{10} + A_3A_6\beta_{11})}{\beta_4\beta_9} \right\},$$

$$A_2A_5 > \max\left\{ y_5\beta_6, \frac{y_5\beta_4}{A_3}, \frac{\beta_6\beta_{11}\beta_{12}}{A_6}, \frac{\beta_4\beta_6\beta_{11}\beta_{12} + A_5\beta_4\beta_{10}\beta_{12}}{A_4A_6}, \frac{\beta_4\beta_{12}(\beta_8\beta_{11} + \beta_9)}{A_3A_6}, \frac{\beta_{11}(\beta_8A_3 + \beta_7)A_2}{\beta_8\beta_9} \right\}$$

Then, by Routh Hurwitz criteria, disease free equilibrium point is said to be locally asymptotically stable.

Theorem 3: The endemic point is said to be locally asymptotically stable if following conditions are satisfied

1. $z_2<0$
2. $A_1 - S\beta_1>0$
3. $(A_3A_4 - (1-\beta_7)(1-\beta_8)) > 0$
4. $$\beta_7\beta_8 > \left\{ \begin{array}{l} \frac{(z_5\beta_8A_3 + z_4A_5)}{z_5}, \frac{(A_3\beta_6 + \beta_4\beta_8)\,z_5\,\beta_7}{z_4\beta_4}, \frac{(\beta_{10}A_3A_5 + A_5\beta_9)}{\beta_{11}}, \frac{\beta_7(A_3\beta_4\beta_{10} + A_3\beta}{\beta_4\beta_9} \\ \frac{\beta_6\beta_7\beta_{11}\beta_{12}}{z_5\beta_4}, \frac{A_3\beta_7\beta_{10}}{\beta_9}, \frac{(A_2 + A_5)\beta_{10}}{\beta_{11}}, \frac{A_3z_5\beta_8 + z_4\beta_3\beta_8}{z_3}, \frac{(A_2\beta_3\beta_7\beta_{11} + A_5z_3\beta_4\beta}{\beta_3\beta_{10}A_2} \\ \frac{(A_2A_5\beta_3\beta_9 + z_3\beta_4\beta_9)\beta_8}{\beta_3\beta_9A_4}, \frac{(A_2A_5\beta_3\beta_{10} + A_5z_3\beta_4\beta_9)}{\beta_3\beta_9A_4}, \frac{\beta_5\beta_8\beta_{11}\beta_{12}}{z_3\beta} \end{array} \right)$$
5. $$A_2A_5 > \max\left\{ \begin{array}{l} z_5\beta_6, \frac{z_5\beta_4}{A_3}, \frac{\beta_6\beta_{11}\beta_{12}}{A_6}, \frac{\beta_4\beta_6\beta_{11}\beta_{12} + A_5\beta_4\beta_{10}\beta_{12}}{A_4A_6}, \frac{\beta_4\beta_{12}(\beta_8\beta_{11}}{A_3A_6}\ \beta \\ \frac{\beta_{11}(\beta_8A_3 + \beta_7)\,A_2}{\beta_8\beta_8}, \frac{A_2\beta_5\beta_{12}\beta_{11}}{A_4A_4}, \frac{A_5\beta_{10}\beta_{12}}{A_4z_4} \end{array} \right.$$
6. $A_5z_4 > \max\left\{ \beta_9\beta_{12}, \frac{z_5\beta_7}{\beta_8}, \frac{A_5\beta_9\beta_{12}}{A_6} \right\}$
7. $A_3z_3\beta_4 > \max\left\{ A_6\beta_3\beta_7, A_2\beta_7\beta_8 \right\}$
8. $$z_5\beta_6 > \max\left\{ \frac{\beta_4\beta_{10}\beta_{12}}{A_4}, \frac{\beta_6\beta_{11}\beta_{12}}{A_6}, \frac{z_4\beta_3\beta_6}{\beta_9}, \frac{(\beta_7\beta_{11}z_3 + \beta_5\beta_9z_5)\beta_6}{\beta_8\beta_9}, \beta_4A_5z_4 + \beta_4 \right.$$

Proof: The Jacobian matrix for endemic equilibrium is

$$J^* = \begin{bmatrix} -\mathrm{E}_M\beta_1 - \mathrm{z}_1 - \mu & z_2 & 0 & -z_4 & 0 & -z_5 & \beta_{12} \\ E_M\beta_1 & S\beta_1 - A_1 & 0 & 0 & 0 & 0 & 0 \\ z_1 & z_3 & -A_2 & z_4 & 0 & z_5 & 0 \\ 0 & \beta_3 & 0 & -A_3 & 1-\beta_8 & 0 & 0 \\ 0 & 0 & \beta_4 & 1-\beta_7 & -A_4 & 0 & 0 \\ 0 & \beta_5 & \beta_6 & \beta_7 & \beta_8 & -A_5 & 0 \\ 0 & 0 & 0 & \beta_9 & \beta_{10} & \beta_{11} & -A_6 \end{bmatrix} \tag{11}$$

where,

$$z_1 = \frac{\beta_D(\mathrm{E}_M\eta_1 + I_M\eta_2 + I_{MD}\eta_3)}{N}, \mathrm{z}_2 = -S\beta_1 - \frac{S\beta_D\eta_1}{N}, \mathrm{z}_3 = \beta_2 + \frac{S\beta_D\eta_1}{N}, \mathrm{z}_4 = \frac{S\beta_D\eta_2}{N}, \mathrm{z}_5 = \frac{S\beta_D\eta_3}{N}$$

The characteristic polynomial for Jacobian J^* is

$$\lambda^7 + c_1\lambda^6 + c_2\lambda^5 + c_3\lambda^4 + c_4\lambda^3 + c_5\lambda^2 + c_6\lambda + c_7$$

Here,

$$c_1 = E_M\beta_1 + A_2 + A_3 + A_4 + A_5 + A_6 + z_1 + \mu + (A_1 - S\beta_1)$$

$$c_2 = (E_M\beta_1 + A_2 + A_3 + A_4 + A_5 + A_6 + z_1 + \mu)(A_1 - S\beta_1) + E_M\beta_1(A_2 + A_3 + A_4 + A_5 + A_6 - z_2) + (A_2A_5 - z_5\beta_6) + (A_3A_4 - (1-\beta_7)(1-\beta_8)) + (A_2 + A_3 + A_4 + A_5 + A_6)(z_1 + \mu) + (A_3 + A_4)(A_2 + A_5 + A_6) + A_6(A_2 + A_5)$$

$$c_3 = (A_3A_4 - (1-\beta_7)(1-\beta_8))((A_1 - S\beta_1) + E_M\beta_1 + A_2 + A_5 + A_6 + z_1 + \mu) + (A_2A_5 - z_5\beta_6)((A_1 - S\beta_1) + E_M\beta_1 + A_4 + A_6 + \mu) + (A_1 - S\beta_1)[(A_2 + A_3 + A_4 + A_5 + A_6)(z_1 + \mu) + (A_2 + A_5 + A_6)(A_3 + A_4) + \mathrm{A}_6(A_2 + A_5) + E_M\beta_1(A_2 + A_3 + A_4 + A_5 + A_6)] + (\mathrm{A}_2\mathrm{A}_3\mathrm{A}_5 - \mathrm{z}_4\beta_4) + (\mathrm{z}_4\beta_4\beta_8 - \mathrm{A}_3\mathrm{z}_5\beta_6 - \mathrm{z}_5\beta_4\beta_8) + \mathrm{E}_M\beta_1[(A_2 + A_5 + A_6)(A_3 + A_4) + \mathrm{A}_6(\mathrm{A}_2 + \mathrm{A}_5) - \mathrm{z}_2(A_2 + A_3 + A_4 + A_5 + A_6) + \mathrm{z}_4\beta_3 + \mathrm{z}_5\beta_5] + (\mathrm{z}_1 + \mu)[\mathrm{A}_6(A_2 + A_3 + A_4 + A_5) + (A_2 + A_5)(A_3 + A_4)] + \mathrm{A}_2\mathrm{A}_5\mathrm{z}_1 + \mathrm{A}_6(A_2 + A_5)(A_3 + A_4)$$

$$c_4 = (A_3A_4 - (1-\beta_7)(1-\beta_8))((A_1 - S\beta_1)(E_M\beta_1 + A_2 + A_5 + A_6 + z_1 + \mu) + (A_2A_5 - z_5\beta_6) + E_M\beta_1(A_2 + A_5 + A_6 - z_2) + (A_2 + A_5 + A_6)(z_1 + \mu) + A_6(A_2 + A_5)) + (A_1 - S\beta_1)(A_2A_5 - z_5\beta_6)(E_M\beta_1 + A_4 + A_6 + \mu) + (E_M\beta_1 + A_6 + \mu + A_1 - S\beta_1)((A_2A_3A_5 - z_4\beta_4) + (z_4\beta_4\beta_8 - A_3z_5\beta_6 - z_5\beta_4\beta_8)) + (A_2A_5 - z_5\beta_6)(E_M\beta_1(A_4 + A_6 - z_2) + A_4(A_6 + \mu) + A_6\mu) + (A_1 - S\beta_1)[E_M\beta_1((A_2 + A_5 + A_6)(A_3 + A_4) + A_6(A_2 + A_5)) + (z_1 + \mu)(A_6(A_2 + A_3 + A_4 + A_5) + (A_2 + A_5)(A_3 + A_4)) + A_2A_5z_1 + A_6(A_2 + A_5)(A_3 + A_4)] + E_M\beta_1(A_3A_5A_6 - \beta_5\beta_{11}\beta_{12}) + z_1((A_2A_4A_6 - \beta_4\beta_{10}\beta_{12}) + (A_2A_5A_6 - \beta_6\beta_{11}\beta_{12})) + E_M\beta_1\beta_3(A_6z_4 - \beta_9\beta_{12}) + \beta_4((A_5z_4\beta_8 - z_5\beta_7) + (z_5\beta_7\beta_8 - A_3z_5\beta_8 - A_5z_4)) + E_M\beta_1(A_6(A_2(A_3 + A_4) + A_4A_5) + z_4\beta_3(A_2 + A_4 + A_5) + z_5\beta_5(A_2 + A_3 + A_4 + A_6) - z_2[(A_2 + A_5 + A_6)(A_3 + A_4) + A_6(A_2 + A_5)] + z_3z_5\beta_6 + z_5\beta_3\beta_7) + (z_1 + \mu)A_6(A_3(A_2 + A_5) + A_4A_5) + A_2A_5z_1(A_3 + A_4) + A_2A_4A_6\mu$$

$$c_5 = (A_3A_4 - (1-\beta_7)(1-\beta_8))(A_1 - S\beta_1)[(A_2 + A_5 + A_6)(E_M\beta_1 + z_1 + \mu) + A_6(A_2 + A_5) + A_2A_5 - z_5\beta_6] + (A_1 - S\beta_1)(A_2A_5 - z_5\beta_6)[E_M\beta_1(A_4 + A_6) + A_4(A_6 + \mu) + A_6\mu] + (A_3A_4 - (1-\beta_7)(1-\beta_8))[(A_2A_5 - z_5\beta_6)(E_M\beta_1 + A_6 + \mu) + E_M\beta_1(-z_2(A_2 + A_5 + A_6) + z_5\beta_5 + A_6(A_2 + A_5)) + A_6(A_2 + A_5)(z_1 + \mu) + A_2A_5z_1] + (A_1 - S\beta_1)[E_M\beta_1(A_2A_3A_5 + A_2A_4A_6 + A_3A_5A_6 + A_4A_5A_6) + A_6(z_1 + \mu)(A_2A_3 + A_3A_5 + A_4A_5) + A_2A_5(A_3 + A_4)z_1 + A_2A_4A_6\mu + z_1((A_2A_4A_6 - \beta_4\beta_{10}\beta_{12}) + (A_2A_5A_6 - \beta_6\beta_{11}\beta_{12}))] + (A_1 - S\beta_1)(E_M\beta_1 + A_6 + \mu)((A_2A_3A_5 - z_4\beta_4) + (z_4\beta_4\beta_8 - A_3z_5\beta_6 - z_5\beta_4\beta_8)) + (A_2A_5 - z_5\beta_6)(E_M\beta_1(A_6A_4 - z_2(A_4 + A_6)) + A_6\mu A_4) + (A_1 - S\beta_1 + \mu + E_M\beta_1 + A_6)\beta_4((A_5z_4\beta_8 - z_5\beta_7) + (z_5\beta_7\beta_8 - A_3z_5\beta_8 - A_5z_4)) + ((A_2A_3A_5 - z_4\beta_4) + (z_4\beta_4\beta_8 - A_3z_5\beta_6 - z_5\beta_4\beta_8))(A_6\mu + E_M\beta_1(A_6 - z_2)) + (A_6z_4 - \beta_9\beta_{12})E_M\beta_1\beta_3(A_2 + A_4 + A_5) + (A_6z_5 - \beta_{11}\beta_{12})E_M\beta_1((A_2 + A_3 + A_4)\beta_5 + \beta_3\beta_7) + E_M\beta_1(A_3z_5z_3\beta_6 + z_3(z_5\beta_4\beta_8 - \beta_6\beta_{11}\beta_{12}) + z_3z_4\beta_4(1-\beta_8) + z_5\beta_3\beta_8(1-\beta_7) + \beta_3(A_2A_4z_4 - \beta_{10}\beta_{12}) + z_3(A_4z_5\beta_6 - \beta_4\beta_{10}\beta_{12}) - z_2A_6(A_2 + A_5)(A_3 + A_4) + A_5(A_2 + A_4)\beta_3z_4 + z_5\beta_5A_2(A_3 + A_4) + \beta_3\beta_7(A_2z_5 + \beta_{10}\beta_{12}) + A_6z_3z_5\beta_6 + A_4z_5\beta_3\beta_7) + z_1\{\beta_{12}(\beta_4\beta_8\beta_9 - \beta_4\beta_{10}A_3 - \beta_6\beta_{11}A_3) + (A_2A_3A_5A_6 - \beta_4\beta_8\beta_{11}\beta_{12} - \beta_4\beta_9\beta_{12}) + (A_2A_4A_5A_6 - A_5\beta_4\beta_{10}\beta_{12} - A_4\beta_6\beta_{11}\beta_{12})\}$$

$$c_6 = (A_3A_4 - (1-\beta_7)(1-\beta_8))\{((A_1 - S\beta_1)[(A_6 + \mu)(A_2A_5 - z_5\beta_6) + A_2A_5z_1 + (A_2 + A_5)A_6(z_1 + \mu) + E_M\beta_1(A_6(A_2 + A_5) + (A_2A_5 - z_5\beta_6))] + (A_6\mu + E_M\beta_1(A_6 - z_2))(A_2A_5 - z_5\beta_6) + z_1(A_2A_5A_6 - \beta_6\beta_{11}\beta_{12}) + E_M\beta_1(-z_2A_6(A_2 + A_5) + z_5(A_2\beta_5 + z_3\beta_6))\} + z_1\beta_4\beta_{12}\{(\beta_7\beta_8\beta_{11} - \beta_{10}A_3A_5 - \beta_9A_5) + (A_5\beta_8\beta_9 - \beta_8\beta_{11}A_3 - \beta_7\beta_{11})\} + \beta_4((A_5z_4\beta_8 - z_5\beta_7) + (z_5\beta_7\beta_8 - A_3z_5\beta_8 - A_5z_4))(E_M\beta_1(A_1 - S\beta_1 + A_6 - z_2) + (A_1 - S\beta_1)(A_6 + \mu) + A_6\mu) + (1-\beta_8)z_3E_M\beta_1\beta_4(\beta_7z_5 + A_5z_4) + (A_6z_5 - \beta_{11}\beta_{12})E_M\beta_1[\beta_5(A_3A_4 - (1-\beta_7)(1-\beta_8)) + z_3\beta_8(\beta_4 + 1) + A_4(z_3\beta_6 + \beta_3\beta_7) + A_2A_3\beta_5 + A_3z_3\beta_6] + (1-\beta_7)E_M\beta_1(A_2z_5\beta_3\beta_8 + A_6z_3z_4\beta_4) + E_Mz_3\beta_1\beta_{12}\beta_4(\beta_8\beta_9 - A_3\beta_{10}) + ((A_2A_3A_5 - z_4\beta_4) + (z_4\beta_4\beta_8 - A_3z_5\beta_6 - z_5\beta_4\beta_8))A_6(E_M\beta_1(A_1 - S\beta_1 - z_2) + \mu(A_1 - S\beta_1)) + E_M\beta_1[(A_2A_5A_6 - \beta_6\beta_{11}\beta_{12})z_4\beta_3 + (z_5\beta_5 + z_4\beta_3)(A_2A_4A_6 - \beta_4\beta_{10}\beta_{12})] + (A_2A_5 - z_5\beta_6)A_6A_4[E_M\beta_1(A_1 - S\beta - z_2) + \mu(A_1 - S\beta_1)] + (A_1 - S\beta)[z_1\{\beta_{12}(\beta_4\beta_8\beta_9 - \beta_4\beta_{10}A_3 - \beta_6\beta_{11}A_3) + (A_2A_3A_5A_6 - \beta_4\beta_8\beta_{11}\beta_{12} - \beta_4\beta_9\beta_{12}) + (A_2A_4A_5A_6 - A_5\beta_4\beta_{10}\beta_{12} - A_4\beta_6\beta_{11}\beta_{12})\}] + E_Mz_5\beta_1\beta_3\beta_6\beta_9\beta_{12} + E_M\beta_1\beta_{12}(A_2\beta_3\beta_7\beta_{10} - A_5z_3\beta_4\beta_{10} - A_2\beta_3\beta_7\beta_{11}) + A_6E_M\beta_1A_2z_5\beta_3\beta_7 + A_2A_4E_M\beta_1\beta_3(A_5z_4 - \beta_9\beta_{12}) + E_M\beta_1\beta_{12}(\beta_3(\beta_7\beta_8\beta_{11} - A_2\beta_{10} - A_5\beta_{10}) + (A_5\beta_3\beta_7\beta_{10} - z_3\beta_4\beta_9 - A_2A_5\beta_3\beta_9)) + A_4E_M\beta_1(A_2(z_5\beta_3\beta_7 - \beta_5\beta_{11}\beta_{12}) + A_5\beta_3(A_6z_4 - \beta_9\beta_{12})) + z_5E_M\beta_1\beta_8(A_3z_3\beta_4 - A_6\beta_3\beta_7)$$

$$c_7 = (A_3A_4 - (1-\beta_7)(1-\beta_8))(A_1 - S\beta_1)[(A_2A_5 - z_5\beta_6)(E_M\beta_1 + \mu)A_6 + z_1(A_2A_5A_6 - \beta_6\beta_{11}\beta_{12})] + (A_3A_4 - (1-\beta_7)(1-\beta_8))E_M\beta_1((A_6z_5 - \beta_{11}\beta_{12})(A_2\beta_5 + z_3\beta_6) - A_6z_2(A_2A_5 - z_5\beta_6)) + A_6\beta_4(\mu(A_1 - S\beta_1) + (A_1 - z_2)\beta_1E_M)((A_5z_4\beta_8 - z_5\beta_7) + (z_5\beta_7\beta_8 - A_3z_5\beta_8 - A_5z_4)) + E_M\beta_1(\beta_3\beta_7\beta_{10}\beta_{12}(A_2A_5 - z_5\beta_6) + (1-\beta_8)\beta_4((A_6z_5\beta_7 + A_5z_4)(S\beta_1 + z_3) + z_4\beta_5\beta_{11}\beta_{12}) + (A_6z_4 - \beta_9\beta_{12})A_2A_4A_5\beta_3 + (A_6z_5 - \beta_{11}\beta_{12})A_2\beta_3(A_4\beta_7 + \beta_8) + A_4\beta_3\beta_6\beta_{12}(z_5\beta_9 - z_4\beta_3) + (\beta_8\beta_9 - A_3\beta_{10})\beta_4\beta_{12}(z_5\beta_5 + A_5z_3) + \beta_4\beta_{12}(z_5\beta_8\beta_9 - z_3\beta_7\beta_{11} - z_5\beta_5\beta_9) + \beta_4\beta_8\beta_{11}\beta_{12}(z_3\beta_7 - z_4\beta_3 - A_3z_5) + \beta_{12}(\beta_3\beta_{10}(\beta_6z_5 - \beta_4A_5z_4 - \beta_4\beta_7z_5) + (\beta_3\beta_7\beta_8\beta_{11}A_2 - \beta_3\beta_{10}A_2A_5 - \beta_4\beta_9A_5z_3))) + A_6z_5\beta_1\beta_8(A_3z_3\beta_4 - A_2\beta_7\beta_8) + z_1\beta_4\beta_{12}(A_1 - S\beta_1)\{(\beta_7\beta_8\beta_{11} - \beta_{10}A_3A_5 - \beta_9A_5) + (A_5\beta_8\beta_9 - \beta_8\beta_{11}A_3 - \beta_7\beta_{11})\} + A_3A_6E_MSz_5\beta_1^2\beta_4\beta_8$$

with, c_1, c_2, c_3, c_4, c_5, c_6, c_7>0 if

$$z_2 < 0, A_1 - S\beta_1 > 0, (A_3A_4 - (1-\beta_7)(1-\beta_8)) > 0,$$

$$\beta_7\beta_8 > \left\{ \begin{array}{l} \frac{(z_5\beta_8A_3 + z_4A_5)}{z_5}, \frac{(A_3\beta_6 + \beta_4\beta_8)z_5\beta_7}{z_4\beta_4}, \frac{(\beta_{10}A_3A_5 + A_5\beta_9)}{\beta_{11}}, \frac{\beta_7(A_3\beta_4\beta_{10} + A_3\beta_6\beta_{11})}{\beta_4\beta_9}, \\ \frac{\beta_6\beta_7\beta_{11}\beta_{12}}{z_5\beta_4}, \frac{A_3\beta_7\beta_{10}}{\beta_9}, \frac{(A_2 + A_5)\beta_{10}}{\beta_{11}}, \frac{A_3z_5\beta_8 + z_4\beta_3\beta_8}{z_3}, \frac{(A_2\beta_3\beta_7\beta_{11} + A_5z_3\beta_4\beta_{10})\beta_8}{\beta_3\beta_{10}A_2}, \\ \frac{(A_2A_5\beta_3\beta_9 + z_3\beta_4\beta_9)\beta_8}{\beta_3\beta_{10}A_5}, \frac{(A_2A_5\beta_3\beta_{10} + A_5z_3\beta_4\beta_9)}{\beta_3\beta_{11}A_2}, \frac{\beta_5\beta_8\beta_{11}\beta_{12}}{z_5\beta_3} \end{array} \right\},$$

$$A_2A_5 > \max\left\{ \begin{array}{l} z_5\beta_6, \frac{z_5\beta_4}{A_3}, \frac{\beta_6\beta_{11}\beta_{12}}{A_6}, \frac{\beta_4\beta_6\beta_{11}\beta_{12} + A_5\beta_4\beta_{10}\beta_{12}}{A_4A_6}, \frac{\beta_4\beta_{12}(\beta_8\beta_{11} + \beta_9)}{A_3A_6}, \\ \frac{\beta_{11}(\beta_8A_3 + \beta_7)A_2}{\beta_8\beta_9}, \frac{A_2\beta_5\beta_{12}\beta_{11}}{A_3A_6}, \frac{A_5\beta_{10}\beta_{12}}{A_4z_4} \end{array} \right\}$$

$$A_5z_4 > \max\left\{\beta_9\beta_{12}, \frac{z_5\beta_7}{\beta_8}, \frac{A_5\beta_9\beta_{12}}{A_6}\right\}, A_3z_3\beta_4 > \max\left\{A_6\beta_3\beta_7, A_2\beta_7\beta_8\right\},$$

$$z_5\beta_6 > \max\left\{\frac{\beta_4\beta_{10}\beta_{12}}{A_4}, \frac{\beta_6\beta_{11}\beta_{12}}{A_6}, \frac{z_4\beta_3\beta_6}{\beta_9}, \frac{(\beta_7\beta_{11}z_3 + \beta_5\beta_9z_5)\beta_6}{\beta_8\beta_9}, \beta_4A_5z_4 + \beta_4\beta_7z_5\right\}$$

Then, by Routh Hurwitz criterion, endemic equilibrium point is said to be asymptotically stable.

Global Stability

In this section we will study global stability of the co-infectious model.

Theorem 4: The disease-free equilibrium point $E^0\left(\frac{B}{\mu},0,0,0,0,0,0\right)$ is globally asymptotically stable.

Proof: Consider Lyapunov Function L^0(t) as

$$L^0(\mathrm{t}) = \mathrm{E}_M(\mathrm{t}) + E_D(\mathrm{t}) + \mathrm{I}_M(\mathrm{t}) + \mathrm{I}_D(t) + \mathrm{I}_{MD}(\mathrm{t}) + \mathrm{R}(\mathrm{t})$$

$$\begin{aligned}\frac{dL^0}{dt} &= (E'_M + E'_D + I'_M + I'_D + I'_{MD} + R')(\mathrm{t})\\ &= \beta_1 S E_M + S\lambda_D - \beta_{12} R - \mu(\mathrm{E}_M + E_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) - \mu_D(\mathrm{I}_D + \mathrm{I}_{MD})\\ &= B - \mu S - \mu(\mathrm{E}_M + E_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) - \mu_D(\mathrm{I}_D + \mathrm{I}_{MD})\\ &= -(\mu(\mathrm{E}_M + E_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) + \mu_D(\mathrm{I}_D + \mathrm{I}_{MD})) < 0\end{aligned}$$

$\frac{dL^0}{dt} = 0$ if E_M+E_D+I_M+I_D+I_{MD}+R=0. Here, all the roots of the system of equation has a condition which Approaches to E^0 as $t\to\infty$. Then by LaSalle's Invariance Principle (LaSalle,1976), disease free equilibrium point is globally stable.

Theorem 5: Malaria free equilibrium point E^1 is asymptotically stable.

Proof: Let us assume Lyapunov Function L^1 as

$$L^1(\mathrm{t}) = S(t) + \mathrm{E}_M(\mathrm{t}) + E_D(\mathrm{t}) + \mathrm{I}_M(\mathrm{t}) + \mathrm{I}_D(t) + \mathrm{I}_{MD}(\mathrm{t}) + \mathrm{R}(\mathrm{t})$$

$$\begin{aligned}\frac{dL^1}{dt} &= (\mathrm{S}' + E'_M + E'_D + I'_M + I'_D + I'_{MD} + R')(\mathrm{t})\\ &= B - \mu(\mathrm{S} + E_M + \mathrm{E}_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) - \mu_D \mathrm{I}_D - \mu_D I_{MD}\\ &= B - \mu E_M - \mu(\mathrm{S} + \mathrm{E}_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) - \mu_D(\mathrm{I}_D + I_{MD})\end{aligned}$$

$\frac{dL^1}{dt} < 0$ when $B - \mu(\mathrm{S} + \mathrm{E}_D + \mathrm{I}_M + \mathrm{I}_D + \mathrm{I}_{MD} + \mathrm{R}) - \mu_D(\mathrm{I}_D + I_{MD}) < 0$

Here, all the roots of the system of equation has a condition which Approaches to E^1 as $t \to \infty$. Then by LaSalle's Invariance Principle (LaSalle,1976), disease free equilibrium point is globally stable.

Theorem 6: The endemic equilibrium point E^* is asymptotically stable.
Proof: Let us assume Lyapunov Function L^* as

$$L^*(t) = \frac{1}{2}[(S-S^*) + (E_M - E_M^*) + (E_D - E_D^*) + (I_M - I_M^*) + (I_D - I_D^*) + (I_{MD} - I_{MD}^*) + (R-R^*)]^2$$

$$\begin{aligned}\frac{dL^*}{dt} &= [(S-S^*) + (E_M - E_M^*) + (E_D - E_D^*) + (I_M - I_M^*) + (I_D - I_D^*) + (I_{MD} - I_{MD}^*) + (R-R^*)] \\ &\quad [S' + E_M' + E_D' + I_M' + I_D' + I_{MD}' + R'] \\ &= [(S-S^*) + (E_M - E_M^*) + (E_D - E_D^*) + (I_M - I_M^*) + (I_D - I_D^*) + (I_{MD} - I_{MD}^*) + (R-R^*)] \\ &\quad [B - \mu(S + E_M + E_D + I_M + I_D + I_{MD} + R) - \mu_D(I_D + I_{MD})] \\ &= -\mu[(S-S^*) + (E_M - E_M^*) + (E_D - E_D^*) + (I_M - I_M^*) + (I_D - I_D^*) + (I_{MD} - I_{MD}^*) + (R-R^*)]^2\end{aligned}$$

where, $B = \mu(S^* + E_M^* + E_D^* + I_M^* + I_D^* + I_{MD}^* + R^*)$

Here, $\frac{dL^*}{dt} \leq 0$ .Hence by LaSalle Invariance principle the endemic equilibrium point is globally stable.

Malaria-Only Sub-Model

Stability analysis for malaria-only model will be studied and necessarily stability condition will be established.

Local Stability

Local stability of the malaria-only model is calculated about two of its equilibrium points i.e. disease free E_M^0 and endemic point E_M^* .Jacobian matrix is formed using system of non-linear differential equations given in (3).

$$J_M = \begin{bmatrix} -E_M\beta_1 - \mu & -\beta_1 S & 0 & \beta_{12} \\ \beta_1 E_M & S\beta_1 - \beta_3 - \mu & 0 & 0 \\ 0 & \beta_3 & -\mu - \beta_9 & 0 \\ 0 & 0 & \beta_9 & -\mu - \beta_{12} \end{bmatrix} \tag{12}$$

Stability is carried out using Routh-Hurwitz criteria (Routh 1877), if all the condition of Routh-Hurwitz is fulfilled the system is said to be locally asymptotically stable.

Theorem 7: The disease-free equilibrium point E_M^0 is locally stable if $\mu - \frac{B\beta_1}{\mu} + \beta_3 > 0$.

Proof: The Jacobian matrix for disease free equilibrium point is given by

$$J_M^0 = \begin{bmatrix} -\mu & -\frac{B\beta_1}{\mu} & 0 & \beta_{12} \\ 0 & \frac{B\beta_1}{\mu} - \beta_3 - \mu & 0 & 0 \\ 0 & \beta_3 & -\mu - \beta_9 & 0 \\ 0 & 0 & \beta_9 & -\mu - \beta_{12} \end{bmatrix} \tag{13}$$

The characteristic polynomial for the J_M^0 is

$$\lambda^4 + d_1\lambda^3 + d_2\lambda^2 + d_3\lambda + d_4$$

where,

$$d_1 = \beta_{12} + \beta_9 + 3\mu + (\mu - \frac{B\beta_1}{\mu} + \beta_3)$$

$$d_2 = (\mu - \frac{B\beta_1}{\mu} + \beta_3)(\beta_{12} + \beta_9 + \beta_3) + \beta_{12}\beta_9 + 2\beta_{12}\mu + 2\beta_9\mu + 3\mu^2$$

$$d_3 = (\mu - \frac{B\beta_1}{\mu} + \beta_3)(\beta_{12}\beta_9 + 2\beta_{12}\mu + 2\beta_9\mu + 3\mu^2) + (\beta_9 + \mu)(\beta_{12}\mu + \mu^2)$$

$$d_4 = (\beta_9 + \mu)(\beta_{12}\mu + \mu^2)(\mu - \frac{B\beta_1}{\mu} + \beta_3)$$

Here $d_1,d_2,d_3,d_4>0$ if $\mu-\frac{B\beta_1}{\mu}+\beta_3>0$, then E_M^0 equilibrium point is said to be locally stable.

Theorem 8: The endemic point E_M^* is said to be locally asymptotically stable if $\mu–S\beta_1+\beta_3>0$.

Proof: The characteristic polynomial of the Jacobian matrix (12) is

$$\lambda^4+e_1\lambda^3+e_2\lambda^2+e_3\lambda+e_4$$

where,

$$e_1=E_M\beta_1+\beta_{12}+\beta_9+3\mu+(\mu-S\beta_1+\beta_3)$$

$$e_2=E_M\beta_1(\beta_{12}+\beta_9+\beta_3+3\mu)$$
$$+(\mu-S\beta_1+\beta_3)(\beta_{12}+\beta_9+\beta_3)+(\beta_9+\mu)(\beta_{12}+2\mu)+(\beta_{12}\mu+\mu^2)$$

$$e_3=E_M\beta_1(\beta_{12}\beta_3+\beta_{12}\beta_9+2\beta_{12}\mu+\beta_3\beta_9+2\beta_3\mu+2\beta_9\mu+3\mu^2)$$
$$+(\mu-S\beta_1+\beta_3)((\beta_9+\mu)(\beta_{12}+2\mu)+(\beta_{12}\mu+\mu^2))+(\beta_9+\mu)(\beta_{12}\mu+\mu^2)$$

$$e_4=E_M\beta_1(\beta_{12}\beta_3\mu+\beta_{12}\beta_9\mu+\beta_{12}\mu^2+\beta_3\beta_9\mu^2+\beta_3\mu^2+\mu^3)$$
$$+(\beta_9+\mu)(\beta_{12}\mu+\mu^2)(\mu-S\beta_1+\beta_3)+E_M\beta_1\beta_9\mu^2$$

Here $e_1,e_2,e_3,e_4>0$ if $\mu–S\beta_1+\beta_3>0$. Then E_M^* is said to be locally asymptotically stable.

Global Stability

Here global stability of malaria free system is studied by assuming some Lyapunov function.

Theorem 9: The disease-free equilibrium point $E_M^0\left(\frac{B}{\mu},0,0,0\right)$ is globally asymptotically stable.

Proof: let us consider Lyapunov function L_M^0 as

$$L_M^0 = E_M + I_M + R$$

$$\begin{aligned}\frac{dL_M^0}{dt} &= E_M^{'} + I_M^{'} + R^{'} \\ &= S\beta_1 E_M - \beta_{12} R - \mu(\mathrm{E}_M + \mathrm{I}_M + \mathrm{R}) \\ &= B - \mu S - \mu(\mathrm{E}_M + \mathrm{I}_M + \mathrm{R}) \\ &= -\mu(\mathrm{E}_M + \mathrm{I}_M + \mathrm{R})\end{aligned}$$

Here, $\frac{dL_M^0}{dt} < 0$ and $\frac{dL_M^0}{dt} = 0$ if E_M+I_M+R=0. Here all the roots of the system of equation has a condition which Approaches to E_M^0 as $t \to \infty$. Then by LaSalle's Invariance Principle (LaSalle,1976), disease free equilibrium point is globally stable.

Theorem 10: The endemic equilibrium point E_M^* is asymptotically stable.
Proof: Let us assume Lyapunov Function L_M^* as

$$L_M^* = \frac{1}{2}[(\mathrm{S} - \mathrm{S}^*) + (\mathrm{E}_M - \mathrm{E}_M^*) + (\mathrm{I}_M - I_M^*) + (\mathrm{R} - \mathrm{R}^*)]^2$$

$$\begin{aligned}\frac{dL_M^*}{dt} &= [(\mathrm{S} - \mathrm{S}^*) + (\mathrm{E}_M - \mathrm{E}_M^*) + (\mathrm{I}_M - I_M^*) + (\mathrm{R} - \mathrm{R}^*)][\mathrm{S}^{'} + E_M^{'} + I_M^{'} + R^{'}] \\ &= [(\mathrm{S} - \mathrm{S}^*) + (\mathrm{E}_M - \mathrm{E}_M^*) + (\mathrm{I}_M - I_M^*) + (\mathrm{R} - \mathrm{R}^*)][B - \mu(S + \mathrm{E}_M + \mathrm{I}_M + \mathrm{R})] \\ &= -\mu[(\mathrm{S} - \mathrm{S}^*) + (\mathrm{E}_M - \mathrm{E}_M^*) + (\mathrm{I}_M - I_M^*) + (\mathrm{R} - \mathrm{R}^*)]^2\end{aligned}$$

where, $B = \mu(S^* + \mathrm{E}_M^* + \mathrm{I}_M^* + \mathrm{R}^*)$

Here, $\frac{dL_M^*}{dt} \leq 0$. Hence by LaSalle Invariance principle the endemic equilibrium point is globally stable.

Dengue-Only Sub-Model

Stability analysis is performed for the dengue-only model. local and global stabilities are established.

Local Stability

Local stability is obtained by using Routh-Hurwitz criteria. Jacobian matrix is calculated for system (4) as follows

$$J_D = \begin{bmatrix} -E_D\gamma - \mu & S\gamma & 0 & \beta_{12} \\ E_D\gamma & S\gamma - \beta_4 - \mu & 0 & 0 \\ 0 & \beta_4 & -\mu - \mu_D - \beta_{10} & 0 \\ 0 & 0 & \beta_{10} & -\mu - \beta_{12} \end{bmatrix} \tag{14}$$

Theorem 11: The disease-free equilibrium point E_D^0 is considered to be locally asymptotically stable if $\mu - \frac{B\beta_1}{\mu} + \beta_4 > 0$.

Proof: The Jacobian matrix for dengue free equilibrium point is

$$J_D^0 = \begin{bmatrix} -\mu & \frac{B\gamma}{\mu} & 0 & \beta_{12} \\ 0 & \frac{B\gamma}{\mu} - \beta_4 - \mu & 0 & 0 \\ 0 & \beta_4 & -\mu - \mu_D - \beta_{10} & 0 \\ 0 & 0 & \beta_{10} & -\mu - \beta_{12} \end{bmatrix} \tag{15}$$

The characteristic polynomial for Jacobian matrix (15) is

$$\lambda^4 + f_1\lambda^3 + f_2\lambda^2 + f_3\lambda + f_4$$

where,

$$f_1 = \mu_D + \beta_{10} + \beta_{12} + 3\mu + (\mu - \frac{B\gamma}{\mu} + \beta_4)$$

$$f_2 = (\mu_D + \beta_{10} + \beta_{12} + 3\mu)(\mu - \frac{B\gamma}{\mu} + \beta_4) + (\beta_{12} + 2\mu)(\mu_D + \mu + \beta_{10}) + (\beta_{12}\mu + \mu^2)$$

$$f_3 = ((\beta_{12} + 2\mu)(\mu_D + \mu + \beta_{10}) + (\beta_{12}\mu + \mu^2))(\mu - \frac{B\gamma}{\mu} + \beta_4) + (\mu_D + \mu + \beta_{10})(\beta_{12}\mu + \mu^2)$$

$$f_4 = (\mu_D + \mu + \beta_{10})(\beta_{12}\mu + \mu^2)(\mu - \frac{B\gamma}{\mu} + \beta_4)$$

Here, $f_1, f_2, f_3, f_4 > 0$ if $\mu - \frac{B\gamma}{\mu} + \beta_4 > 0$ .Hence system is asymptotically locally stable.

Theorem 12: The endemic equilibrium point E_D^* is locally asymptotically stable if $\mu - S\gamma + \beta_4 > 0$.

Proof: The characteristic polynomial for J_D is

$$\lambda^4 + g_1\lambda^3 + g_2\lambda^2 + g_3\lambda + g_4$$

Here,

$$g_1 = E_D\gamma + \mu_D + \beta_{10} + \beta_{12} + 3\mu + (\mu - S\gamma + \beta_4)$$

$$g_2 = E_D\gamma(\mu_D + \beta_{10} + \beta_{12} + \beta_4 + 3\mu) + (\mu_D + \beta_{10} + \beta_{12} + 3\mu)(\mu - S\gamma + \beta_4) + (\beta_{12} + 2\mu)(\mu_D + \mu + \beta_{10}) + (\beta_{12}\mu + \mu^2)$$

$$g_3 = E_D\gamma((2\mu + \beta_{12} + \beta_4)(\mu_D + \mu + \beta_{10}) + (\beta_4 + \mu)(\beta_{12} + \mu)) + ((\beta_{12} + 2\mu)(\mu_D + \mu + \beta_{10}) + (\beta_{12}\mu + \mu^2))(\mu - S\gamma + \beta_4) + (\mu_D + \mu + \beta_{10})(\beta_{12}\mu + \mu^2)$$

$$g_4 = E_D\gamma((\beta_{12} + \beta_4 + \mu)\mu(\mu_D + \mu + \beta_{10}) + \beta_4\beta_{12}(\mu + \mu_D)) + (\mu_D + \mu + \beta_{10})(\beta_{12}\mu + \mu^2)(\mu - S\gamma + \beta_4)$$

Now, $g_1,g_2,g_3,g_4>0$ if $\mu-S\gamma+\beta_4>0$, by Routh -Hurwitz the system is locally stable.

Global Stability

Here global stability of dengue free system is studied by assuming some Lyapunov function.

Theorem 13: The disease-free equilibrium point $E_D^0\left(\frac{B}{\mu},0,0,0\right)$ is globally asymptotically stable.

Proof: let us consider Lyapunov function L_D^0 as

$$L_D^0 = E_D + I_D + R$$

$$\begin{aligned}\frac{dL_D^0}{dt} &= E_D' + I_D' + R' \\ &= S\gamma E_D - \beta_{12}R - \mu(\mathrm{E}_D + \mathrm{I}_D + \mathrm{R}) - \mu_D I_D \\ &= B - \mu S - \mu(\mathrm{E}_D + \mathrm{I}_D + \mathrm{R}) - \mu_D I_D \\ &= -\{\mu(\mathrm{E}_D + \mathrm{I}_D + \mathrm{R}) + \mu_D I_D\} < 0\end{aligned}$$

Here, $\frac{dL_D^0}{dt} = 0$ if $E_D+I_D+R=0$. Here all the roots of the system of equation (4) has a condition which approaches to dengue disease free equilibrium point as $t\to\infty$. Then by LaSalle's Invariance Principle (LaSalle,1976), disease free equilibrium point is globally stable.

Theorem 14: The endemic equilibrium point E_D^* is asymptotically stable.

Proof: Let us assume Lyapunov Function L_D^* as

$$L_D^* = \frac{1}{2}[(\mathrm{S}-\mathrm{S}^*) + (\mathrm{E}_D - \mathrm{E}_D^*) + (\mathrm{I}_D - I_D^*) + (\mathrm{R}-\mathrm{R}^*)]^2$$

$$\frac{dL_D^*}{dt} = [(\mathrm{S}-\mathrm{S}^*)+(\mathrm{E}_D-\mathrm{E}_D^*)+(\mathrm{I}_D-I_D^*)+(\mathrm{R}-\mathrm{R}^*)][\mathrm{S}^{'}+E_D^{'}+I_D^{'}+R^{'}]$$
$$= [(\mathrm{S}-\mathrm{S}^*)+(\mathrm{E}_D-\mathrm{E}_D^*)+(\mathrm{I}_D-I_D^*)+(\mathrm{R}-\mathrm{R}^*)][B-\mu(S+\mathrm{E}_D+\mathrm{I}_D+\mathrm{R})]$$
$$= -\mu[(\mathrm{S}-\mathrm{S}^*)+(\mathrm{E}_D-\mathrm{E}_D^*)+(\mathrm{I}_D-I_D^*)+(\mathrm{R}-\mathrm{R}^*)]^2$$

Where, $B = \mu(S^* + \mathrm{E}_D^* + \mathrm{I}_D^* + \mathrm{R}^*)$

Here, $\frac{dL_D^*}{dt} \le 0$. Hence by LaSalle Invariance principle the endemic equilibrium point is globally stable.

NUMERICAL SIMULATION

In this section numerical simulation is studied for all the three models.

From Figure 4 it is observed that out of 20 susceptible, 15 (approx.) individuals get exposed to malaria in almost less than half a week and around 5 individual gets exposed to dengue. Individuals exposed to dengue also become infectious to dengue at the same time. Co-infection occurs to 5 individual who has malaria. Individuals exposed to Malaria get cured after consulting doctor but dengue being infectious needs proper medication. Co-infected individuals after treatment joins susceptible class.

Figure 4. Transmission of Malaria-Dengue co-infection

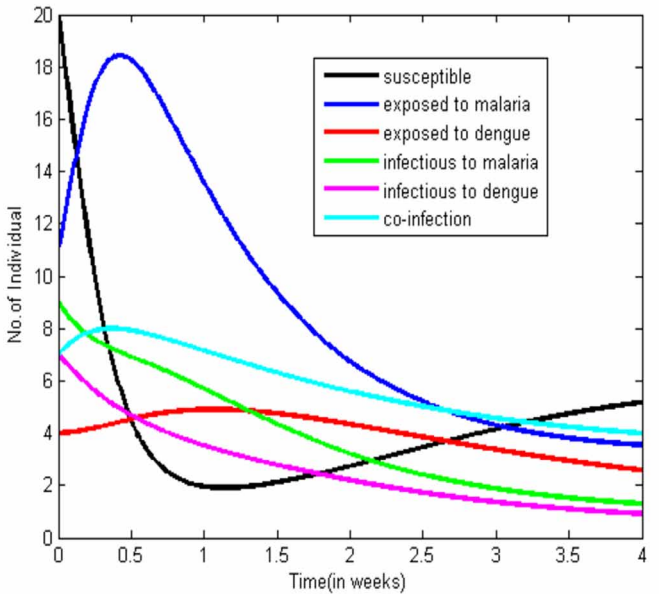
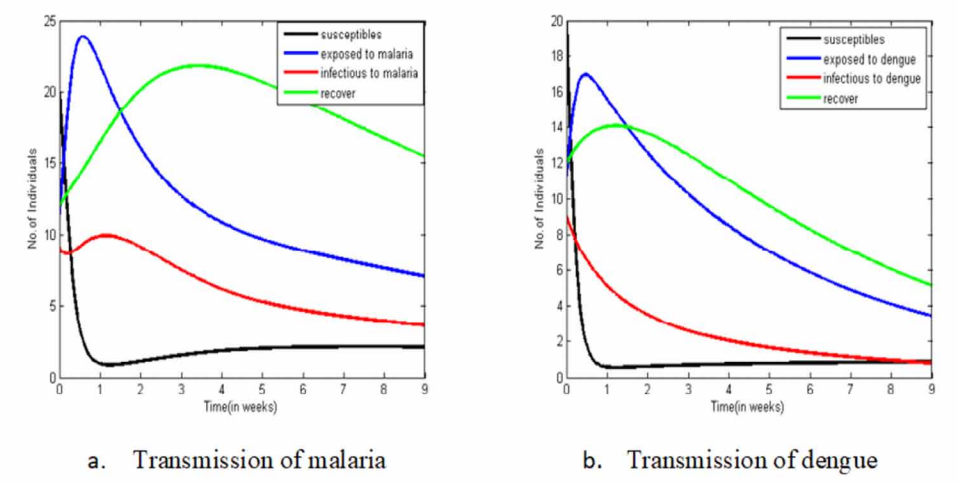


Figure 5(a) shows that in almost less than half a week approximately 15 individuals get exposed to malaria from susceptible class and among them 8 becomes infectious

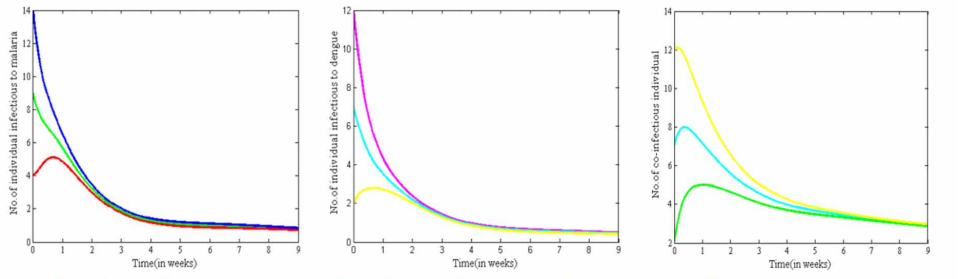
Figure 5.



a. Transmission of malaria b. Transmission of dengue

to malaria. After some time, 18 individuals exposed to malaria gets recovered in near about 1 week. From Figure 5(b), it is observed that almost 13 individuals in 0.1 weeks get exposed to dengue from susceptible individuals. 7 become infectious to dengue in 0.2 weeks and 14 individuals exposed to dengue gets recovered in 1.47 weeks.

Figure 6.



a. Variation in I_M compartment b. Variation in I_D compartment c. Variation in I_{MD} compartment

In Figure 6(a),6(b) and 6(c), it is seen that the compartments I_M, I_D and I_{MD} are asymptotically stable.

Figure 7. Intensity of exposed dengue individual versus co-infectious individual

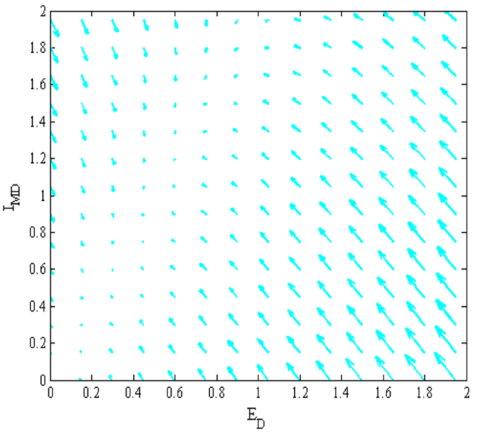
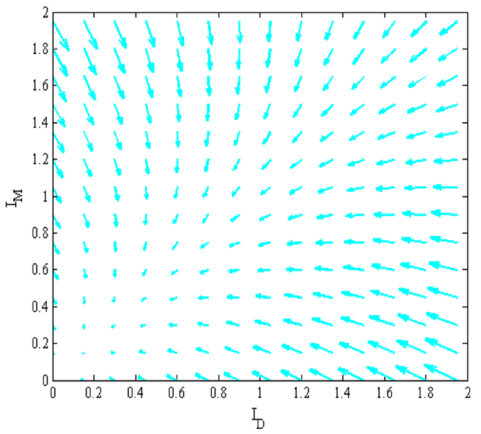


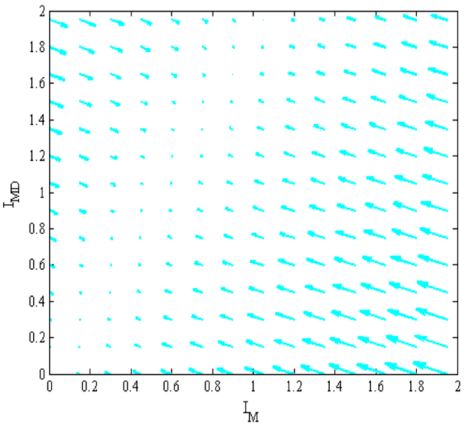
Figure 7 shows that dengue exposed individuals move towards co-infectious compartment and co-infected individuals moves towards zero i.e. they are cured.

Figure 8. Behavior of individual exposed to malaria and dengue



From Figure 8, After proper treatment individual infectious to malaria and dengue die out.

Figure 9. Transmission of infected malaria individuals towards co-infection



In Figure 9, it is observed that individuals suffering from malaria are likely to suffer from dengue also, but it also converges to zero indicating that both the diseases die out after proper medication.

CONCLUSION

In this chapter, mathematical models of malaria, dengue and its co-infection is studied. Basic reproduction number is calculated to understand the rate of disease spread. Malaria spreads at the rate of 33%, moreover 50% of the susceptible get dengue fever. Co-infection occurs to the 17% of the susceptible population. Local stability for disease free, malaria free and endemic equilibrium point is established using Routh Hurwitz criterion. It shows that the malaria and dengue infected individuals moving towards co-infection should be controlled by taking proper treatment at the initial stage or earlier. Studying global stability helps us to come to a point that growth rate should not exceed recovery rate.

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Chapter 8
Fractional-Order Model to Visualize the Effect of Plastic Pollution on Rain

Ekta N. Jayswal
Department of Mathematics, Gujarat University, Ahmedabad, India

Purvi M. Pandya
Department of Mathematics, Gujarat University, Ahmedabad, India

ABSTRACT

In this era, one of the biggest issues faced by humans is due to plastic pollution as it dwells in environment and depletes the ecosystem. This affects the climate and disturbs the chain of rain, which is the common source of obtaining water body. Also, this resulting pollution causes the toxicity in rain. Accordingly, the mathematical model is framed by considering fractional order derivative. Pollution free and endemic equilibrium points are worked out for integer order system of non-linear differential equations. Local stability of equilibrium points brings attention on dynamical behavior of model with sufficient condition. With the help of basic reproduction number, bifurcation is analyzed, which shows the chaotic nature of this model. Providing Caputo derivative of fractional order, a numerical simulation has been done by taking different values of order for the system.

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INTRODUCTION

Plastic is most flexible and ubiquitous material hence now a days it is people's essential need. World produces masses of plastic every day and million tons of plastics are used up every year out of which only one-quarter part is recycled, others going to landfills. This led to a high prominence of plastic pollution in the environment. It affects the environmental key-resources pollution in soil, water and air. Plastic pollution releases harmful toxic chemicals in surroundings and became barrier to the ecosystem via air currents which causes potentially unsafe environment. Rain toxicity is due to access of two gases sulfur dioxide and nitrogen oxides, most of it comes from burning of plastic as it contain fossil fuels. These gases react when it mixes up with water. Upcoming years have solution for this plastic pollution through dumping, burning or recycling according to the plastic category. Some people also use the fund for public health and environmental policy instead of burning and producing of plastic. Vasudevan (2010) (from Madurai, India) has patented a method to reuse plastic wastes mainly municipal solid waste to construct roads and known as "Plastic man".

Mathematical modeling of dynamical system for integer order is vast branch for formulating the epidemic disease models and its control strategies. Above mentioned details can help to form a fractional order dynamical model for plastic. The word fraction means that any arbitrary non-negative real number. Fractional differential equation is extra ordinary differential equations. This calculus is generalization of ordinary differentiation and integration to a fractional order may be real or complex. For the first time fractional calculus is introduced by Gottfried Wilhelm Leibniz in 1695. Since many years, it is used only for many branches of science and engineering viscoelastic material, electrical networks, fluid flow, rheology, diffusive transport, bioengineering, finance and also in electromagnetic theory, hence become very popular in recent years. This study is used in studying the inconsistent behavior of viscoelasticity, bioengineering chaotic system. Fractional order is used in PID (proportional, integral and derivative) controllers which increase their degree of freedom and also measures its error function. Fractional order derivative is defined using mathematical term known as gamma function. Many mathematicians like Grünwald–Letnikov, Riemann-Liouville (2014), Caputo (1967), Hadamard, Atangana-Baleanu (2016), Riesz (2014) have given the definitions about fractional order derivative in which all are mathematically acceptable as in Ahmed *et al.* (2007). In signal classification task, Gomolka (2018) has proposed neural network with back propagation rule in which fractional derivative is used. Atangana-Baleanu (2016) has established a new kernel based upon Mitag-Leffler function; this description is filter of fractional regulator and fractional derivative. They all had done very useful work in this field. In this paper, we have used Caputo derivative for observing results. It is

prevailing tool which have ability to model traditional phenomenon with long-term memory and long-term spatial interface.

Chang *et al.* (1996) formulate a model to control air pollution using dynamic optimization by mixed integer programming. Using equilibrium partitioning method Teuten *et al*. (2009) observed that how pollutants transmission from plastic to organisms. With environmental and health policy, on dynamical system Dubey (2010) elaborates the study the effect of contaminant on human population dependent on a sources. By analyzing the optimization and sensitivity methods to determine the optimal conditions for identify ecologically positive recycle routes; Song (1999) *et al.* has developed a model. Using induced fuzzy cognitive maps, Pathinathan (2014) *et al.* analyzes the extortions of plastic pollution by taking an algorithmic approach. With the concept of two-shot modeling, Donovan (1975) *et al.* prepared a mathematical model for recycling of plastic. Matlob and Jamali (2019) focused on two type Riemann-Liouville and Caputo fractional derivative to investigate viscoelastic material. Tavassoli (2013) *et al.* found that changing in area by changing the value of order of derivative. By using basic reproduction number, Salman (2017) has analyzed stability and bifurcation. Mouaouine (2018) *et al.* has considered a SIR model to check local and global stability of the fractional order system. Using generalized Euler method Selvam *et al.* (2017) developed and observed chaotic behavior epidemic model. ÖZalp and Demirci (2011) have formulated *SEIR*- vertical transmission model. El-Shahed *et al.* (2011) used fractional order SIRC dynamical model of influenza-A in human population. Matignon (1996) has investigated internal and external stability of fractional order model. Ahmed *et al.* (2007) considered stability of prey-predator model using fractional order derivative. Using center manifold theory Abdelaziz *et al.* (2018) bifurcation analysis has been conducted. Bonyah *et al.* (2019) solved a model using recently introduced method Adams-Bashforth for fractional differential equations. Vargas (2015) has developed a system of Volterra-type equation for fractional order. Yongjin *et al.* (2017) has compared the results of LADM (Laplace adomian decomposition method) and HPM (Homotopy perturbation). Li *et al.* (2007) has found chaos in Chen system of fractional order so they put control on it. Mainardi *et al.* (2012) has made survey on fractional calculus in linear viscoelastic material for an historical perspective. Using Lyapunov direct method Aguila *et al.* (2014) demonstrate stability of the model. Ravi *et al.* (2010) has derived solution for fractional differential equation with uncertainty. Metzler, R., and Klafter, J. (2000) has anomalous diffusion for transportation and non-exponential relaxation patterns. Several methods are discussed for dynamical process in complex system. Bayın (2016) shows different representation of Riesz derivative as it is plays an important role space fractional quantum mechanics and diffusion anomalous.

This whole review says the idea about how plastic waste effects on the environment and methods to reduce the pollution due to plastic by controlling it. Then it gives a notion about fractional calculus. Last the work in fractional calculus is done is shown; in which some has mathematical methods to control pollution using integer order derivative. In this paper fractional calculus is used to formulate a non-linear dynamical system for plastic pollution effects on rain toxicity.

The model related to plastic pollution is formulated in section below. Next, stability analysis has been done followed by the simulation for the proposed model. Finally, concluding the findings for the validated data.

Definition 1: The Caputo fractional order derivative of a function y in the interval $[0,T]$ is defined by,

$$ {}^{C}D_{0+}^{\alpha} y(t) = \frac{1}{\Gamma(n-\alpha)} \int_{0}^{t} (t-s)^{n-\alpha-1} y^{(n)}(s)\, ds $$

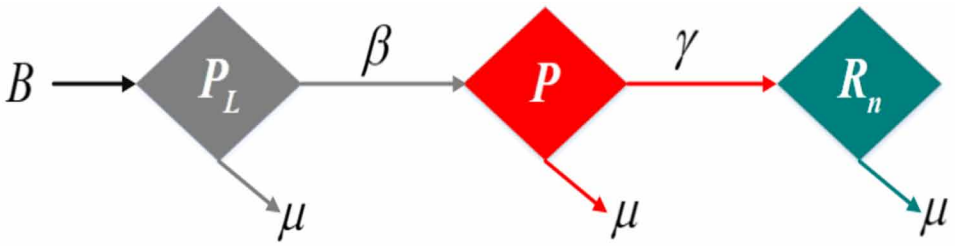
where, C represents Caputo derivative, D^{α} denotes Caputo fractional derivative of order $n=[\alpha]+1$ and $[\alpha]$ represents the integer part of α.

Definition 2: Laplace transform of Caputo derivative is defined as,

$$ L\left\{D^{\alpha} y(t)\right\} = s^{\alpha} y(s) - \sum_{k=0}^{n-1} s^{\alpha-k-1} y^{(k)}(0)\,,\ n-1<\alpha<n,\ n\in N. $$

Fractional order derivative can be applied on linear and non-linear epidemic model of either for diseases or viscoelastic material or prey-predator model. This present paper is applying the concept of fractional order derivative on environmental issues due to plastic pollution to show the vertical transmission in the model. The model related to plastic-pollution is formulated in section below. Next, stability analysis has been done followed by the simulation for the proposed model. Finally, concluding the findings for the validated data is last section.

Figure 1. Schematic diagram



MATHEMATICAL MODEL

Our tremendous attraction towards the plastic causes undeniable environmental things. Usage of millions of plastic (P_L) generates pollution (P) worldwide which emerges toxicity in the outside environment affects into the rain density (R_n) - considering this scenario; this model is established having following three compartments.

Each compartment is connected with rates and formulates a system of three non-linear differential equations.

$$\frac{dP_L}{dt} = B - \mu P_L - \beta P_L P$$

$$\frac{dP}{dt} = \beta P_L P - \gamma P - \mu P \tag{1}$$

$$\frac{dR_n}{dt} = \gamma P - \mu R_n$$

with $P_L>0$ and $P, R_n \geq 0$ where, B is growth rate, β is pollution occurring rate due to plastic, γ is toxicity rate affected on rain due to pollution and μ is disposal rate.

The feasible region for the system (1) is given by,

$$\Lambda = \left\{ (P_L, P, R_n) \in R_+^3 : P_L + P + R_n \leq \frac{B}{\mu} \right\}.$$

where,

$$R_+^3 = \left\{(P_L, P, R_n) \in R^3 : P_L > 0,\ P \geq 0,\ R_n \geq 0\right\}.$$

By accumulating the system we get two equilibrium points *i.e.*

(i) Pollution-free equilibrium point $E_0\left(\frac{B}{\mu}, 0, 0\right)$

(ii) (ii) Endemic equilibrium point

$$E_1\left(\frac{\mu+\gamma}{\beta}, \frac{B\beta-\mu(\mu+\gamma)}{\beta(\mu+\gamma)}, \frac{\gamma\left(B\beta-\mu(\mu+\gamma)\right)}{\beta\mu(\mu+\gamma)}\right)$$

After using Caputo fractional order derivative to the system one can write system as,

$$^{C}D^{\alpha_1}P_L = B - \mu P_L - \beta P_L P$$

$$^{C}D^{\alpha_2}P = \beta P_L P - \gamma P - \mu P \tag{2}$$

$$^{C}D^{\alpha_3}R_n = \gamma P - \mu R_n$$

with initial conditions $P_L(0) = P_{L_0}$, $P(0)=P_0$ and $R_n(0) = R_{n_0}$.

Then, applying laplace transform for the solution of this system (2) get,

$$P_L = P_{L_0} + L^{-1}\left[\frac{1}{s^{\alpha_1}}\left\{B - \mu P_L - \beta P_L P\right\}\right]$$

$$P = P_0 + L^{-1}\left[\frac{1}{s^{\alpha_2}}\left\{\beta P_L P - \gamma P - \mu P\right\}\right]$$

$$R_n = R_{n_0} + L^{-1}\left[\frac{1}{s^{\alpha_3}}\left\{\gamma P - \mu R_n\right\}\right]$$

General solution recursively one can get,

$$P_L(i+1) = P_L(i) + \frac{r^{\alpha_1}}{\Gamma(\alpha_1+1)}\left(B - \mu P_{L_i} - \beta P_{L_i} P_i\right)$$

$$P(i+1) = P(i) + \frac{r^{\alpha_2}}{\Gamma(\alpha_2+1)}\left(\beta P_{L_i} P_i - \gamma P_i - \mu P_i\right) \tag{3}$$

$$R_n(i+1) = R_n(i) + \frac{r^{\alpha_3}}{\Gamma(\alpha_3+1)}\left(\gamma P_i - \mu R_{n_i}\right)$$

Stability of model is analyzed through basic reproduction number denoted by R_0 [5]. This model is stable if $R_0<1$ and unstable if $R_0>1$. Defining $F(X)$ and $V(X)$ such that,

$$F(X) = \begin{bmatrix} \beta P_L P \\ 0 \\ 0 \end{bmatrix} \text{ and } V(X) = \begin{bmatrix} \mu P + \gamma P \\ -\gamma P + \mu R_n \\ -B + \mu P_L + \beta P_L P \end{bmatrix}$$

Now,

$$DF(E_0) = \begin{bmatrix} f & 0 \\ 0 & 0 \end{bmatrix} \text{ and } DV(E_0) = \begin{bmatrix} v & 0 \\ J_1 & J_2 \end{bmatrix}$$

where, f and v are 3×3 matrices defined as

$$f = \left[\frac{\partial F_i(E_0)}{\partial \mathrm{X}_j}\right] \text{ and } v = \left[\frac{\partial V_i(E_0)}{\partial \mathrm{X}_j}\right].$$

Finding f and v it becomes,

$$f = \begin{bmatrix} \beta P_L & 0 & \beta P \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{and} \, v = \begin{bmatrix} \mu+\gamma & 0 & 0 \\ -\gamma & \mu & 0 \\ \beta P_L & 0 & \beta P + \mu \end{bmatrix}$$

where, v is non-singular matrix.

Thus, using next generation matrix method; the mathematical expression for basic reproduction number R_0 is given by $\frac{\beta B}{\mu(\mu+\gamma)}$.

As $R_0 = \frac{\beta B}{\mu(\mu+\gamma)}$, one can reformulate the system (3) in terms of R_0 as follows:

$$P_L(i+1) = P_L(i) + \frac{r^{\alpha_1}}{\Gamma(\alpha_1+1)}\left(B - \mu P_{L_i} - \beta P_{L_i} P_i\right)$$

$$P(i+1) = P(i) + \frac{r^{\alpha_2}\left(\mu+\gamma\right)}{\Gamma(\alpha_2+1)}\left[\frac{R_0 P_{L_i} P_i \mu}{B} - P_i\right] \tag{4}$$

$$R_n(i+1) = R_n(i) + \frac{r^{\alpha_3}}{\Gamma(\alpha_3+1)}\left(\gamma P_i - \mu R_{n_i}\right)$$

STABILITY

For any system one can analyze the behavior of system around its equilibrium points, which is known as stability. Stability analysis is stability of solutions of the dynamical system. In this section, it is examined through locally and globally (Mouaouine *et al.*(2018)) using Jacobian matrix and Lyapunov function respectively.

Local Stability

Stability at equilibrium points is scrutinized by Jacobian matrix using system (2) around its two equilibrium points of the model. It is stable if for each λ_1 of Jacobian matrix at equilibrium point $\left|\arg(\lambda_i)\right| > \frac{\pi}{2}$. Jacobian matrix of system (2)

$$J = \begin{bmatrix} -\mu - \beta P & -\beta P_L & 0 \\ \beta P & \beta P_L - \mu - \gamma & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

Now, Jacobian matrix at both the equilibrium points is and corresponding eigenvalues are evaluated will help to derive the stability conditions.

Jacobian matrix at E_0,

$$J_0 = \begin{bmatrix} -\mu & \frac{-\beta B}{\mu} & 0 \\ 0 & \frac{\beta B}{\mu} - \mu - \gamma & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

have three eigenvalues, $\omega_1=\omega_2=$-μ and $\omega_3 = \frac{\beta B}{\mu} - (\mu + \gamma)$.

Jacobian matrix at E_1,

$$J_1 = \begin{bmatrix} -\frac{\beta B - \gamma\mu - \mu^2}{\mu + \gamma} & -\mu - \gamma & 0 \\ \frac{\beta B - \gamma\mu - \mu^2}{\mu + \gamma} & 0 & 0 \\ 0 & \gamma & -\mu \end{bmatrix}$$

have three eigenvalues, $\omega_1=$-μ and

$$\omega_2 = -\left(\frac{\frac{1}{2} B\beta \pm \sqrt{B^2\beta^2 - 4(B\beta - \gamma\mu - \mu^2)(\gamma + \mu)^2}}{\mu + \gamma} \right).$$

A condition for equilibrium points E_0 and E_1 to be locally stable is eigenvalues should be negative, so model has one condition after simplifying the eigenvalues *i.e.* $\frac{\beta B}{\mu} < \mu + \gamma$. Moreover, $|\arg(E_0)| > \frac{\pi}{2}$ and $|\arg(E_1)| > \frac{\pi}{2}$ which depicts that E_0 and E_1 are locally asymptotically stable.

Global Stability

For each equilibrium points global stability is analyzed by taking Lyapunov's function.

Theorem 1: The pollution-free equilibrium point E_0 is globally asymptotically stable whenever $R_0 \leq 1$.

Proof: Consider Lyapunov function $L_0(t) = \varphi\left(\frac{P_L}{P_{L_0}}\right) + P$, where $\varphi(x)=x-1-\ln(x)$, $x>0$. This function $\varphi(x)\geq 0$ is increasing function.

Now, by taking derivative

$$D^{\alpha} L_0(t) = \left(1 - \frac{P_{L_0}}{P_L}\right) D^{\alpha} P_L + D^{\alpha} P$$

$$\therefore D^{\alpha} L_0(t) = \left(1 - \frac{P_{L_0}}{P_L}\right)\left(B - \mu P_L - \beta P_L P\right) + \left(\beta P_L P - \gamma P - \mu P\right)$$

Using $B = \mu P_{L_0}$ we have,

$$\therefore D^{\alpha} L_0(t) \leq -\frac{\mu}{P_L}\left(P_L - P_{L_0}\right)^2 + \left(\gamma + \mu\right)\left(\frac{P_L R_0 \mu}{B} - 1\right)$$

$\therefore D^{\alpha} L_1(t) \leq 0$ at pollution-free equilibrium point E_0 with $R_0 \leq 1$. Hence, E_0 is globally asymptotically stable.

Theorem 2: The endemic equilibrium point E^* is globally asymptotically stable.

Proof: Consider Lyapunov function, $L_1(t) = \varphi\left(\frac{P_L}{P_L^*}\right) + \varphi\left(\frac{P}{P^*}\right) + \varphi\left(\frac{R_n}{R_n^*}\right)$

$$\therefore D^{\alpha} L_1(t) = \left(1 - \frac{P_L^*}{P_L}\right) D^{\alpha} P_L + \left(1 - \frac{P^*}{P}\right) D^{\alpha} P + \left(1 - \frac{R_n^*}{R_n}\right) D^{\alpha} R_n$$

$$\therefore D^{\alpha}L_1(t)=\left(1-\frac{P_L^*}{P_L}\right)\left(B-\mu P_L-\beta P_L P\right)+\left(1-\frac{P^*}{P}\right)\left(\beta P_L P-\gamma P-\mu P\right)+\left(1-\frac{R_n^*}{R_n}\right)\left(\gamma P-\mu R_n\right)$$

$$\therefore D^{\alpha}L_1(t)=\frac{-\mu}{P_L}\left(P_L-P_L^*\right)^2+\beta P_L^* P^*\left(1-\frac{P_L^*}{P_L}\right)$$
$$-\left(\frac{P^*}{P}-\frac{P_L^*}{P_L}\right)\beta P_L P-\mu P\left(1-\frac{P^*}{P}\right)-\gamma P\left(\frac{R_n^*}{R_n}-\frac{P^*}{P}\right)-\mu R_n\left(1-\frac{R_n^*}{R_n}\right)$$

$\therefore D^{\alpha}L_1(t)\leq 0$ when $P_L=P_L^*$, $P=P^*$, $R_n=R_n^*$ and $\frac{P_L^*}{P_L}=\frac{P^*}{P}=\frac{R_n^*}{R_n}<1$.

This shows that E^* is globally asymptotically stable.

NUMERICAL SIMULATION

In this section, numerical simulations to illustrate our theoretical results have been done by taking integer and fractional order to the system. Here, we are taking different values of parameters to simulate different outcomes for this discretized model and also studying the effect of parameter.

Figure 2. Transmission flow

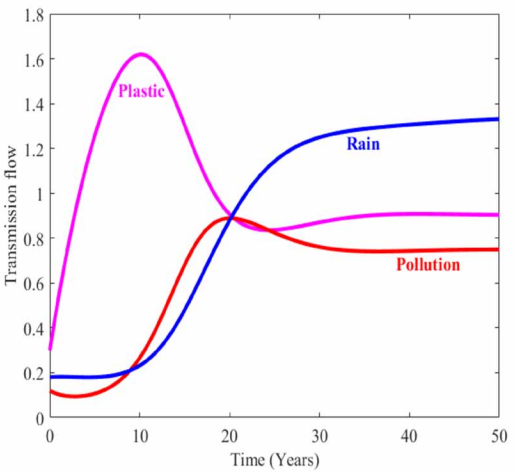
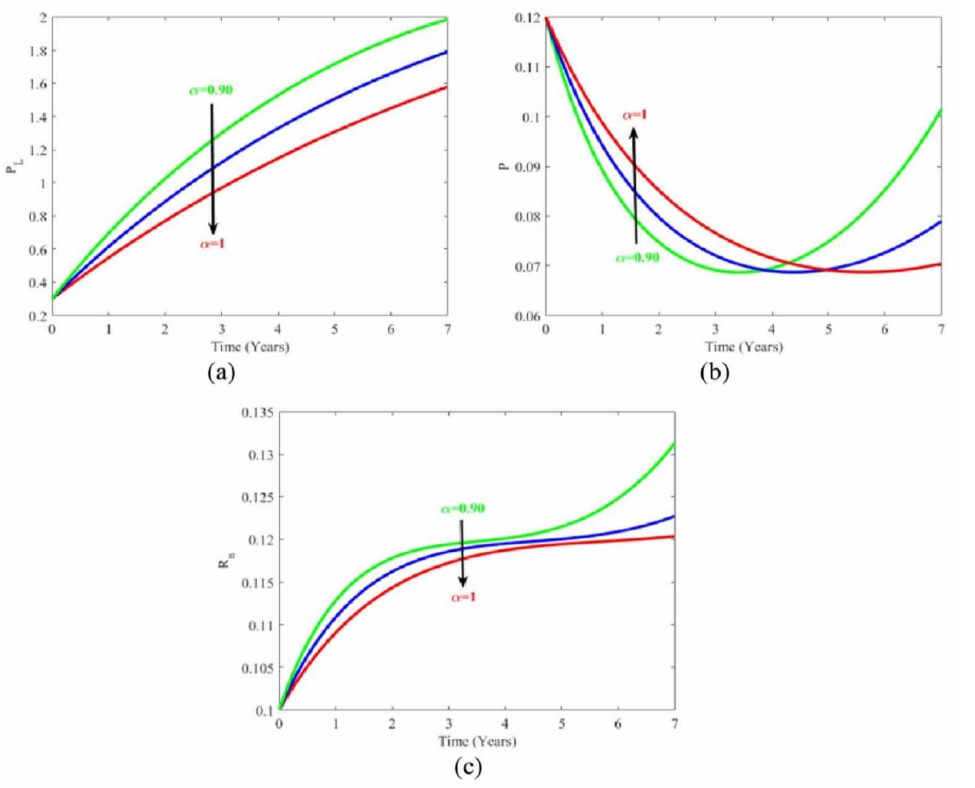


Figure 2 is framed by using parametric values B=0.30, β=0.31, γ=0.18, μ=0.10, r=0.01 and α=1 containing all three compartments. After approximately 20 years, this pollution due to plastic increases and then decreases, consequently density of rain increases as it reduce the spreading of toxicity in the environment. These parametric values suggest that if they are in their dimension then pollution will be under the control for safe environment.

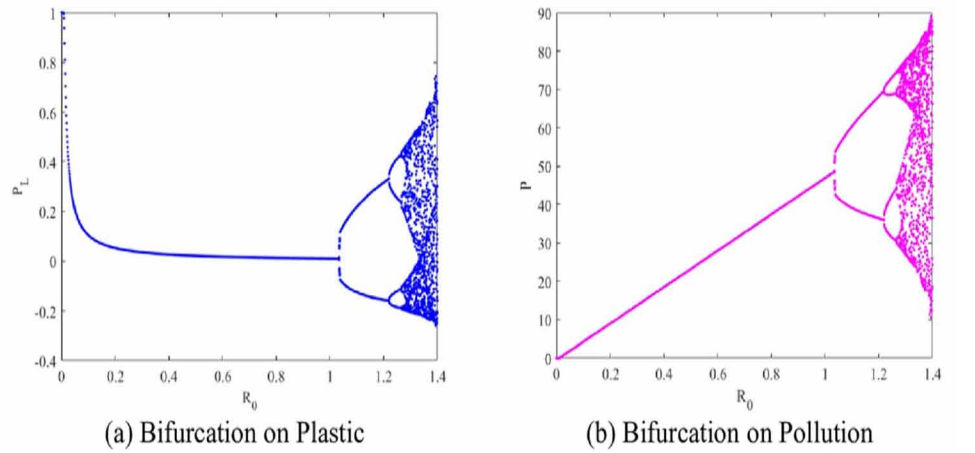
Figure 3. Compartments with different values of α



In this figure 3 (a-c), observations are simplified for each compartment *i.e.* density of plastic, pollution and rain respectively by changing the fractional value of α at β= 20%. It interprets that; by increasing the value of α, this density of pollution is reversed after certain period of time as production of plastic increases in starting phase then it gradually decreases which affecting monotonicity to the density of rain. It suggests that value of α increases by 10%, plastic density decreases by 20%, pollution increases by 12% but after approximately three years it decreases by 26%

consequently toxicity also decreases by 8%. Plastic production should be controlled to escalate intensity of rain.

Figure 4.

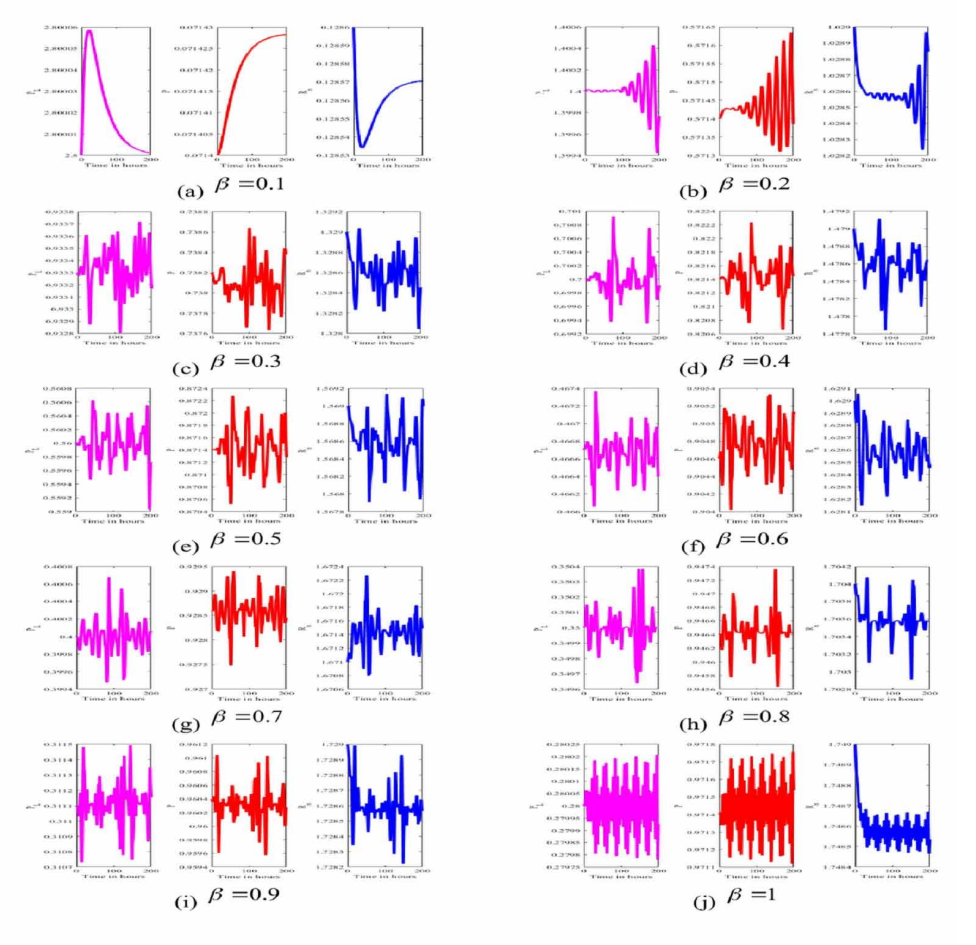


(a) Bifurcation on Plastic (b) Bifurcation on Pollution

Figure 4 has two figures, which show the bifurcation around equilibrium point of plastic and pollution respectively using the parametric values h=0.01, B=1, β=2, γ=0.18, μ=1at α=0.85. Bifurcation occurs usually when equilibrium points change their behavior from stable to unstable as small change has been taken in only one parameter. This endemic equilibrium point bifurcated at R_0>1 suggest that it loses its stability and stable solution of two period appears. Then after R_0>1.2 it again loses its stability and stable solution of four periods performs and this process will continue till the chaos. Here, figure shows the bifurcation periodically *i.e.* two-periodic, four periodic, then turns into chaos in both the situation plastic and its pollution. One can also conclude that the solution points try to approach to chaotic attractors.

Figure 5 (a-j) represents the different chaotic oscillations by changing the value of pollution occurring rate (β). These oscillations have motion which repeats itself over and over again after some interval of time. Initially started with less number of reoccurrences around its equilibrium point, and then gradually increases its period; lastly for β=1 it turns into chaos. Performed analysis seeks to show that β is very effective parameter for this epidemic system causing the rain toxicity as it primary parameter which effects during the plastic production. More plastic usage makes more system chaotic.

Figure 5. Oscillations due to change in β



CONCLUSION

The proposed problem is setup with compartmental model by considering rain issues due to plastic pollution. System of non-linear fractional order differential equations is designed by using Caputo order derivative. Basic reproduction number is derived for simulating the bifurcation results. The model is locally asymptotically stable at equilibrium point with sufficient conditions in the feasible region Λ which enhances the knowledge of fractional calculus. Numerical simulations gave effective results on different value of α and also suggest the importance of value of *h*. It suggests that this chaotic behavior of pollution is only in controlled if plastic production is regulated. Everyone should make effort in reducing the usage of plastic and promote

the recycling or to avoid its usage. Recycling is not the solution as people turn the one toxic to another. It is advisable that, best thing is to cut down the usage of plastic and carry own bags.

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Chapter 9
Control of Pest Population by Sterile Insect Technique Considering Logistic Growth With Spatial Spread Invasion and Optimal Production Policies

Sudipa Chauhan
Amity Institute of Applied Science, Amity University, Noida, India

Kuldeep Chaudhary
Amity Institute of Applied Science, Amity University, Noida, India

Prianka Bose
Department of Mathematical Sciences, New Jersey Institute of Technology, Newark, USA

Sumit Kaur Bhatia
Amity Institute of Applied Science, Amity University, Noida, India

ABSTRACT

In this chapter, the authors have proposed a SIT model to eradicate the pest population. It has been assumed that the females after mating with wild males grow logistically. Pest population is being controlled with the release of sterile insects in their habitat. The model is formulated with the system of differential equations, and the authors have discussed the local stability analysis of deterministic logistic growth

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rate model. Further, they have also obtained a potential function by incorporating one-dimensional insect release with an invasion on patch size L, which has a toxic exterior as its surrounding. It has been obtained that, in the presence of spatial spread over a finite patch size, the sterile release of the insects produces a sudden declination of the pest population. Finally, the authors have obtained the optimal production of sterile male population using Pontryagin's maximum principle. The applicability of the proposed model is finally illustrated through numerical solution.

INTRODUCTION

The sterile insect technique or more commonly known as SIT, was originally invented by (Knipling,1955) It is the process of introducing a large number of sterile male insects into a wild population so that it ceases reproduction when these sterile species mate with the female species. Hence, as an outcome, the growth rate in pest population is diminished. SIT seems to be more effective when only sterile males are released for the following reasons:

1. Males are more efficient carriers of the sterility trait, as they often copulate more than once, whereas females may be restricted in the number of their mating.
2. When only males are released, assortative mating amongst the laboratory-reared insects can be avoided, increasing the exploitation of the sterile sperm.
3. When no females are released, males disperse more rapidly in the native (wild) population.
4. Female insects might damage crops even if they are sterile, as they could cause oviposition wounds.
5. Rearing only males reduces the cost of mass-rearing, provided females can be eliminated early during the rearing process.

The objective of SIT is the interaction of females with the sterile males such that it leads to no further reproduction, and thereby, decreasing the population of the next generation. Hence, the main challenge is faced between the wild and the sterile male to mate with the female. It is further noticed that over low population density, continuous deliverance of sterile males controls the pest population, but for economic purposes dense populations are an ideal situation for pest control before the release. The introduction of large number of sterile males leads to the emergence

of Allee effect (Courchamp, Berec & Gascoigne, 2008; Liebhold & Tobin, 2008; Tobin, Berec & Liebhold, 2011) (a phenomenon in biology which is represented by the interdependence of the per capita population growth rate and population density or size of the species). SIT is preferred over insecticide application as it is harmless for farmer's health (Klassen & Curtis, 2005), does not cause pollution to the environment and is not aimed at other insect population. Along with applying SIT for pest management, it is also important to obtain the optimal rate of production of sterile male and the optimal pest population.

Optimal control theory deals with the problem of dynamic optimization and provides a powerful tool for understanding the dynamic systems. Several papers on application of optimal control theory in epidemiological models exist in the literature (Agusto, 2013; Okosun, Rachid & N.Marcus, 2013; Agusto & Adekunle, 2014; Apreutesei & Strugariu, 2014; Zhou, Liang & Wu, 2014). In fact, recently various papers have also discussed optimality of SIT technique (Sergio Ramirez & Luis F.Gordillo, 2016, Luis F. Gordillo, 2015, Luis F. Gordillo, 2014). Due to the dynamic nature of the present problem and loses due to pest damage, we formulate a optimal control problem to find an optimal production rate of sterile male population over time so that the cost of producing sterile male and feeding cost can be minimized.

Keeping in view the above discussion, in this paper, we have discussed a deterministic mathematical model in which further we incorporate spatial spread over a finite patch. In section 2, we have proposed our model followed by the dynamics of the model in section 3. In section 4, we have discussed about the spatial spread invasion (Lewis & Van Den Driessche, 1993) and the model displays non trivial solutions over the finite patches, even if sterile males released is more than the threshold required to eliminate the pest population. The optimal production policy has been discussed in section 5 which provides the optimal production rate of sterile male population with the concept of total cost minimization. Finally the numerical section is discussed in support of our analytical result.

MATHEMATICAL MODEL

The model consist of a female pest population who are sexually mature and are at post-copulatory stage that is, the female insects are not sexually responsive. We proceed with the following assumptions:

- Sterile and wild males both are extensively competitive.
- Males and females are mixed consistently.
- Mating encounter of the female is random and directly proportional to amount of total males present.

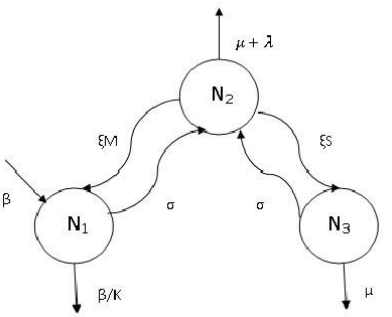
- The density of the release of sterile males is kept constant at all times.
- Immediately after the sterile males are released, they mix uniformly with the entire population.

Here, the average lifespan of the female species is significantly more than the average time used up at the stage of reproduction. This sudden sexually unresponsive behaviour is a physical response due to the substance passed on by the male species at the time of mating. Let $N=N(t)$ be the total female density at any time t and we classify female population in following 3 parts:

(i) N_1: females after mating with wild males and at reproductive stage.
(ii) N_2: females available for mating.
(ii) N_3: females after mating with sterile males and at reproductive stage.

Now, the female species in class N_2 will either come in contact with a wild male and mate or then shift to class N_1 or they come in contact and mate with a sterile male and switch toN_3. The average time of remaining in N_1 is same as N_3 with the only difference being, those in N_1 will reproduce successfully. The female in state N_1 or N_3 go back to state N_2 after the laying of eggs. Figure 1 shows the various transformations of the female species from one stage to another.

Figure 1.



We assume that a female that has accomplished at least one successful copulation with a wild male is considered to be sexually mature, and after the occurrence of the first encounter the female is included in the adult population. Accordingly, new sexually mature females are brought at a rate to stage N_1 and the females which only have encounters with sterile males their entire life or those who go missing without

having the exposure of any male, are eliminated from the dynamics. Also, K is taken as the carrying capacity and represents the average per capita rate of mating encounters, then $p = 1 - e^{-\xi M}$ denotes the probability of the female to have one successful encounter with a wild male at least once in her lifetime, with M being the number of wild males present. The small part of offspring contacting a wild male later on is given by $\beta = (birthrate) \times p$ assuming that none of the offsprings die. Let μ denote the per capita death rate, satisfying $\beta > \mu$, and let λ denote the additional death rate that defines the higher risk of the female species from leaving its home while being prepared for mating. In addition to that, $1/\sigma$ represent the average time the females spend in carrying eggs and S is the density of sterile males released.

Thus, the proposed model given by its set of equations is as follows:

$$N_1' = \beta N_1 \left(1 - N_1 / K\right) - \sigma N_1 + \xi M N_2 \tag{1}$$

$$N_2' = -\left(\mu + \lambda\right) N_2 + \sigma \left(N_1 + N_3\right) - \xi \left(M + S\right) N_2 \tag{2}$$

$$N_3' = -\mu N_3 - \sigma N_3 + \xi S N_2 \tag{3}$$

where, ' is the derivative with respect to the time, and the meaning of the parameters are explained in Table 1.

Table 1.

Parameter	*Meaning*
K	carrying capacity
S	number of sterile males
λ	additional death rate
ξ	average per capita rate of mating encounter
ξS	average rate of contact of female with sterile male
M	number of wild males
β	growth rate
$1/\sigma$	average time females spend in reproductive state
ξM	average rate of contact of female with wild male
μ	death rate

DYNAMICS OF THE MODEL

We assume wild population of the sex ratio as 1:1, and hence, we can substitute N with M in the equations. If the average lifespan is substantially more than the average time spent in carrying eggs, then, we can reduce the system of equations to a single equation which is of Holling type-II functional response with a logistic growth rate, where N denotes the total female population. As $M=N$;

$$N_1^{'} = \beta N_1\left(1-\frac{N_1}{K}\right)-\sigma N_1+\xi NN_2$$

$$N_2^{'} = -\left(\mu+\lambda\right)N_2+\sigma\left(N_1+N_3\right)-\xi\left(M+S\right)N_2$$

$$N_3^{'} = -\mu N_3-\sigma N_3+\xi SN_2$$

Since, $N=N_1+N_2+N_3$, We have

$$N' = N_!^{'}+N_2^{'}+N_3^{'}$$

Now, with, and rescaling the time,, we get;

$$\dot{N} = \frac{dN}{ds}=\frac{dN}{dt}\frac{dt}{ds}=\frac{N'}{\mu}$$

$$N' = \beta N-\left(\mu+\beta\right)N_2-\left(\mu+\beta\right)N_3-\lambda N_2-\frac{\beta}{K}(N-N_2-N_3)^2$$

$$N_2 = \frac{N_2^{'}}{\mu}=-\left(1+\frac{\lambda}{\mu}\right)N_2+\frac{\sigma}{\mu}\left(N-N_2\right)-\frac{\xi}{\mu}\left(N+S\right)N_2$$

$$N_3 = \frac{N_3^{'}}{\mu}=-N_3-\frac{\sigma}{\mu}N_3+\frac{\xi}{\mu}SN_2 \qquad (4)$$

Now, =μ/σ, then, we have,

$$\epsilon N_2 = -\left(\epsilon + \frac{\lambda}{\sigma}\right) N_2 + \left(N - N_2\right) - \frac{\xi}{\sigma}\left(N + S\right) N_2 \tag{5}$$

$$\epsilon N_3 = -\epsilon N_3 - N_3 + \frac{\xi}{\sigma} S N_2 \tag{6}$$

Let $\epsilon \to 0$, therefore, we get

$$\Rightarrow N_2 = \frac{N}{1 + \frac{\lambda}{\sigma} + \frac{\xi}{\sigma}\left(N + S\right)}$$

Again,

$$\Rightarrow N_3 = \frac{\xi}{\sigma} S \left(\frac{N}{1 + \frac{\lambda}{\sigma} + \frac{\xi}{\sigma}\left(N + S\right)} \right)$$

Therefore,

$$N' = \phi N \left(1 - \frac{\eta K\left(1 + \theta S\right) + K\alpha + N\gamma}{K\left(1 + \gamma\left(N + S\right)\right)} \right) \tag{7}$$

Where, $\phi = \beta$, $\gamma = \frac{\xi}{\sigma + \lambda}$, $\eta = \frac{\left(\mu + \beta\right)\sigma}{\beta\left(\sigma + \lambda\right)}$, $\theta = \frac{\xi}{\sigma}$ and $\alpha = \frac{\lambda\left(\sigma K + \beta\right)}{\beta K\left(\sigma + K\right)}$.

Now, from equation (7), in the following the per capita reproduction is demonstrated:

$$\frac{N'}{N}=\phi\frac{K-1}{K}\left(\frac{N+\dfrac{K\gamma S+K-\eta K\left(1+\theta S\right)-K\alpha}{\gamma\left(K-1\right)}}{N+\dfrac{K+K\gamma S}{K\gamma}}\right)$$

Thus,

$$\frac{N'}{N}=\phi\frac{K-1}{K}\left(\frac{A+N}{B+N}\right) \tag{8}$$

where A and B are the combination of the parameters with respect to S, ξ or λ. Besides the trivial stable state, equation (7) also has an unstable equilibrium N^* with $S=S_C$which is the critical sterile male density, and its relation is given by:

With $N^{'}=0$, $N=N^*$ and $S=S_C$,

$$\phi N^*\left(1-\frac{\eta K\left(1+\theta S_C\right)+K\alpha+N^*\gamma}{K\left(1+\gamma\left(N^*+S_C\right)\right)}\right)=0$$

$$\Rightarrow N^*\gamma\left(K-1\right)=S_C\left(\eta K\theta-K\gamma\right)+K\left(\eta+\alpha-1\right)$$

$$\Rightarrow N^*=\frac{K-1}{K}\left(\frac{\eta+\alpha-1}{\gamma}+\left(\frac{\eta\theta}{\gamma}-1\right)S_C\right)$$

Putting the value of η,α,γ and θ, we get,

$$N^*=\frac{K}{K-1}\left(\frac{\left(\sigma+\lambda\right)\left[\left(\sigma+K\right)K\left(\mu\sigma-\beta\lambda\right)+\lambda\left(\sigma+\lambda\right)\left(\sigma K+\beta\right)\right]}{\xi}+\frac{\mu}{\beta}S_C\right)$$

Thus we have,

$$N^*=\frac{K}{K-1}\left(\frac{\eta+\alpha-1}{\gamma}+\left(\frac{\eta\theta}{\gamma}-1\right)S_C\right)=\frac{K}{K-1}\left(\frac{\left(\sigma+\lambda\right)\left[\left(\sigma+K\right)K\left(\mu\sigma-\beta\lambda\right)+\lambda\left(\sigma+\lambda\right)\left(\sigma K+\beta\right)\right]}{\xi}+\frac{\mu}{\beta}S_C\right)$$

Consequently, if the amount of sterile males introduced (denoted by S), is greater than the critical value

$$S_C = \beta\left(\left(\frac{N^*(K-1)}{K}\right) - \frac{(\sigma+\lambda)\left((\sigma+K)K(\mu\sigma-\beta\lambda)+\lambda(\sigma+\lambda)(\sigma K+\beta)\right)}{\xi}\right)/\mu,$$

there is a noticeable declination in population of pests. When there is an unavailability of the sterile males which are dispersed, that is, S=0, then equation (7) represents the invasion which is likely to lead to failure in the formation due to inadequate mating encounter, giving rise to the non-trivial equilibrium $N^*=K(\frac{\eta+\alpha-1}{\gamma(K-1)})$ which is the threshold for the reproduction caused by Allee effect.

SPATIAL SPREAD INVASION

Further, we include a one-dimensional insect release and the probability of endurance in various patches of fixed area. With an invasion on a patch size L which has a toxic exterior as its surrounding, the boundary conditions,

$$N\left(-\frac{L}{2},t\right) = N\left(\frac{L}{2},t\right) = 0 \tag{9}$$

hold. Such a case arise when beyond the patch the insecticides are used. Independent of the spatial position, the females can find any sterile males to copulate with, the assumption of uniform dispersion of the sterile males over the occupied space. Now, the female species movement is represented by the introduction of a diffusion term $D(\partial^2N/\partial x^2)$ in equation (7) with D denoting the diffusion constant. Let us consider $\hat{x} = (\phi / D)x$ and $\hat{t} = \phi t$, and after discarding the hats, we have,

$$\frac{\partial N}{\partial t} = \phi N\left(1 - \frac{\alpha+\gamma N}{K(1+\gamma(N+S))} - \frac{\eta(1+\theta S)}{1+\gamma(N+S)}\right) + D\left(\frac{\partial^2 N}{\partial x^2}\right)$$

$$\frac{\partial N}{\partial \hat{t}}\frac{\partial \hat{t}}{\partial t} = \frac{\partial N}{\partial \hat{t}}\phi,$$

$$\frac{\partial N}{\partial x}\frac{\partial x}{\partial x} = \frac{\partial N}{\partial x}\frac{\phi}{D}$$

Removing caps, we finally obtain,

$$\frac{\partial N}{\partial t} = N\left(1 - \frac{\alpha + \gamma N}{K\left(1+\gamma\left(N+S\right)\right)} - \frac{\eta\left(1+\theta S\right)}{1+\gamma\left(N+S\right)}\right) + \left(\frac{\partial^2 N}{\partial x^2}\right)$$

Considering the steady state solutions of above equation we can obtain the conditions of spatial persistence, that is,

$$N\left(1 - \frac{\alpha + \gamma N}{K\left(1+\gamma\left(N+S\right)\right)} - \frac{\eta\left(1+\theta S\right)}{1+\gamma\left(N+S\right)}\right) + \left(\frac{\partial^2 N}{\partial x^2}\right) = 0, \textit{if } |x| < \frac{L}{2}$$

With N=0 if $|x| = \frac{L}{2}$

By initiating a new variable $v = \frac{\partial N}{\partial x}$, we derive a system of differential equations with the origin and,

$$1 - \frac{\alpha + \gamma N^*}{K\left(1+\gamma\left(N^*+S\right)\right)} - \frac{\eta\left(1+\theta S\right)}{1+\gamma\left(N^*+S\right)} = 0$$

$$\Rightarrow N^*\left(K\gamma - \gamma\right) = \eta K\theta S + \eta K - K - K\gamma S$$

$$\Rightarrow N^* = \frac{K\left(\eta - 1\right)}{\gamma\left(K-1\right)} + \frac{\alpha}{\gamma\left(K-1\right)} + \frac{SK\left(\eta\theta - \gamma\right)}{\gamma\left(K-1\right)}$$

as equilibrium points. Thus, system has two phase-plane equilibria, and .The linearization of the system about, is

$$\begin{bmatrix} N' \\ v' \end{bmatrix} = \begin{bmatrix} 0 & -1 \\ -1 & 0 \end{bmatrix}\begin{bmatrix} N \\ v \end{bmatrix}$$

The eigenvalues corresponding to (0,0) is $\lambda = \pm i$. The linearization of the system about (N^*,0) is

$$\begin{bmatrix} N' \\ v' \end{bmatrix} = \begin{bmatrix} 0 & 1 \\ -N & 0 \end{bmatrix} \begin{bmatrix} N \\ v \end{bmatrix}$$

The eigenvalues corresponding to (N^*,0) is $\lambda = \pm\sqrt{N^*}$. The first equilibrium point is a center and the second equilibrium point is clearly a saddle point.

Further, multiplying equation (4) by N' , we get,

$$N'\left(\frac{\partial^2 N}{\partial x^2}\right) + N\left(N - N^*\right)N' = 0 \tag{11}$$

Integrating the above equation with respect to *x*, we get

$$\frac{1}{2}\left(N'\right)^2 + \left(\frac{N^3}{3} - \frac{N^2}{2}N^*\right) = c \tag{12}$$

This equation can be further rewritten as

$$\frac{v^2}{2} + \left(\frac{N^3}{3} - \frac{N^2 N'}{2}\right) = C \tag{13}$$

Let us assume that we have a solution to equation (4) that satisfies the boundary conditions. Equation (13) may now be rewritten:

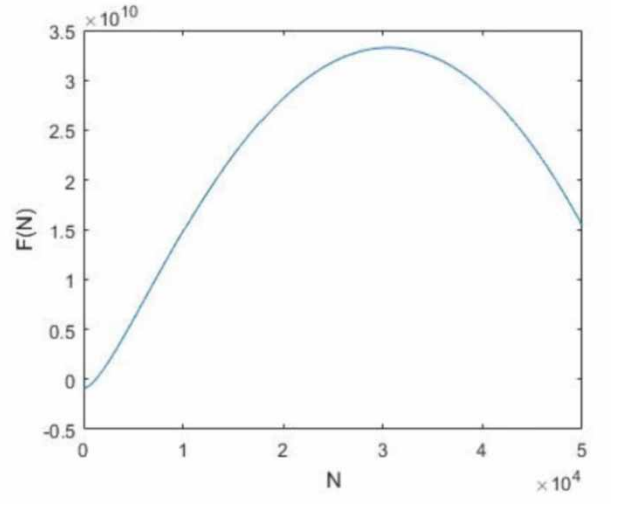
$$\frac{v^2}{2} + F\left(N\right) = F\left(\mu\right) \tag{14}$$

where u=μ when v=0 at $x = \frac{L}{2}$ .Thus,

$$v = \frac{du}{dx} = \begin{cases} +\sqrt{2}\left[F\left(\mu\right) - F\left(N\right)\right] 0 < x < \frac{L}{2} \\ -\sqrt{2}\left[F\left(\mu\right) - F\left(N\right)\right] \frac{L}{2} < x < L \end{cases}$$

If we separate above equation and integrate over the first half of the orbit, we get

Figure 2. Graph of potential function



$$\sqrt{\frac{1}{2}}\int_0^{\mu}\frac{du}{\sqrt{F(\mu)-F(N)}}=\int_0^{\frac{L}{2}}dx \tag{15}$$

Similarly, if we integrate over the second half of the orbit, we obtain

$$\sqrt{\frac{1}{2}}\int_{\mu}^{0}\frac{-dN}{\sqrt{F(\mu)-F(N)}}=\int_{\frac{L}{2}}^{L}dx \tag{16}$$

Either way,

$$L=\sqrt{2}\int_0^{\mu}\frac{dN}{\sqrt{F(\mu)-F(u)}} \tag{17}$$

We also obtain the following equation by multiplying (4) by N' and integrating it with respect to *x*:

$$\frac{1}{2}\left(\frac{\partial N}{\partial x}\right)^2+F(N)=c \tag{18}$$

Where,

$$F(N)=\int_0^N u\left(1-\frac{\alpha+\gamma u}{K\left(1+\gamma\left(u+S\right)\right)}-\frac{\eta\left(1+\theta S\right)}{1+\gamma\left(u+S\right)}\right)du$$

and c is a constant. $F(N)$ is the required potential function. Thus, steady pest population distributions in space, with corresponding maximum female density M, can be related to the size of the patch through the formula:

$$L(M)=\sqrt{2D}\int_0^{\mu}\frac{dM}{\sqrt{F(\mu)-F(M)}} \tag{19}$$

In the next section, we will be discussing the optimal production policy.

OPTIMAL PRODUCTION POLICY

We begin our analysis by stating the assumption that S(t) is the sterile male population at time t. The evolution of sterile male population is described by the following differential equation as:

$$\frac{dS}{dt}=u(t)-\mu_s S(t) \tag{20}$$

where u(t) is the rate of population of sterile male population. The equation (20) shows that sterile male population will increase by u(t) and decrease at a certain rate μ_S.

For determining an optimal production policy for sterile male population and losses due to pest damage. We consider a cost function depending continuously on time given as follows:

$$C(t)=\int_0^T\left(pu(t)+qN(t)\right)dt \tag{21}$$

where p the cost per sterile male and q is feeding cost per unit pest. A standard optimal control problem can be mathematically formulated as follows:

$$\max_{u(t)} J = -\int_0^T (pu(t) + qN(t))dt \tag{22}$$

subject to satisfying

$$\dot{N}(t) = \varphi N\left(1 - \frac{\eta K\left(1 + \theta S + K\alpha + N\gamma\right)}{K\left(1 + \gamma\left(N + S\right)\right)}\right) \tag{23}$$

$$N(0) = N_0 \tag{24}$$

$$\frac{dS}{dt} = u(t) - \mu_s S(t), S(0) = S_0, S(T) \geq 0 \tag{25}$$

where J is the objective functional (cost function). Now we have an optimal control problem with control variable $u(t)$ and state variable $N(t)$ and $S(t)$. Using Maximum Principle (Sethi & Thompson, 2000), the Hamiltonian can be defined as,

$$H = -(pu(t) + qN(t) + \lambda_1(t)\dot{N}(t) + \lambda_2(t)\left(u(t) - \mu_s S(t)\right)$$

The Hamiltonian represent the total cost of various policy decision with both the immediate and future taken into account where $\lambda_1(t)$ and $\lambda_2(t)$ are adjoint variables and describe the similar behaviour in optimal control theory as dual variables have in non-linear programming.

From the necessary optimality conditions of maximum principle (Seierstad & Sydsaeter, 1987), we have,

$$\frac{\partial H}{\partial u(t)} = 0 \tag{26}$$

$$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial N}, \lambda_1(T) = 0 \tag{27}$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial S}, \lambda_2(T)S(T) = 0 \tag{28}$$

The Hamiltonian is concave in $u(t)$. According to Mangasanian Sufficiency Theorem, there exists unique value of control $u^*(t)$. Since Hamiltonian is linear in $u(t)$, optimal production rate for sterile male population as obtained by Maximum principle is given by,

$$u^*(t) = \begin{cases} \bar{U} & if \ D(t) \le 0 \\ \underline{U} & if \ D(t) > 0 \end{cases} \tag{29}$$

where $D(t)=\lambda_2(t) - p$ is production rate switching function and called "Bang-Bang" control. However, interior control is possible on an arc along $u(t)$ and known as "Singular Arc" (Seierstad & Sydsaeter, 1987; Bryson & Ho, 1997). Here, $\bar{U}$ and $\underline{U}$ are positive constants which are minimum and maximum acceptable production rate for sterile male population. Using Equation (27) and (28), the adjoint variables can be calculated as respectively, we get,

$$\lambda_1(t) = \int_t^T e^{-\mu_s(t-\tau)} \lambda_1 \left(\frac{\partial \dot{N}}{\partial S} \right) d\tau \tag{30}$$

$$\lambda_2(t) = \frac{\left(q - e^{\left(\frac{\partial \dot{N}}{\partial S}\right)\left((T-t) - \frac{\log q}{\left(\frac{\partial \dot{N}}{\partial S}\right)T}\right)} \right)}{\frac{\partial \dot{N}}{\partial S}} \tag{31}$$

The value of $\lambda_1(t)$ and $\lambda_2(t)$ define the marginal valuation of state variables $N(t)$ and $S(t)$ at time t respectively. Here, λ_1 and λ_2 stand for per unit change in future total cost of having one more unit of variables $N(t)$ and $S(t)$. Due to complicated

analytical expression and to illustrate the applicability of the above problem through a numerical illustration, the continuous time optimal problem (22-25) is transformed into equivalent discrete problem (Rosen, 1968). The equivalent discrete optimal control can be written as follows,

$$J = Min\sum_{k=1}^{T}\left[\, pu\left(k\right) + qN\left(k\right)\right] \tag{32}$$

such that

$$N\left(K+1\right) = N\left(k\right) + \varphi N\left(k\right)\left(1 - \frac{\eta K\left(1+\theta S\left(k\right) + K\alpha + N\left(k\right)\gamma\right)}{K\left(1+\gamma\left(N\left(k\right)+S\left(K\right)\right)\right)}\right), N\left(0\right) = N_0 \tag{33}$$

$$S\left(k+1\right) = S\left(k\right) - \mu_s S\left(k\right) + u\left(k\right) \tag{34}$$

$$S\left(0\right) = S_0,\ S\left(T\right) \geq 0 \tag{35}$$

The discretized version of the model is Non-Linear Programming. We discuss the solution of above discrete version of optimal control theory problem using Lingo 11 (Thirez, 2000) in next section.

NUMERICAL SECTION

We have considered the parametric values as indicated in Table 2.

The graph denoting the plotting of $F(N)$, the potential function (4) is shown in Fig 2. Fig 2 shows that it reaches a maximum value before dropping again, therefore, it is bounded above and thus have a maximum value. Equation (7) is used to understand the approximation of the effect of SIT during the invasion in initial stages. The non trivial solution is steady and it disappears as there is a decrease in the value of μ. With the hypotheses we have currently considered, an invasion over a critical patch length is possible and thus, we have our desired result. Consequently, there is no requirement for the inclusion of density dependence death rate as the spatial spread invasion produces a declination in the pest population.

Table 2. Description and values of parameters (In Units)

Description	Values of Parameter
μ: Death rate	$10day^{-1}$
β: Growth rate	$0.3day^{-1}$
λ: Additional death rate	0
1/σ: Average time females spend in reproductive state	½ day
ξ: Avg per capita mating encounter	0.001
K: Carrying capacity	100
S: No. of sterile males	300

Finally, the optimal control problem of model proposed is solved by Maximum Principle. The model is nonlinear in nature and leads to complex analytical expression. The proposed problem is continuous but in practical the data available is discrete. Because of its nonlinearity it becomes difficult to solve it by traditional methods. Thus we discretized the proposed optimal control problem and discrete problem is solved by using Lingo 11 (Thirez, 2000). For discrete problem, the values of the parameters while solving the problem is given in Table 2. In addition, we have taken p=30, q=20 and T=20. The time horizon has been divided into 20 equal time periods. The optimal production of sterile male population and female population obtained is given in Table 3 [Figure 3]. The optimal value of the total cost is 18737 unit in given time horizon.

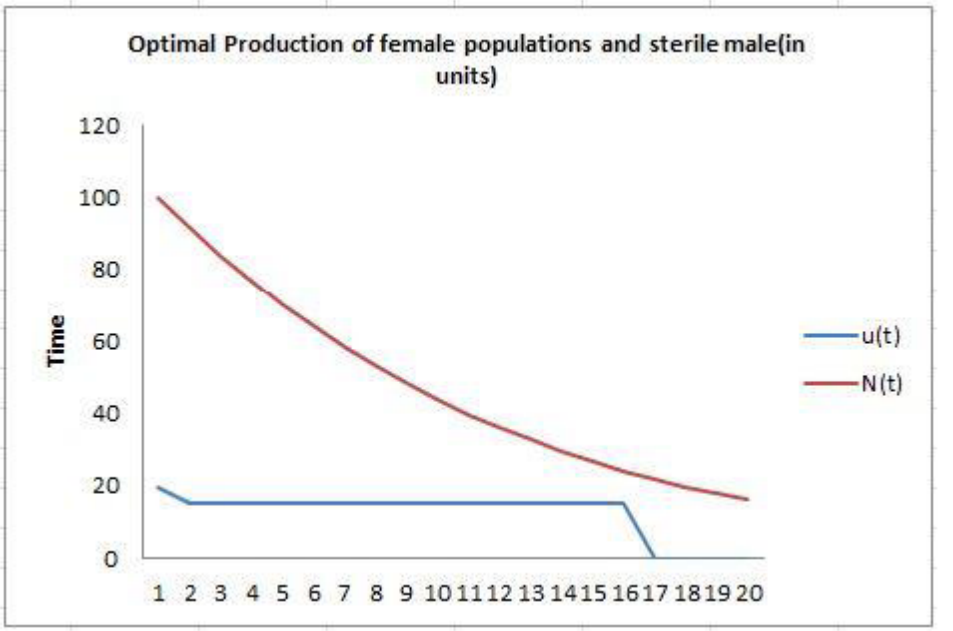
Table 3. Optimal production of female populations and sterile male (In Units)

	T1	T2	T3	T4	T5	T6	T7	T8	T9	T10	T11	T12	T13	T14	T15	T16	T17
N(t)	100	92	84	77	70	64	58	53	48	44	40	36	33	29	27	24	22
u(t)	10	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15	15

DISCUSSION

In this paper, we propose a logistic growth rate model undergoing sterile male release, commonly known as SIT, under specific condition and understand the behaviour of the pest population. This model is essentially about the female species which are sexually mature and are grouped into: female population looking for a mate

Figure 3. Optimal production of female and sterile male population



and the female species who are at reproductive stage. Further, the second group is classified into: female population impregnated by the wild males and the ones by sterile male species. Evidently, the latter will not lead to future reproduction. We incorporate a spatial spread invasion, where a limited number of sterile male species are included over a certain patch size surrounded by a toxic exterior, we observe a sudden declination in the population of pests. Thus, we obtained that the logistic growth rate of the female population N_1 with SIT technique will help in eradication of the pest population. Consequently, there is no requirement for the inclusion of density dependence death rate as the spatial spread invasion produces a declination in the pest population. Further, from the optimal policies also we have obtained that the rate of production of the sterile male will be higher and gradually it gets less and still it helps in eradicating the pest population. In future, we would be discussing the comparison between integrated pest management technique and SIT technique and also try to study the dynamical analysis of the same model by involving both biological and chemical control impulsively.

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Chapter 10
Transmission of Water and Food Waste in Aquaponic Systems

Moksha H. Satia
Department of Mathematics, Gujarat University, Ahmedabad, India

ABSTRACT

In order to conserve natural resources, the quest for recycling water and food waste culture is ongoing. One of the possible and good ways to reuse these wastes is hydroponic culture. It is an advanced technology that cultivates plants without soil. Instead of using root system, it needs nutrient-rich water. Most of the nutrients used in hydroponic culture come from aqua culture, the branch for propagation, emergence, and maintenance of aquatic (water) organisms. Humans convolve aqua culture with hydroponic culture that has come up as an aquaponic system. It has been universally adopted for indoor food production. The solution arising out of this system has eliminated the lack of vegetable and fish. The continuous nature of these cultures gives rise to the system of non-linear ordinary differential equations. This system is investigated through logistic growth rate. Logistic growth rate offers an oscillating threshold. The simulative results analyse the periodicity of the system solutions, which will help the ecosystem survive.

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INTRODUCTION

People need water and food the most in order to live. Most of them are not item that people use and they last for a long time or an eternity. The item that is no longer useful is called waste. But in this innovative human world, we may convert waste into its by-products, composite products or resources. This is an experimental way to increase the value of waste from zero. Moreover, humans are constantly searching the various ways to recycle waste in reusable products. In this study, waste water and food have been targeted for vegetable production and fish preservation.

Any water that has been affected by human use can be classified as waste water. Waste water is water sourced from the combination of domestic, industrial, commercial or agricultural activities. Therefore, waste water can be bifurcated into domestic water, industrial water, commercial water and agricultural water. Among these waste waters, industrial waste water is the most difficult type to recycle. Vacuum process helps to effectively treat industrial waste water which sometime lead to a system with zero discharge. This technology has many profits like it is hygienic, secure, versatile and has low management cost. Moreover, it is one of the most adequate and efficient techniques for treating aqueous effluents. Treated water uses in branch of algae culture. Algae culture involves the plants grow in water resource.

Waste food reflects the food that has been used by living creature. The word "waste food" is either wasted, lost or uneaten food whose disposal is the global problem. It can be biodegraded using fly/worm in order to make feed for fishes. Fish is the creature lives in aqua culture, a branch of propagating, rising and keeping of aquatic organisms.

Algae culture and aqua culture are closely related. They contain definite level of uneaten nutrients. These nutrients are excreted by fish and algae which supplies to hydroponic culture. Hydroponics is a method of growing plants in water based, nutrient rich solution not on the growing using soil on the land. In this method, the root system is supported using an inert medium such as perlite, rockwool, clay pellets, peat moss, or vermiculite. The key purpose behind hydroponics is to allow the plants roots to come in direct contact with the nutrients, while also having access to oxygen, which is essential for proper growth. When aqua culture convolves with hydroponic culture then one branch is emerged that is called aquaponics. In this system, the toxic water accumulates from aqua culture that is fed to hydroponic system and the by-products are broken down by nitrifying bacteria initially into nitrites and subsequently into nitrates that are utilized by the plants as nutrients. Then, the water is recirculated back to the aquaculture system. In this way, the aquaponic system results in the production of vegetables and the preservation of fish which is very fruitful for the human society.

Aquaponic system is emerging concept for research world. Diver and Rinehart (2000) have studied the branch of Aquaponics in which hydroponics integrated with aquaculture. Extended study was done through Chen *et al.* (2018) using breakthrough technologies for the biorefining of organic solid and liquid wastes. Mathematical Modeling has been done for waste water and food from various researchers. Xiang *et al.* (2013) prepared dynamic modeling and simulation of water environment management with a focus on water recycling. The concept of mathematical modeling for water usage and treatment network design is used by Huang *et al.* (1999). Similarly, for residential food generation, Benitez *et al.* (2008) have developed a mathematical model.

The objective of this chapter is to revive agriculture and aquatic balance. In section 2, a mathematical model for aquaponic system using sinusoidal periodic function is developed along with its equilibrium points and threshold. The global stability analysis is derived in section 3. Section 4 represents the numerical simulation to validate our results with parametric data.

MATHEMATICAL MODEL

We live in the society where aquaponic culture is essential for production of vegetable and conservation of fish. To achieve this goal, we have constructed system of non-linear differential equations having ten compartments. Here, volume of waste water is denoted by W_W, density of waste food is noted as W_F, depletion of waste water through vacuum process is represented as V_P, depletion of waste food through fly/worms is renowned as F_W, algae culture is symbolised through A_L, hydroponic culture is signified by H_Y, aqua culture is suggested by A_Q, density of leftover vegetable scrap is marked as V_S, production of number of vegetables is indicated using V and conservation of fish is expressed by F. The usage of water and food is not constant every day, even more the waste disposal of these wastes is also infrequent. Therefore, this non-autonomous aquaponic system is studied by replacing the constant transmission rate with the periodic transmission rate. This transmission is considered as sinusoidal (Sun *et al.* 2013) such that $\beta(t) = b\sin\left(\frac{\pi t}{n}\right)$, where n is the period. In this chapter, we have considered n is equal to one week and hence n=7 which implies $\beta(t) = b\sin\left(\frac{\pi t}{7}\right)$. This aquaponic system has two transmission rates i.e. $\beta_1(t) = b_1\sin\left(\frac{\pi t}{n}\right)$ and $\beta_2(t) = b_2\sin\left(\frac{\pi t}{n}\right)$. To design this model, some assumptions are taken.

Assumptions:

(1) Depletion of waste water and food can happen when they are sent for processing.
(2) In hydroponic culture, vegetable scrap can be produced.
(3) There is a cycle between algae culture and aqua culture.
(4) Vacuum process free equilibrium point exists for this aquaponic system.
(5) For the analysis purpose, the exist rate of each compartment is taken as constant. Here, it is denoted by μ.

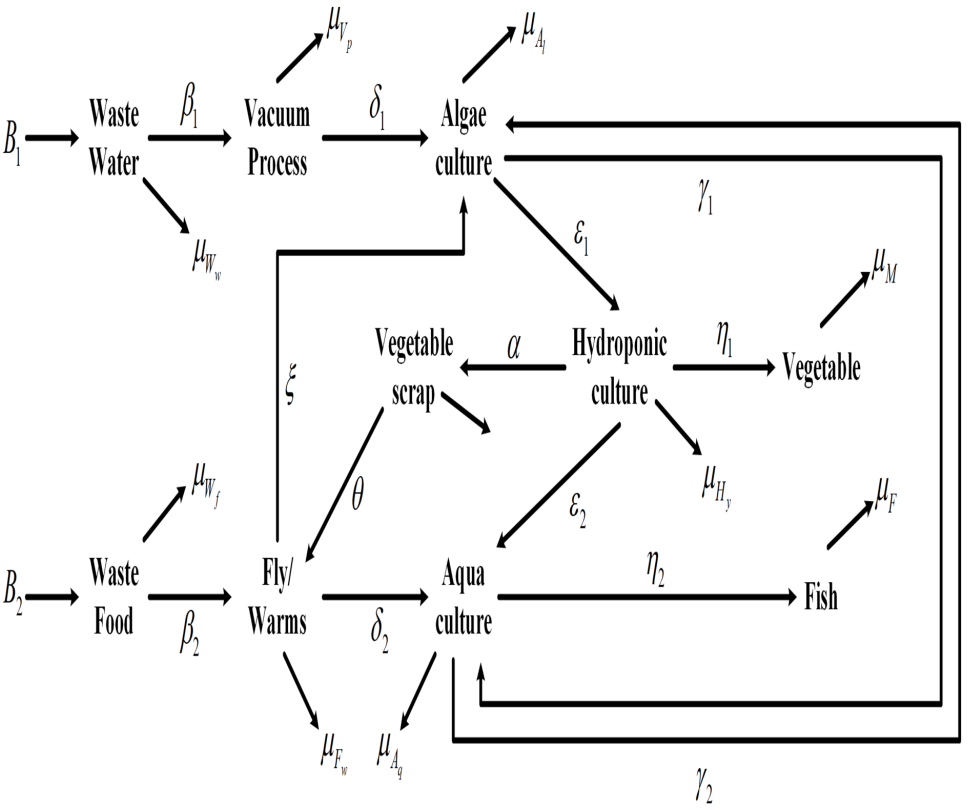
Figure 1 is the model diagram of our aquaponic system which shows the transmission of waste water and food which end up to produce vegetables and conserve fishes with transmission parameter given in the table 1. With the help of figure 1 and table 1, the aquaponic system is formulated as given below:

$$\frac{dW_W}{dt} = B_1 - \beta_1 W_W V_P - \mu W_W$$

Table 1. Notation and its description

Notation	Description
B_1	The growth rate of waste water
B_2	The growth rate of waste food
β_1	The rate at which waste water sent for vacuum process
β_2	The rate at which waste food sent to fly/worm
δ_1	Transmission rate of processed water in algae culture
δ_2	Transmission rate of processed food in aqua culture
ξ	The rate of fly/worm supports in algae culture
ε_1	The preservation rate of water used in hydroponic culture through algae culture
ε_2	The transfer rate of water used in hydroponic culture into aqua culture
γ_1	The rate at which algae culture helps to maintain the level of aqua culture
γ_2	The rate at which aqua culture comes in the contact of algae culture
α	The rate of vegetable scrap produced during hydroponics
θ	The rate at which vegetable scrap are sent to fly/worm for process
η_1	The production rate of vegetables
η_2	The conservation rate of fishes
μ	The exit rate

Figure 1. Transmission diagram of aquaponic system



$$\frac{dW_F}{dt} = B_2 - \beta_2 W_F F_W - \mu W_F$$

$$\frac{dV_P}{dt} = \beta_1 W_W V_P - \delta_1 V_P - \mu V_P$$

$$\frac{dF_W}{dt} = \beta_2 W_F F_W - \xi F_W - \delta_2 F_W + \theta V_S - \mu F_W$$

$$\frac{dA_L}{dt} = \delta_1 V_P + \xi F_W - \varepsilon_1 A_L - \gamma_1 A_L + \gamma_2 A_Q - \mu A_L \quad (1)$$

$$\frac{dH_Y}{dt} = \varepsilon_1 A_L - \varepsilon_2 H_Y - \alpha H_Y - \eta_1 H_Y - \mu H_Y$$

$$\frac{dA_Q}{dt} = \delta_2 F_W + \gamma_1 A_L + \varepsilon_2 H_Y - \gamma_2 A_Q - \eta_2 A_Q - \mu A_Q$$

$$\frac{dV_S}{dt} = \alpha H_Y - \theta V_S - \mu V_S$$

$$\frac{dV}{dt} = \eta_1 H_Y - \mu V$$

$$\frac{dF}{dt} = \eta_2 A_Q - \mu F$$

This represents non-negative R_+^{10} manifold with initial conditions:

$$W_W(0) > 0, W_F(0) > 0, V_P(0) > 0, F_W(0) > 0, A_L(0) > 0,$$
$$H_Y(0) > 0, A_Q(0) > 0, V_S(0) > 0, V(0) > 0, F(0) > 0$$

Thus, we have following lemma

Lemma: For any positive initial condition

$$\left(W_W(0), W_F(0), V_P(0), F_W(0), A_L(0), H_Y(0), A_Q(0), V_S(0), V(0), F(0)\right),$$

the system has a unique positive solution

$$\left(W_W, W_F, V_P, F_W, A_L, H_Y, A_Q, V_S, V, F\right)$$

for all $t \geq 0$. Moreover, the compact set

$$\Lambda = \left\{ \left(W_W, W_F, V_P, F_W, A_L, H_Y, A_Q, V_S, V, F \right) \right.$$
$$\left. \in \mathbb{R}_+^{10} / W_W + W_F + V_P + F_W + A_L + H_Y + A_Q + V_S + V + F \le \frac{B_1 + B_2}{\mu} \right\}$$

is a positively invariant set, which attracts all positive orbits in $\mathbb{R}_+^{10}$ and all the solutions are bounded.

Proof: Define $N(t) = W_W + W_F + V_P + F_W + A_L + H_Y + A_Q + V_S + V + F$

$$\frac{dN(t)}{dt} = \frac{dW_W}{dt} + \frac{dW_F}{dt} + \frac{dV_P}{dt} + \frac{dF_W}{dt} + \frac{dA_L}{dt} + \frac{dH_Y}{dt} + \frac{dA_Q}{dt} + \frac{dV_S}{dt} + \frac{dV}{dt} + \frac{dF}{dt}$$
$$= B_1 + B_2 - \mu \left(W_W + W_F + V_P + F_W + A_L + H_Y + A_Q + V_S + V + F \right)$$

Then

$$\frac{dN(t)}{dt} = B_1 + B_2 - \mu N(t) \tag{2}$$

Thus, if $N(t) > \frac{B_1 + B_2}{\mu}$, then $\dot{N}(t) < 0$

Now, let us consider the ordinary differential equation (2)

$$\frac{dN(t)}{dt} = B_1 + B_2 - \mu N(t)$$

with the general equation

$$N(t) = \frac{B_1 + B_2}{\mu} + \left(N(0) - \frac{B_1 + B_2}{\mu} \right) e^{-\mu t}$$

where $N(0)$ is the initial condition of $N(t)$ as $t \to \infty$.

By applying the standard comparison theorem, we have,

$N(t) \leq \frac{B_1 + B_2}{\mu}$, for all $t \geq 0$.

Thus, the compact set Λ is a positively invariant set, attracts all positive orbits in $\mathbb{R}_+^{10}$ and then the solutions are bounded.

Now, consider

$$\delta_1 + \mu = x_1, \xi + \delta_2 + \mu = x_2 \varepsilon_1 + \gamma_1 + \mu = x_3, \varepsilon_2 + \alpha + \eta_1 + \mu = x_4, \gamma_2 + \eta_2 + \mu = x_5, \theta + \mu = x_6$$

then we get a following new system

$$\frac{dW_W}{dt} = B_1 - \beta_1 W_W V_P - \mu W_W$$

$$\frac{dW_F}{dt} = B_2 - \beta_2 W_F F_W - \mu W_F$$

$$\frac{dV_P}{dt} = \beta_1 W_W V_P - x_1 V_P$$

$$\frac{dF_w}{dt} = \beta_2 W_f F_w + \theta V_s - x_2 F_w$$

$$\frac{dA_L}{dt} = \delta_1 V_P + \xi F_W + \gamma_2 A_Q - x_3 A_L \tag{3}$$

$$\frac{dH_Y}{dt} = \varepsilon_1 A_L - x_4 H_Y$$

$$\frac{dA_Q}{dt} = \delta_2 F_W + \gamma_1 A_L + \varepsilon_2 H_Y - x_5 A_Q$$

$$\frac{dV_S}{dt} = \alpha H_Y - x_6 V_S$$

$$\frac{dV}{dt} = \eta_1 H_Y - \mu V$$

$$\frac{dF}{dt} = \eta_2 A_Q - \mu F$$

The dynamical behaviour of system (1) is equivalent to the system (3). Hence, system (3) will study our lemma.

Existence of Equilibrium Points

Equilibrium points are the points which satisfies the system. It means they are the solution of the system. The existence of equilibrium points is important and therefore it is worked out here for aquaponic system. Equating all the equation of the system (3) to the zero, we get three equilibrium points.

i. i. Trivial equilibrium point

$$E_0\left(\frac{B_1}{\mu}, \frac{B_2}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right)$$

ii. ii. Vacuum process free equilibrium point

$E^1\left(W_W^1, W_F^1, 0, F_W^1, A_L^1, H_Y^1, A_Q^1, V_S^1, V^1, S^1\right)$ where

$$W_W^1 = \frac{B_1}{\mu}, W_F^1 = \frac{\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2}{\beta_6 x_6 m_2}, F_W^1 = -\frac{\left(x_6 m_3 m_2 + \alpha\theta\varepsilon_1\mu m_1\right)}{\beta_1\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right)},$$

$$A_L^1 = \frac{\left(x_6 m_3 m_2 + \alpha\theta\varepsilon_1\mu m_1\right) x_4 m_1}{m_2}, H_Y^1 = \frac{\varepsilon_1\left(x_6 m_3 m_2 \alpha\theta\varepsilon_1\mu m_1\right) m_1}{\beta_2\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right) m_2},$$

$$A_Q^1 = \frac{\left(x_6 m_3 m_2 \alpha\theta\varepsilon_1\mu m_1\right) m_4}{\beta_2\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right) m_2}, V_S^1 = \frac{\alpha\varepsilon_1\left(x_6 m_3 m_2 + \alpha\theta\varepsilon_1\mu m_1\right) m_1}{\beta_2\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right) m_2 x_6},$$

$$V^1 = \frac{\eta_1\varepsilon_1\left(x_6 m_3 m_2 + \alpha\theta\varepsilon_1\mu m_1\right) m_1}{\beta_2\mu\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right) m_2}, F^1 = \frac{\eta_2\left(x_6 m_3 m_2 + \alpha\theta\varepsilon_1\mu m_1\right) m_4}{\beta_2\mu\left(\alpha\theta\varepsilon_1 m_1 + x_2 x_6 m_2\right) m_2}$$

Here,

$$m_1 = \delta_2\gamma_2 + x_5\xi, m_2 = \gamma_1\gamma_2 x_4 + \gamma_2\varepsilon_1\varepsilon_2 - x_3x_4x_5, m_3 = -B_2\beta_2 + \mu x_2, m_4 = \delta_2 x_3 x_4 + \gamma_1 x_4\xi + \xi\varepsilon_1\varepsilon_2$$

The equilibrium point E^1 exists only when m_2 and m_3 are positive.

iii. Endemic equilibrium point

$$E^*\left(W_W^*, W_F^*, V_P^*, F_W^*, A_L^*, H_Y^*, A_Q^*, V_S^*, V^*, F^*\right)$$

where

$$W_W^* = \frac{x_1}{\beta_1}, W_F^* = \frac{\alpha\beta_1 r\theta x_1\varepsilon_1 m_1 + \beta_1 r x_1 x_2 x_6 m_2 - \alpha\delta_1\theta x_5\varepsilon_1 m_5}{r\beta_1\beta_2 x_1 x_6 m_2}, V_P^* = -\frac{m_5}{\beta_1 x_1}, F_W^* = r,$$

$$A_L^* = \frac{x_4\left(-\beta_1 r x_1 m_1 + \delta_1 x_5 m_5\right)}{\beta_1 x_1 m_2}, H_Y^* = \frac{\varepsilon_1\left(-\beta_1 r x_1 m_1 + \delta_1 x_5 m_5\right)}{\beta_1 x_1 m_2}, A_Q^* = \frac{-\beta_1 r x_1 m_4 + \delta_1\left(\gamma_1 x_4 + \varepsilon_1\varepsilon_2\right)m_5}{\beta_1 x_1 m_2},$$

$$V_S^* = \frac{\alpha\varepsilon_1\left(-\beta_1 r x_1 m_1 + \delta_1 x_5 m_5\right)}{\beta_1 x_1 m_2 x_6}, V^* = \frac{\eta_1\varepsilon_1\left(-\beta_1 r x_1 m_1 + \delta_1 x_5 m_5\right)}{\beta_1 x_1 m_2\mu}, F^* = \frac{\eta_2\left(-\beta_1 r x_1 m_4 + \delta_1\left(\gamma_1 x_4 + \varepsilon_1\varepsilon_2\right)m_5\right)}{\beta_1 x_1 m_2}$$

The endemic equilibrium point exists only when r is negative and m_5 is positive. The existence of equilibrium points suggest that they exist when

a) $\gamma_1\gamma_2 x_4 + \gamma_2\varepsilon_1\varepsilon_2 - x_3x_4x_5 > 0$

b) $r<0$

c) $\min\left\{\frac{B_1\beta_1}{x_1}, \frac{B_2\beta_2}{x_2}\right\} < \mu$

Computation of Threshold Quantity

In this section, the threshold quantity for the aquaponic system is computed. It well-known as basic reproduction number denoted by R_0. The expression of the threshold quantity is calculated using next generation matrix method (Diekmann *et al*., 2009). Threshold quantity helps to recognize the stability nature of the system which is dependent upon the numerical value of R_0. If the value is less than one than the system has not reach the epidemic stage. The threshold quantity is the ratio of newly infected waste affected by secondary infectious waste for the production

of vegetables and conservation of fishes. Using next generation matrix method, Jacobian matrices *f* and *v* are found as

$$f=\begin{bmatrix} \beta_1 W_W & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_1 V_P & 0 \\ 0 & \beta_2 W_F & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_2 F_W \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

$$v=\begin{bmatrix} x_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & x_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\delta_1 & -\xi & x_3 & 0 & -\gamma_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\varepsilon_1 & x_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\delta_2 & -\gamma_1 & -\varepsilon_2 & x_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\alpha & 0 & x_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\eta_1 & 0 & 0 & \mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta_2 & 0 & 0 & \mu & 0 & 0 \\ \beta_1 W_W & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_1 V_P+\mu & 0 \\ 0 & \beta_2 W_F & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_2 F_W+\mu \end{bmatrix}$$

Here, *v* is non-singular matrix. Hence, the largest eigenvalue of next generation matrix fv^{-1} has the following expression which our threshold quantity:

$$R_0=\sqrt{\frac{B_1\beta_1 B_2\beta_2 x_6 m_2}{\mu^2 x_1\left(\alpha\theta\varepsilon_1 m_1+x_2 x_6 m_2\right)}} \tag{4}$$

GLOBAL STABILITY ANALYSIS

In this section, global stability of the aquaponic system is discussed about each three-equilibrium point. For the trivial equilibrium point, the theory of Lyapunov function is used where for other two equilibrium points, theory of graph is incorporated.

Theorem 1: When $B_1+B_2<2\mu$ then the trivial equilibrium point E_0 is globally asymptotically stable.

Proof: Consider a positive Lyapunov function L_0 such as $L_0 = k_1 W_W{}^2 + k_2 W_F{}^2$ where k_1 and k_2 are constants.

Now, differentiate L_0 w.r.t t, we get

$$L_0{}' = \begin{bmatrix} 2k_1 W_W & 2k_2 W_F \end{bmatrix} \begin{bmatrix} B_1 - \beta_1 W_W V_P - \mu W_W \\ B_2 - \beta_2 W_F F_W - \mu W_F \end{bmatrix}$$

$$= 2k_1 B_1 W_W - 2k_1 \beta_1 W_W{}^2 V_P - 2k_1 \mu W_W{}^2 + 2k_2 B_2 W_F - 2k_2 \beta_2 W_F^2 F_W - 2k_2 \mu W_F^2$$

$$\le -2k_1 \mu W_W{}^2 - 2k_2 \mu W_F{}^2 + \left(2k_1 B_1 + 2k_2 B_2\right) W_W W_F - 2k_1 \beta_1 W_W{}^2 V_P - 2k_2 \beta_2 W_F^2 F_W$$

Let us choose $k_1 = k_2 = \dfrac{1}{\mu}$ then

$$L_0{}' = -2W_W{}^2 - 2W_F{}^2 + \left(\frac{2B_1 + 2B_2}{\mu}\right) W_W W_F - 2\frac{\beta_1}{\mu} W_W{}^2 V_P - 2\frac{\beta_2}{\mu} W_F{}^2 F_W$$

$$= -2\left[\left(W_W - \frac{1}{2}\left(\frac{B_1 + B_2}{\mu}\right) W_F\right)^2 - \left(\frac{B_1 + B_2}{\mu}\right)^2 W_F{}^2 + W_F{}^2 + \frac{1}{\mu}\left(\beta_1 W_W{}^2 V_P + \beta_2 W_F{}^2 F_W\right)\right]$$

$$= -2\left[\left(W_W - \frac{1}{2}\left(\frac{B_1 + B_2}{\mu}\right) W_F\right)^2 + \left(1 - \left(\frac{B_1 + B_2}{\mu}\right)^2\right) W_F{}^2 + \frac{1}{\mu}\left(\beta_1 W_W{}^2 V_P + \beta_2 W_F{}^2 F_W\right)\right]$$

Since $V_P = F_W$ at E_0, we have

$$L_0{}' = -2\left[\left(W_W - \frac{1}{2}\left(\frac{B_1 + B_2}{\mu}\right) W_F\right)^2 + \left(1 - \left(\frac{B_1 + B_2}{\mu}\right)^2\right) W_F{}^2\right] \le 0 \text{ iff } B_1+B_2<2\mu$$

Hence, LaSalle's Invariance Principle (La Salle, 1976) suggests that the trivial equilibrium point E_0 is globally asymptotically stable.

Next, we study the global stability of vacuum process free E^1 and endemic equilibrium point E^* using graph theory (Shuai and van den Driessche 2013, Din *et al.* 2016). Some graph theoretical results (Harary 1969, West and Douglas 2001) are used to establish the stability.

- Any graph will consist of the set of vertices and the set of edges.
- (*i,j*) is called *an edge* from initial vertex *i* to terminal vertex *j*.
- *A directed graph G* is the set of vertices and the set of edges where all the edges are directed from one vertex to another. (https://mathinsight.org/definition/directed_graph)
- *The out-degree of a vertex i* is the number of edges whose initial vertex is *i* denoted as $d^+(i)$.
- *The in-degree of a vertex i* is the number of edges whose terminal vertex is *i* denoted as $d^-(i)$.
- A directed graph *G* is called *a weighted directed graph* if each edge is assigned a positive weight.
- *The weight w*(*H*) of sub-directed graph *H* is the product of weights on all its edges.
- *A path* in a graph is a finite or infinite sequence of edged which connect the sequence of vertices where all are distinct from others. (https://en.wikipedia.org/wiki/Path_(graph_theory))
- *A directed path* in a directed graph is a sequence of edges which connect a sequence of edges which connect a sequence of vertices where all the edges should be directed in the same direction.
- *A cycle graph* is a graph where some number of vertices connected in a closed chain. (https://en.wikipedia.org/wiki/Cycle_graph)
- *A directed cycle graph* is a directed version of a cycle graph with all the edges being oriented in the same direction.
- *A loop* (or *buckle*) is an edge that connects a vertex *i* to itself. (https://en.wikipedia.org/wiki/Loop_(graph_theory))
- *A tree* is any acyclic connected graph. (https://en.wikipedia.org/wiki/Tree_(graph_theory))
- If tree is directed then it is called *directed tree*.
- *A spanning tree* is a subgraph of a graph *G* which includes all the vertices of *G* with minimum number of edges. (https://en.wikipedia.org/wiki/Spanning_tree)
- If *G* is a weighted directed graph with *n* vertices then the weight matrix has order $n \times n$ denoted as $A=[a_{ij}]$ with entries $a_{ij}>0$ which is equal to the weight

of edge if it exists otherwise it is 0. This kind of weighted directed graph is noted by (G,A).

- G is called *strongly connected directed graph* if for any pair of discrete vertices there exists a directed path.
- (G,A) is called *a strongly connected weighted directed graph* if and only if the weighted matrix A is irreducible.

The Laplacian matrix $L=[l_{ij}]$ of (G,A) is defined as $l_{ij}=\begin{cases}-a_{ij} & ; i\neq j\\ \sum_{k\neq i} a_{ik} & ; i=j\end{cases}$.

Proposition (Kirchhoff's matrix tree theorem): Assume $n\geq 2$ and let c_i be the cofactor of l_{ii} in L. Then $c_i=\sum_{\tau\in T_i} w(\tau), i=1,2,...,n$ where T_i is the set of all spanning trees τ of weighted directed graph (G,A) which makes tree at vertex i and $w(\tau)$ is the weight of τ. If the weighted graph (G,A) is strongly connected then $c_i>0$ for $1\leq i\leq n$.

Theorem 2: Let c_i be as given in the Kirchhoff's matrix tree theorem. If $a_{ij}>0$ and $d^+(j)=1$ for some i,j then $c_i a_{ij}=\sum_{k=1}^{n} c_j a_{jk}$.

Theorem 3: Let c_i be as given in the Kirchhoff's matrix tree theorem. If $a_{ij}>0$ and $d^-(i)=1$ for some i,j then $c_i a_{ij}=\sum_{k=1}^{n} c_k a_{ki}$.

Theorem 4: Suppose that the following assumptions are satisfied:

(1) There exists function $V_i: U\to\mathbb{R}, G_{ij}: U\to\mathbb{R}$ and constants $a_{ij}\geq 0$ such that for every $1\leq i\leq n$, $V_i'\leq\sum_{j=1}^{n} G_{ij}(z)$ for $z\in U$.

(2) For $A=[a_{ij}]$, each directed cycle C of (G,A) has $\sum_{(s,r)\in\varepsilon(C)} G_{rs}(z)\leq 0$ for $z\in U$, where $\varepsilon(C)$ denotes the arc set of the directed cycle C.

Then, the function $V(z)=\sum_{i=1}^{n} c_i V_i(z)$, with constant $c_i\geq 0$ as given in the proposition of Kirchhoff's matrix tree theorem, satisfies $V'\leq 0$ then V is a Lyapunov function for the system.

Theorem 5: The vacuum process free equilibrium point E^1 is globally asymptotically stable in int(Λ).

Proof: Let us construct Lyapunov function $V(t)$ so that

$V = V_1 + V_2 + V_3 + V_4 + V_5 + V_6 + V_7 + V_8 + V_9$ where

$$V_1 = \frac{1}{2}\left(W_W - W_W^1 + W_F - W_F^1\right)^2, V_2 = \frac{1}{2}\left(W_F - W_F^1\right)^2, V_3 = \frac{1}{2}\left(F_W - F_W^1\right)^2, V_4 = \frac{1}{2}\left(A_L - A_L^1\right)^2,$$
$$V_5 = \frac{1}{2}\left(H_Y - H_Y^1\right)^2, V_6 = \frac{1}{2}\left(A_Q - A_Q^1\right)^2, V_7 = \frac{1}{2}\left(V_S - V_S^1\right)^2, V_8 = \frac{1}{2}\left(V - V^1\right)^2, V_9 = \frac{1}{2}\left(F - F^1\right)^2$$

Differentiating V_1 with respect to time t, we get

$$\begin{aligned} V_1' &= \left(\left(W_W - W_W^1\right) + \left(W_F - W_F^1\right)\right)\left(B_1 - \mu W_W + B_2 - \beta_2 W_F F_W - \mu W_F\right) \\ &\le 2\mu\left(W_W - W_W^1\right)\left(W_F - W_F^1\right) \\ &= a_2^1 G(1,2) \end{aligned}$$

Similarly, differentiating others with respect to time t, we have

$$\begin{aligned} V_2' &= \left(W_F - W_F^1\right)\left(\beta_2 W_F^1 F_W^1 - \beta_2 W_F F_W + \mu W_F^1 - \mu W_F\right) \\ &\le \beta_2 W_F^1 F_W^1 \left(W_F - W_F^1\right)\left(1 - \frac{W_F F_W}{W_F^1 F_W^1}\right) \\ &= a_3^2 G(2,3) \end{aligned}$$

$$\begin{aligned} V_3' &= \left(F_W - F_W^1\right)\left(\beta_2 W_F F_W + \theta V_S - x_2 F_W\right) \\ &\le \beta_2 W_F^1 F_W^1 \left(F_W - F_W^1\right)\left(1 - \frac{W_F F_W}{W_F^1 F_W^1}\right) + \theta\left(F_W - F_W^1\right)\left(V_S - V_S^1\right) \\ &= a_2^3 G(3,2) + a_7^3 G(3,7) \end{aligned}$$

$$\begin{aligned} V_4' &= \left(A_L - A_L^1\right)\left(\xi F_W + \gamma_2 A_Q - x_3 A_L\right) \\ &\le \xi\left(A_L - A_L^1\right)\left(F_W - F_W^1\right) + \gamma_2\left(A_L - A_L^1\right)\left(A_Q - A_Q^1\right) \\ &= a_3^4 G(4,3) + a_6^4 G(4,6) \end{aligned}$$

$$\begin{aligned} V_5' &= \left(H_Y - H_Y^1\right)\left(\varepsilon_1 A_L - x_4 H_Y\right) \\ &\le \varepsilon_1 \left(H_Y - H_Y^1\right)\left(A_L - A_L^1\right) \\ &= a_4^5 G(5,4) \end{aligned}$$

$$\begin{aligned} V_6' &= \left(A_Q - A_Q^1\right)\left(\delta_2 F_W + \gamma_1 A_L + \varepsilon_2 H_Y - x_5 A_Q\right) \\ &\le \delta_2 \left(A_Q - A_Q^1\right)\left(F_W - F_W^1\right) + \gamma_1 \left(A_Q - A_Q^1\right)\left(A_L - A_L^1\right) + \varepsilon_2 \left(A_Q - A_Q^1\right)\left(H_Y - H_Y^1\right) \\ &= a_3^6 G(6,3) + a_4^6 G(4,6) + a_5^6 G(6,5) \end{aligned}$$

$$\begin{aligned} V_7' &= \left(V_S - V_S^1\right)\left(\alpha H_Y - x_6 V_S\right) \\ &\le \alpha \left(V_S - V_S^1\right)\left(H_Y - H_Y^1\right) \\ &= a_5^7 G(7,5) \end{aligned}$$

$$\begin{aligned} V_8' &= \left(V - V^1\right)\left(\eta_1 H_Y - \mu V\right) \\ &\le \eta_1 \left(V - V^1\right)\left(H_Y - H_Y^1\right) \\ &= a_5^8 G(8,5) \end{aligned}$$

$$\begin{aligned} V_9' &= \left(F - F^1\right)\left(\eta_2 A_Q - \mu F\right) \\ &\le \eta_2 \left(F - F^1\right)\left(A_Q - A_Q^1\right) \\ &= a_6^9 G(9,6) \end{aligned}$$

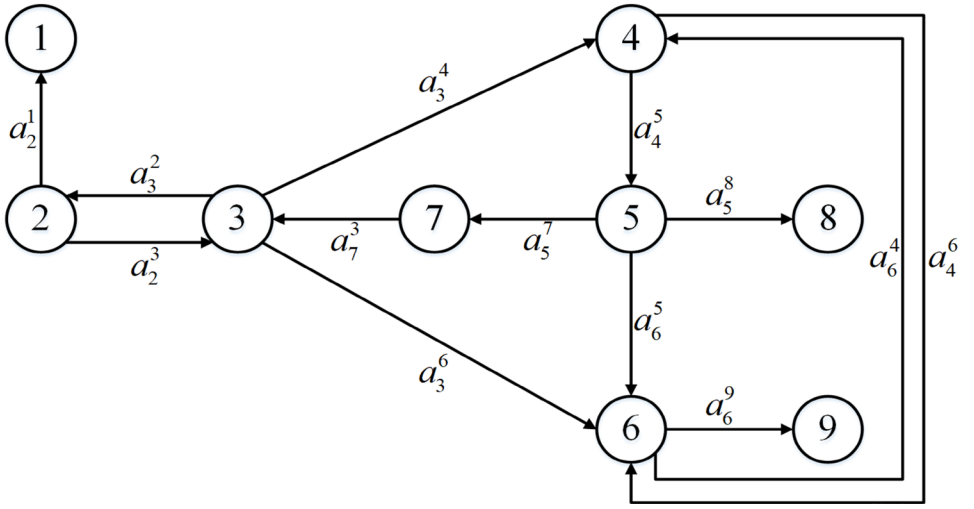
Using the set of vertices and its combination, we result with a weighted graph as shown in figure 2 with

$$a_2^1 = 2\mu, a_3^2 = a_2^3 = \beta_2 W_F^1 F_W^1, a_7^3 = \theta, a_3^4 = \varepsilon, a_6^4 = \gamma_2,$$
$$a_4^5 = \varepsilon_1, a_3^6 = \delta_2, a_4^6 = \gamma_1, a_5^6 = \varepsilon_2, a_5^7 = \alpha, a_5^8 = \eta_1, a_6^9 = \eta_2$$

and others a_{ij}=0.

This weighted graph has 9 vertices and 4 cycles viz.

Figure 2. The weighted graph of aquaponic system for vacuum process free equilibrium point



$$G(2,3)+G(3,2)=0, G(3,2)+G(4,3)+G(5,4)+G(7,5)+G(3,7)+G(2,3)=0,$$
$$G(4,3)+G(5,4)+G(7,5)+G(3,7)=0, G(5,4)+G(6,5)+G(6,4)=0$$

Then as assumptions taken in theorem 4, there exists c_i, $1 \le i \le 9$ such that $V=\sum_{i=1}^{9} c_i V_i$ becomes the Lyapunov function. Using theorem 2 and 3, we have

$$d^+(7)=1 \Rightarrow c_3 a_7^3 = c_7 a_5^7 \tag{5}$$

$$d^-(1)=1 \Rightarrow c_1 a_2^1 = 0 \Rightarrow c_1 = 0 \tag{6}$$

$$d^-(2)=1 \Rightarrow c_2 a_3^2 = c_1 a_2^1 + c_3 a_2^3 \Rightarrow c_2 a_3^2 = c_3 a_2^3 \tag{7}$$

$$d^-(5)=1 \Rightarrow c_5 a_4^5 = c_6 a_5^6 + c_7 a_5^7 + c_8 a_5^8 \tag{8}$$

$$d^{-}(7)=1\Rightarrow c_7a_5^7=c_3a_7^3 \tag{9}$$

$$d^{-}(8)=1\Rightarrow c_8a_5^8=0\Rightarrow c_8=0 \tag{10}$$

$$d^{-}(9)=1\Rightarrow c_9a_6^9=0\Rightarrow c_9=0 \tag{11}$$

Now, taking c_2=t_1 and since $a_3^2=a_2^3$ then by (7) c_2=c_3=t_1, Also, by (5) and (9), $c_7=\frac{t_1\theta}{\alpha}$. Again taking c_5=t_2 and by (4),

$$c_6=\frac{t_2\varepsilon_1-\frac{t_1\theta}{\alpha}}{\varepsilon_2}\Rightarrow c_6=\frac{t_2\varepsilon_1\alpha-t_1\theta}{\varepsilon_2}$$

Therefore,

$$V=\sum_{i=1}^{9}c_iV_i=c_1V_1+c_2V_2+c_3V_3+c_4V_4+c_5V_5+c_6V_6+c_7V_7+c_8V_8+c_9V_9$$
$$=t_1V_2+t_1V_3+t_2V_5+\left(\frac{t_2\varepsilon_1\alpha-t_1\theta}{\varepsilon_2}\right)V_6+\left(\frac{t_1\theta}{\alpha}\right)V_7$$

where t_1 and $t_2$are arbitrary constants.

This verifies that E^1 is the only invariant set in int(Λ), where $V'=0$. Hence, E^1 is globally asymptotically stable in int(Λ).

Theorem 6: The endemic equilibrium point E^* is globally asymptotically stable in int(Λ).

Proof: Let us construct Lyapunov function $D(t)$ so that

$$D=D_1+D_2+D_3+D_4+D_5+D_6+D_7+D_8+D_9+D_{10}$$

where

$$D_1 = W_W - W_W^* - \ln\frac{W_W}{W_W^*}, D_2 = W_F - W_F^* - \ln\frac{W_F}{W_F^*}, D_3 = V_P - V_P^* - \ln\frac{V_P}{V_P^*},$$
$$D_4 = F_W - F_W^* - \ln\frac{F_W}{F_W^*}, D_5 = A_L - A_L^* - \ln\frac{A_L}{A_L^*}, D_6 = H_Y - H_Y^* - \ln\frac{H_Y}{H_Y^*},$$
$$D_7 = A_Q - A_Q^* - \ln\frac{A_Q}{A_Q^*}, D_8 = V_S - V_S^* - \ln\frac{V_S}{V_S^*}, D_9 = V - V^* - \ln\frac{V}{V^*}, D_{10} = F - F^* - \ln\frac{F}{F^*}$$

Differentiating D_1 with respect to time *t*, we get

$$\begin{aligned} D_1' &= \left(1-\frac{W_W^*}{W_W}\right)\left(B_1 - \beta_1 W_W V_P - \mu W_W\right) \\ &= \left(1-\frac{W_W^*}{W_W}\right)\left(\beta_1 W_W^* V_P^* - \beta_1 W_W V_P + \mu W_W^* - \mu W_W\right) \\ &= \beta_1\left(1-\frac{W_W^*}{W_W}\right)\left(W_W^* V_P^* - W_W V_P\right) - \mu\frac{\left(W_W - W_W^*\right)^2}{W_W} \\ &\le \beta_1\left(1-\frac{W_W^*}{W_W}\right)\left(W_W^* V_P^* - W_W V_P\right) \\ &= e_3^1 G^*(1,3) \end{aligned}$$

Similarly, differentiating others with respect to time *t*, we have

$$\begin{aligned} D_2' &= \left(1-\frac{W_F^*}{W_F}\right)\left(B_2 - \beta_2 W_F F_W - \mu W_F\right) \\ &\le \beta_2\left(1-\frac{W_F^*}{W_F}\right)\left(W_F^* F_W^* - W_F F_W\right) \\ &= e_4^2 G^*(2,4) \end{aligned}$$

$$
\begin{aligned}
D_3' &= \left(1-\frac{V_P^*}{V_P}\right)\left(\beta_1 W_W V_P - x_1 V_P\right) \\
&\le \beta_1\left(1-\frac{V_P^*}{V_P}\right)\left(W_W V_P - W_W^* V_P^*\right) \\
&= e_1^3 G^*\left(3,1\right)
\end{aligned}
$$

$$
\begin{aligned}
D_4' &= \left(1-\frac{F_W^*}{F_W}\right)\left(\beta_2 F_W W_F + \theta V_S - x_2 F_W\right) \\
&\le \beta_2\left(1-\frac{F_W^*}{F_W}\right)\left(F_W W_F - W_F^* F_W^*\right) + \theta\left(1-\frac{F_W^*}{F_W}\right)\left(V_S - V_S^*\right) \\
&= e_2^4 G^*\left(4,2\right) + e_8^4 G^*\left(4,8\right)
\end{aligned}
$$

$$
\begin{aligned}
D_5' &= \left(1-\frac{A_L^*}{A_L}\right)\left(\delta_1 V_P + \xi F_W + \gamma_2 A_Q - x_3 A_L\right) \\
&\le \delta_1\left(1-\frac{A_L^*}{A_L}\right)\left(V_P - V_P^*\right) + \xi\left(1-\frac{A_L^*}{A_L}\right)\left(F_W - F_W^*\right) + \gamma_2\left(1-\frac{A_L^*}{A_L}\right)\left(A_Q - A_Q^*\right) \\
&= e_3^5 G^*\left(5,3\right) + e_4^5 G^*\left(5,4\right) + e_7^5 G^*\left(5,7\right)
\end{aligned}
$$

$$
\begin{aligned}
D_6' &= \left(1-\frac{H_Y^*}{H_Y}\right)\left(\varepsilon_1 A_L - x_4 H_Y\right) \\
&\le \varepsilon_1\left(1-\frac{H_Y^*}{H_Y}\right)\left(A_L - A_L^*\right) \\
&= e_5^6 G^*\left(6,5\right)
\end{aligned}
$$

$$D_7' = \left(1-\frac{A_Q^*}{A_Q}\right)\left(\delta_2 F_W + \gamma_1 A_L + \varepsilon_2 H_Y - x_5 A_Q\right)$$
$$\leq \delta_2\left(1-\frac{A_Q^*}{A_Q}\right)\left(F_W - F_W^*\right) + \gamma_1\left(1-\frac{A_Q^*}{A_Q}\right)\left(A_L - A_L^*\right) + \varepsilon_2\left(1-\frac{A_Q^*}{A_Q}\right)\left(H_Y - H_Y^*\right)$$
$$= e_4^7 G^*(7,4) + e_5^7 G^*(7,5) + e_6^7 G^*(7,6)$$

$$D_8' = \left(1-\frac{V_S^*}{V_S}\right)\left(\alpha H_Y - x_6 V_S\right)$$
$$\leq \alpha\left(1-\frac{V_S^*}{V_S}\right)\left(H_Y - H_Y^*\right)$$
$$= e_6^8 G^*(8,6)$$

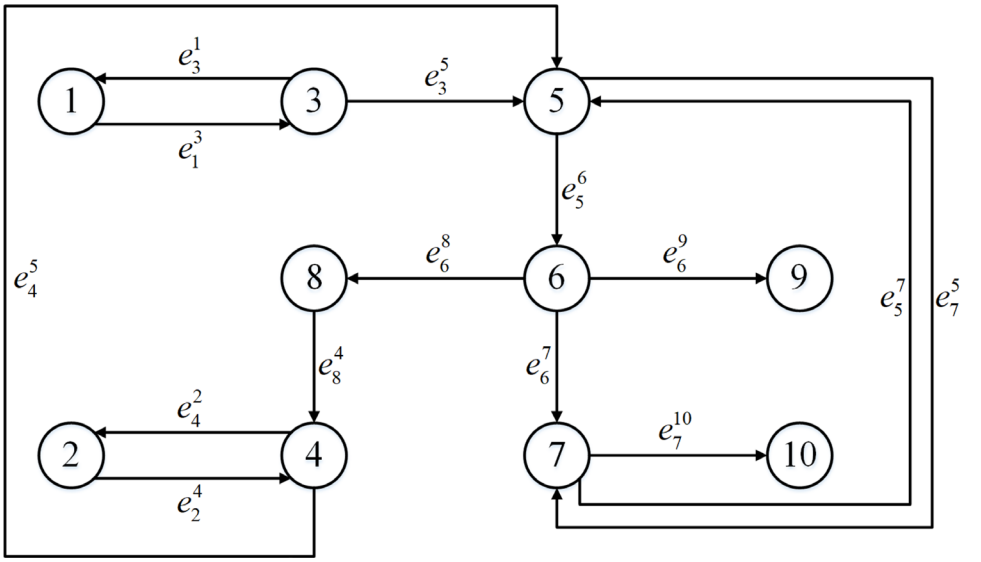
$$D_9' = \left(1-\frac{V^*}{V}\right)\left(\eta_1 H_Y - \mu V\right)$$
$$\leq \eta_1\left(1-\frac{V^*}{V}\right)\left(H_Y - H_Y^*\right)$$
$$= e_6^9 G^*(9,6)$$

$$D_{10}' = \left(1-\frac{F^*}{F}\right)\left(\eta_2 A_Q - \mu F\right)$$
$$\leq \eta_2\left(1-\frac{F^*}{F}\right)\left(A_Q - A_Q^*\right)$$
$$= e_7^{10} G^*(10,7)$$

With help of the set of 10 vertices and its combination, a resultant weighted graph is displayed in figure 3 with

$$e_3^1 = e_1^3 = \beta_1, e_4^2 = e_2^4 = \beta_2, e_8^4 = \theta, e_3^5 = \delta_1, e_4^5 = \xi, e_7^5 = \gamma_2,$$
$$e_5^6 = \varepsilon_1, e_4^7 = \delta_2, e_5^7 = \gamma_1, e_6^7 = \varepsilon_2, e_6^8 = \alpha, e_6^9 = \eta_1, e_7^{10} = \eta_2$$

Figure 3. The weighted graph of aquaponic system



and others a_{ij}=0.

This weighted graph is having 10 vertices as well as 4 cycles namely

$$G^*(1,3)+G^*(3,1)=0, G^*(2,4)+G^*(4,2)=0,$$
$$G^*(6,5)+G^*(7,6)+G^*(5,7)=0, G^*(6,5)+G^*(8,6)+G^*(4,8)+G^*(5,4)=0.$$

Then as assumptions taken in theorem 4, there exists d_i, $1 \le i \le 10$ such that $D=\sum_{i=1}^{10} d_i D_i$ becomes the Lyapunov function. Using theorem 2 and 3, we have

$$d^+(1)=1 \Rightarrow d_3 e_1^3 = d_1 e_3^1 \tag{12}$$

$$d^+(2)=1 \Rightarrow d_4 e_2^4 = d_2 e_4^2 \tag{13}$$

$$d^+(8)=1 \Rightarrow d_4 e_8^4 = d_8 e_6^8 \tag{14}$$

$$d^{-}(1)=1 \Rightarrow d_1 e_3^1 = d_3 e_1^3 \tag{15}$$

$$d^{-}(2)=1 \Rightarrow d_2 e_4^2 = d_4 e_2^4 \tag{16}$$

$$d^{-}(3)=1 \Rightarrow d_3 e_1^3 = d_1 e_3^1 + d_5 e_3^5 \tag{17}$$

$$d^{-}(6)=1 \Rightarrow d_6 e_5^6 = d_7 e_6^7 + d_8 e_6^8 + d_9 e_6^9 \tag{18}$$

$$d^{-}(8)=1 \Rightarrow d_8 e_6^8 = d_4 e_8^4 \tag{19}$$

$$d^{-}(9)=1 \Rightarrow d_9 e_6^9 = 0 \Rightarrow d_9 = 0 \tag{20}$$

$$d^{-}(10)=1 \Rightarrow d_{10} e_7^{10} = 0 \Rightarrow d_{10} = 0 \tag{21}$$

Considering $d_1=h_1$ and since $e_3^1 = e_1^3 = \beta_1$ then by (12) $d_1=d_3=h_1$. Moreover, taking $d_2=h_2$ and since $e_4^2 = e_2^4 = \beta_2$ then by (13) $d_2=d_4=h_2$.

By (14) and (17), we have $d_8 = \dfrac{h_2\theta}{\alpha}$ and $d_5=0$, respectively. Again taking $d_6=h_3$ then by (18) $d_7 = \dfrac{h_3\varepsilon_1 - \left(\dfrac{h_2\theta}{\alpha}\right)}{\varepsilon_2} = \dfrac{h_3\alpha\varepsilon_1 - h_2\theta}{\alpha\varepsilon_2}$

Therefore,

$$D = \sum_{i=1}^{10} d_i D_i = d_1D_1 + d_2D_2 + d_3D_3 + d_4D_4 + d_5D_5 + d_6D_6 + d_7D_7 + d_8D_8 + d_9D_9 + d_{10}D_{10}$$

$$= h_1D_1 + h_2D_2 + h_1D_3 + h_2D_4 + h_3D_6 + \left(\frac{h_3\alpha\varepsilon_1 - h_2\theta}{\alpha\varepsilon_2}\right)D_7 + \left(\frac{h_2\theta}{\alpha}\right)D_8$$

where h_1,h_2 and h_3 are arbitrary constants.

This verifies that E^* is the only invariant set in int(Λ), where $D' = 0$. Hence, E^* is globally asymptotically stable in int(Λ).

NUMERICAL SIMULATION

In this section, transmission of water and food waste into aquaponic system is calculated. The system of the model is simulated numerically to validate our mathematical results. To study the periodicity of the model, we have considered the parametric values as B_1=0.20, B_2=0.30, b_1=0.20, b_2=0.50, δ_1=0.15, δ_2=0.30, γ_1=0.10, γ_2=0.70, ε_1=0.05, ε_2=0.60, α=0.10, θ=0.60, ξ=0.50, η_1=0.20, η_2=0.28, μ=0.40.

The endemic equilibrium point E^* has all negative eigenvalues when t=0 which are obtained as -0.4000, -0.4000, -0.4442, -1.0820, -1.0820, -1.4130, -1.4130, -0.4000, -0.4000 and -0.5500. All negative eigenvalues corresponding to E^* establish the local asymptotical stability.

To measure, oscillating waste disposal, the periodic functions $\beta_1(t)$ and $\beta_2(t)$ have been considered as indicated in the following form

$$\beta_1(t) = b_1 \sin\left(\frac{\pi t}{7}\right) \text{ and } \beta_2(t) = b_2 \sin\left(\frac{\pi t}{7}\right)$$

where b_1 and b_2 are constants whereby $\beta_1(t)$ and $\beta_2(t)$ remain positive.

Figure 4. Process of waste

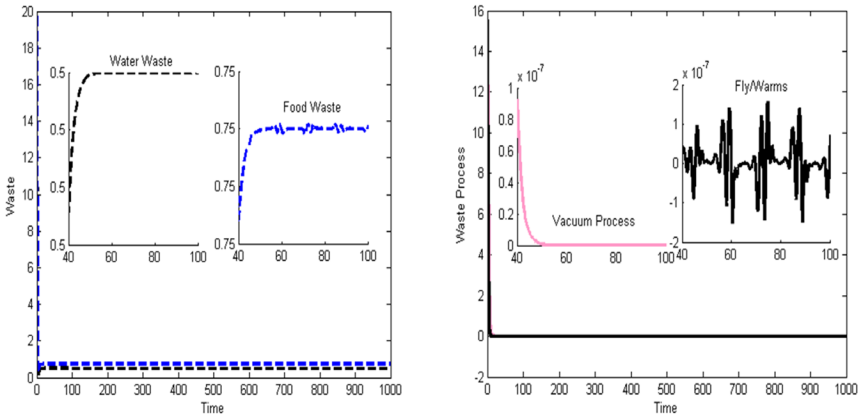


Figure 4 indicates that waste water continuously proceeds for vacuum process where waste food passes through fly/worms to serve fish food in asymmetric periodic manner.

Figure 5. Transmission of Algae, hydroponic and aqua cultures

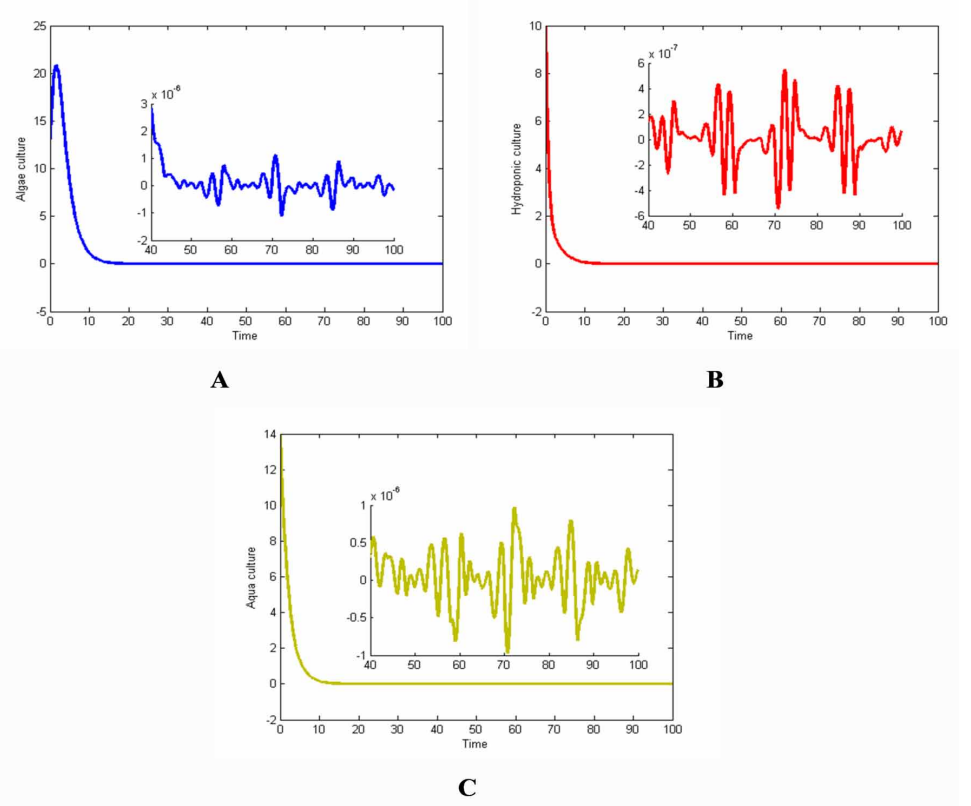


Figure 5 directs that in hydroponic system, nutrients taken from algae culture are less than needed in compared to nutrients taken from aqua culture. These cultures suggest that hydroponic vegetables production is oscillating.

Figure 6 denotes the by-products of hydroponic vegetables i.e. vegetable scrap that are also used in hydroponic vegetables cultivation cycle. The fluctuation of leftover vegetable scrap depends upon the production of hydroponic vegetables.

The production of vegetables and conservation of fishes is reflected through figure 7. As hydroponic and aqua cultures fluctuate, the results of these cultures also change over the time. This means the production of vegetables does not persist

Figure 6. Oscillation of vegetable scrap

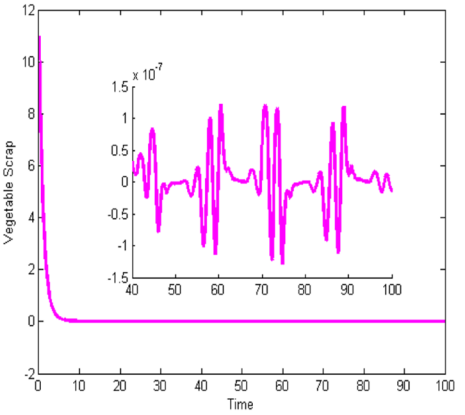
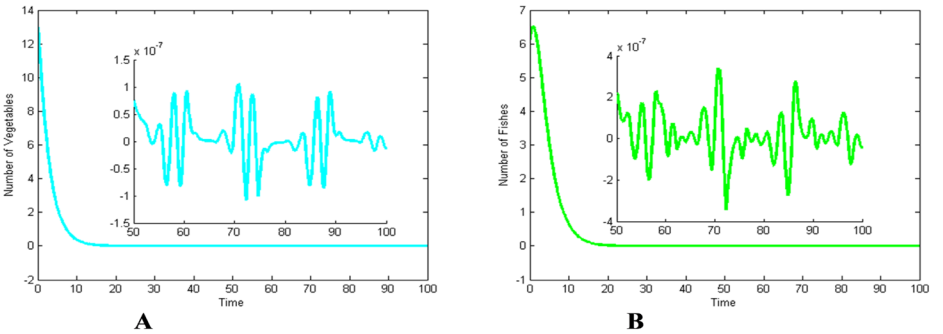


Figure 7. Fluctuation of vegetable production and fish conservation



stable, although it depends based on the scrap on the other hand fish conservation depends on the existing nutrients in the water.

Figure 8 reflects damp harmonic oscillation in whole aquaponic system. This variation is due to seasonal change that is if this study periodically falls between end of summer and beginning of rainy seasons. Also, it depends upon the close waster loop.

Figure 9 shows the bifurcation analysis of the aquaponic system. For the bifurcation, we have taken b_1 and b_2 as bifurcation parameters Figures A, C, E, F, H, J, L, N, P and R express the bifurcation is when b_1 is varied and figures B, D, G, I, K, M, O, Q, S denote the same when b_2 is varied. Here, system does not lead to change because aquatic nutrients and vegetables scrap are sent back to be reused in the cycle of aquaponic system. Figures A – D suggest that variation of b_1 is more useful than b_2. In the case of waste water, it may be because b_1 is directly related

Figure 8. Aquaponic system

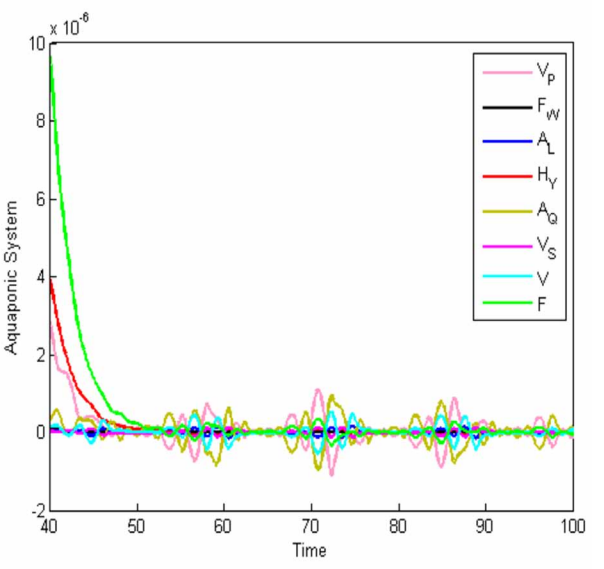
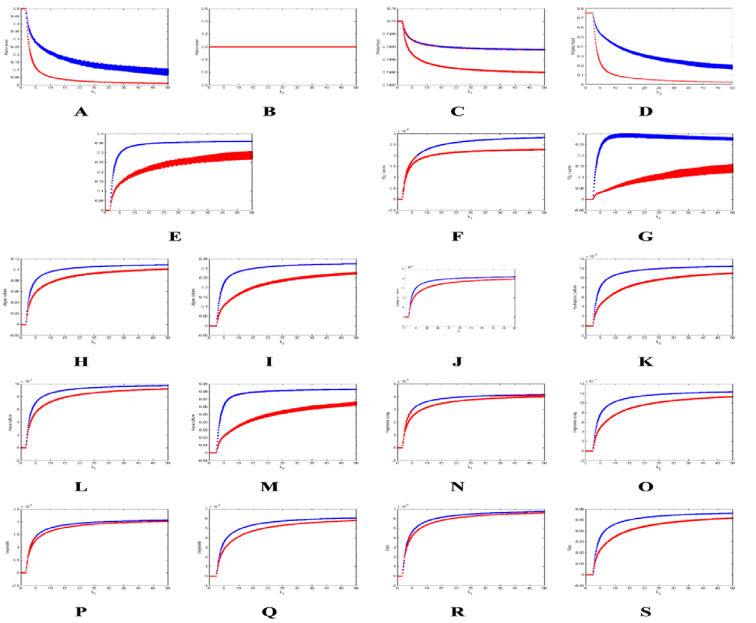


Figure 9. Bifurcation analysis when b_1 and b_2 varied



to waste water. Also, in the case of waste food, the same scenario can be observed. Figure E gives the idea to waste water which is going for a vacuum process which is further used in algae culture. Similarly, figures F and G show the waste food sent for processing using fly/worms which helps to produce fish food. It also confirms that when b_2 is varied the process becomes more effective. Figures H – S reflect the state of producing vegetables and fishes using algae, aquaponic and hydroponic culture which sometimes left with the vegetable scrap. Here, blue colour denotes the maximum production whereas red colour signifies the minimum production.

CONCLUSION

In this study, aquaponic system has been considered with sinusoidal periodic function. The model is taken care by computing threshold quantity to revive agriculture and aquatic balance. The threshold quantity is based upon the periodic function consequently on the values of b_1 and b_2 constants which can be concluded from figure 10.

Figure 10. Variation of R_0 w.r.t. b_1 and b_2

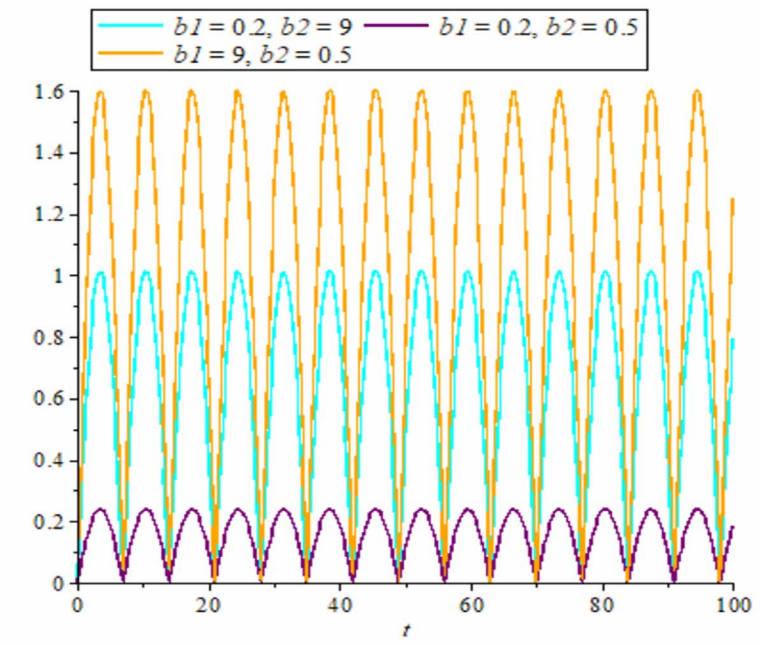
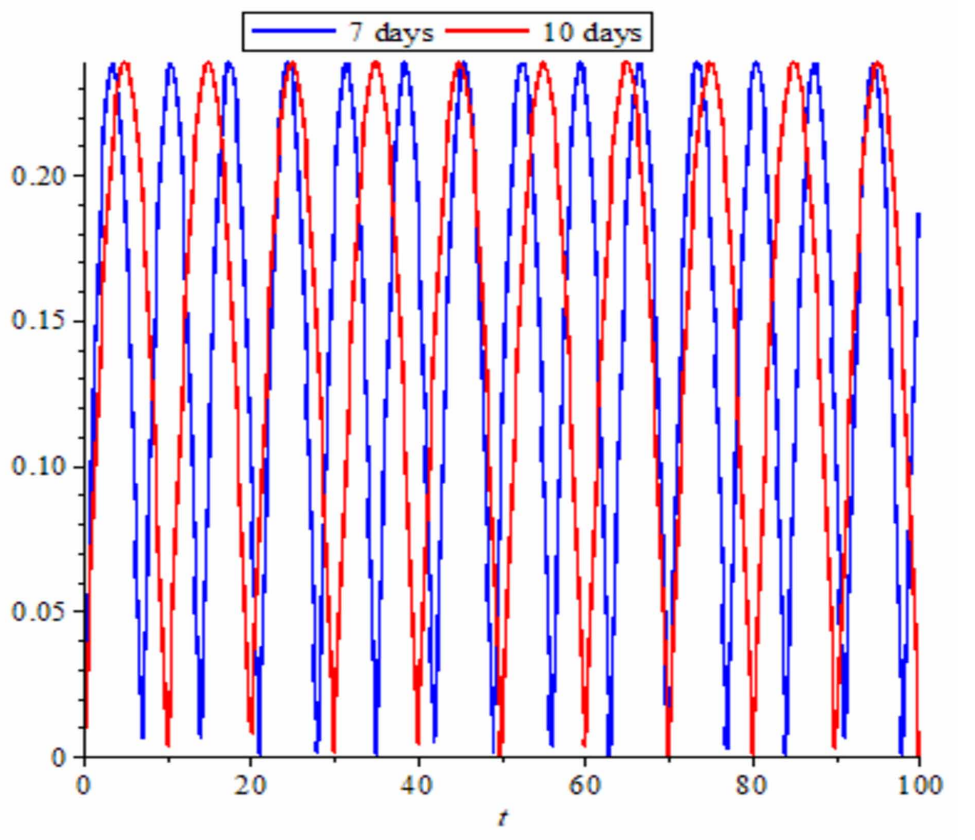


Figure 10 also states that with larger values of b_1 and b_2, the absolute oscillations are bigger than the smaller values. Here, we have taken n in days for periodic function, this means R_0 is also dependent upon the value of n. To study, how much waste is processed we have plotted figure 11.

Figure 11. R_0 of different values of n



For 7 days, waste is processed but higher than that in 10 days. This advocates recycling of waste should be done in few days as possible.

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Chapter 11
Vertical Transmission of Syphilis With Control Treatment

Zalak Ashvinkumar Patel
L. D. College of Engineering, India

Nita H. Shah
https://orcid.org/0000-0003-1605-4778
Department of Mathematics, Gujarat University, Ahmedabad, India

ABSTRACT

Syphilis is a sexually transmitted disease having different signs and symptoms with four main stages, namely primary, secondary, latent, and tertiary. Congenital (vertical) transmission of syphilis from infected mother to fetus or neonatal is still a cause of high perinatal morbidity and mortality. A model of transmission of syphilis with three different ways of transmission, namely vertical, heterosexual, and homosexual, is formulated as a system of nonlinear ordinary differential equations. Treatment is also incorporated at various stages of infection. Total male and female population is divided in various classes (i.e., were susceptible, exposed, primary and secondary infected, early and late latent, tertiary, infected treated, latent treated, infected child [newborn], and treated infected child [at birth time]). Stability of disease-free equilibrium and endemic equilibrium is established. Control treatment is applied. It is observed that safe sexual habits and controlled treatment in each stage including pregnancy are effective parameters to curb disease spread.

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INTRODUCTION

Syphilis is a sexually transmitted disease caused by infection from bacteria *Treponema pallidum (T. pallidum)*. Treponema pallidum is a spirochete bacterium usually transmitted by sexual contact through exposure to mucocutaneous syphilitic lesions. Wright and Jones (2003) have observed that in body fluid the infecting organism starts to replicate locally. As per WHO (2007) report, annually 12 million active infections of syphilis are getting reported. According to CDC (2010) report, there were total 13,774 cases of primary and secondary syphilis reported during 2010. Values *et al.* (2000) have reported that about 30 to 50% of individuals suffering from primary or secondary syphilis are at serious risk of spreading disease through sexual contacts. They also have observed that more than 80% of women with syphilis are in their reproductive (20 to 35 years old) age which causes high risk of congenital transmission. Walker and Walker (2002) have observed that each year high numbers of pregnancies are getting badly affected in all over world and yearly around 4,60,000 pregnancies end in abortion and 2,70,000 babies born prematurely or with low birth weight because of maternal syphilis. As per CDC (2010) report, rates of female and congenital syphilis were increased during 2005-2008 in the USA. In 2008, WHO estimated that 1.9 million pregnant women had active syphilis. Stolte *et al.* (2001), Simms *et al.* (2005), Heffelfinger *et al.* (2007) and Read *et al.* (2015) have reported revival of syphilis amongst homosexual male population in early 2000 even though overall decrease is reported from 1990.

In 2006, lifetime medical cost per case of syphilis was estimated as $572. Chesson *et al.* (2008) observed that the treatment cost per case could get increased in case of congenital infection. Blandford and Gift (2003) suggested early stage treatment to reduce the cost per case as early stage treatment is less expensive than treatment for later stage disease.

Dynamics of Syphilis Amongst Adults

After infection in humans, syphilis thrives through multiple stages if not treated. As per Garnett *et al.* (1997) after inoculation, first the exposed stage of an average 28 days executes in which individuals remains infected but not infectious. Garnett *et al* (1997), Singh *et al*. (1999) and LaFond *et al.* (2006) observed that the primary infected stage, characterized by a single painless chancre at the place of inoculation lasts up to an average 46 days of its (chancre) appearing to heal after the exposed period. The primary infected untreated patients progresses to secondary syphilis, characterized by multiple symptoms, like copper coloured skin lesions that tend to be universally distributed on whole body, the soles with painless lymphadenopathy, occurrence of meningism and headache and with less common symptoms like

alopecia, laryngitis, mild hepatitis, nephrotic syndrome, bone ache, and uveitis. Garnett *et al.* (1997), Singh *et al.* (1999) and LaFond *et al.* (2006) notified that this stage lasts up to an average of about 15 weeks. Singh *et al.* (1999) and LaFond *et al.* (2006) have reported that an untreated secondary syphilis develops asymptomatic state, also called latency which starts with two arbitrarily subdivided stages early (up to 2 years from primary infection) and late latency (approximately 2-46 years after primary infection or till tertiary stage progresses). LaFond *et al.* (2006) and French (2007) observed that about 30 to 40% of untreated cases develop the tertiary stage from latency. French (2007) and Marco De (2012) observed that during tertiary stage of syphilis infection individual may develop either cardiovascular syphilis (10%) or gummatous syphilis (15%) or neurosyphilis (6.5%) or meningovascular syphilis with the incubation period of 5 to12 years. Singh *et al.* (1999) noted that disease induced mortality occurs at tertiary stage only. Kent and Romanelli (2008) reported that people with tertiary syphilis are not infectious.

Dynamics of Syphilis in Pregnancy

Irrelevant to downward trend in syphilis cases, vertical transmission of syphilis during pregnancy or at the birth time remains a large concern. Ingall and S´anchez (2001) notified that vertical transmission of syphilis can occur at any stage of syphilis and it is threat to the pregnancy and fetus. Values (2000), Goldenberg and Thompson (2003) have observed that fetus infection occurs while spirochetes cross the placenta from about 14 weeks' gestation which increases risk of congenital syphilis. Oswal (2008) has notified that women around 50% from primary stage and 50% from secondary stage, 40% from early latent and 10% from late latent stage of syphilis are responsible for vertical transmission. About 70% of infants born to untreated infected mothers are infected as per Hawkes *et al.* (2011). Saloojee *et al.* (2004) studied that the gestational age, stage of maternal syphilis, maternal treatment, and immunological response of the fetus are decisive terms for progress of congenital syphilis. Jensen (1999) has studied that vertical transmission can lead to abortion after the first trimester, or late-term stillbirth in 30 to 40 percent of cases or premature delivery of live infants who may have obvious signs of infection or be fully asymptomatic (approximately two-thirds of live born cases).

Watson-Jones *et al.* (2002) and Sheffield *et al.* (2002) has observed that approximately 35% of infected fetus takes birth with congenital syphilis, out of which 60% are asymptomatic at birth. Congenital syphilis has two stages of infection early congenital syphilis and late congenital syphilis. Ingall and S´anchez (2001) studied that baby having early congenital syphilis develop symptoms in the first 2 years of life while having late congenital syphilis develop symptoms up to 20 years of life.

Ingall and S´anchez (2001) and Woods (2005) have observed that approximately 40% of untreated children undergoes with late congenital syphilis. Nabarro (1954) has studied that irritability, failure to thrive, non-specific fever, a rash and condyloma lata on the borders of the mouth, anus, and genitalia are symptoms of early stage. A small percentage of infants have a watery nasal discharge (sniffles) and a saddle nose deformity resulting from destruction of the cartilage of the nose. Later signs appear as tooth abnormalities (Hutchinson teeth), bone changes (sabre shins), neurological disorder, blindness, and deafness as per Oswal (2008).

Treatment for syphilis does exist. Singh and Romanowski (1999) and Workowski and Bolan (2015) suggested that a single dose of benzanthine penicillin G, 2.4 mU is administered in the primary infection, secondary infection and early latent stages while in the late latent stage three doses of benzanthine penicillin G, 2.4 mU at 1 week intervals are administered. Singh and Romanowski (1999) studied that treatment in the tertiary stage is not much intensive. Also, organ failure or neurological disorder caused by syphilis in tertiary stage cannot be undone. To treat Fetal infection and to prevent fetus from vertical transmission treatment of penicillin G is administrated parentally for treating of syphilis as per study of Workowski and Berman (2010), Wendel Jr. *et al.* (2002), Workowski and Berman (2010) have suggested that aqueous crystalline penicillin G is administrated in case of neurosyphilis treatment.

Several mathematical models of syphilis have been analyzed. Garnett *et al.* (1997) suggested a model having common latent class with no treated class and suggested that individuals after getting treatment at any stage return to susceptible class. Pourbohloul *et al.* (2002) suggested a model of heterosexual syphilis transmission in East Vancouver, with 210 ordinary differential equations formed by dividing population in to multiple groups based on sex, sexual activity and age. Fenton *et al.* (2008) reviewed the results of published mathematical models of syphilis up to 2008. A multistage model for syphilis including early and late latent stages with immunity followed by treatment in an infectious or latent stage is formulated and analyzed by Iboi and Okuonghae (2016). A mathematical model of syphilis transmission amongst MSM population with treatment class at each infectious and latent stage is suggested by Saad-Roy *et al.* (2016).

The objective of this chapter is to analyse the effect of control treatment on vertical transmission of syphilis. The whole population is divided in to eleven compartments viz. Susceptible, Exposed, primary Infected, Secondary Infected, early latent, late latent, Tertiary and Child Infected with disease induced death rate in Tertiary stage and natural death rate at any stage.

The chapter is organised as follow. Section 2, starts with notation, calculation of force of infection and followed by mathematical model. In Section 3, basic reproduction number is calculated using next generation matrix method. The disease free and endemic equilibrium of the system are established and their stability analysis

is carried out. In section 4, the optimal control calculation is carried out, which is followed by numerical simulation in section 5. In section 6 conclusions are drawn.

Mathematical Model

The mathematical model is developed with the notations from Table 1.

A non-linear dynamical system of differential equations is suggested to study the spread of syphilis with vertical transmission and controlled treatment. Total population is divided in to eleven compartments, viz. Susceptible, Exposed, Primary infected, Secondary infected, Early latent, Late latent, tertiary, child infected, infected (Primary and secondary both) treated, latent (early and late) treated, child infected treated.

The flow of population amongst above compartments is shown in figure 1.

To prepare the model, we have considered following possibilities of disease spread.

(1) Vertical Transmission of infection to new-borns.
(2) Heterosexual transmissions amongst adults (from man to women).
(3) MSM transmission (from man to man sexual transmission).

To formulate the model following assumption are taken in account

It is assumed that the tertiary individuals are not taking part in sexual activities and hence not responsible for disease spread via sexual activity. It is also assumed that, new born child either enters in susceptible group having no infection at birth time due to maternal treatment or enters in child infected class with neonatal infection.

Hence force of infections are defined by,

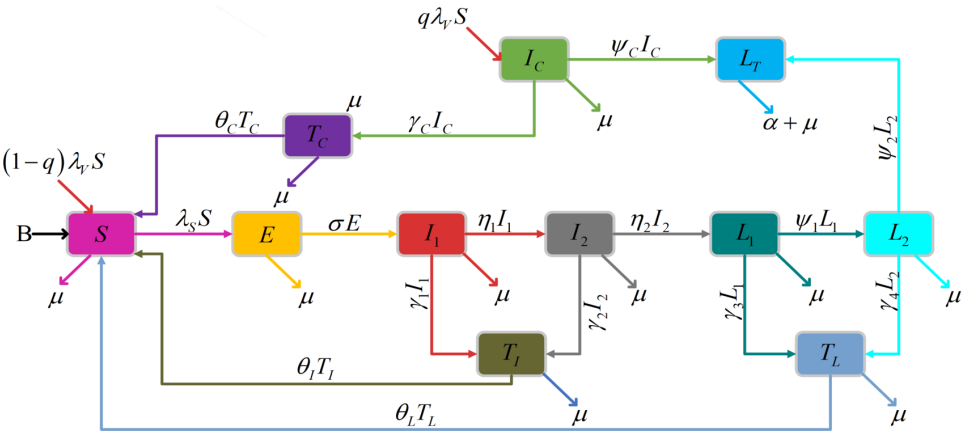
$$\lambda_S = \beta_1 \frac{I_1}{N} + \beta_2 \frac{I_2}{N} + \beta_3 \frac{I_1}{N} + \beta_4 \frac{I_2}{N} \text{ and } \lambda_V = \delta\left(P_1 \frac{I_1}{N} + P_2 \frac{I_2}{N} + P_3 \frac{L_1}{N} + P_4 \frac{L_2}{N} \right)$$

The susceptible individual gets infection from sexual (both heterosexual and MSM activities) contacts with primary and secondary infected individual and moves towards exposed compartment at rate γ_S. Exposed individual moves towards primary infected compartment at rate σ. Primary infected individual either gets treatment at rate γ_1 and moves towards treated infected compartment or develops next stage of infection and moves towards secondary infected compartment at rate η_1. Secondary infected individual either gets treatment at rate γ_2 and moves towards treated infected compartment or develops next stage of infection and moves towards early latent compartment at rate η_2. Either with treatment early latent individual joins treated latent compartment at rate γ_3 or joins late latent compartment at rate ψ_1 if not treated.

Table 1. Notation and parametric values

Notations	Description	Parametric value
$N(t)$	Total Population at time t	10000
$S(t)$	Number of susceptible individuals at time t	500
$E(t)$	Number of exposed individuals at time t	70
$I_1(t)$	Number of primary infected individuals at time.	50
$I_2(t)$	Number of secondary infected individuals at time t	30
$I_C(t)$	Number of new-born infected child individuals at time t	8
$L_1(t)$	Number of early latent individuals at time t	50
$L_2(t)$	Number of late latent individuals at time t	35
$L_T(t)$	Number of tertiary (final stage) individuals at time t	4
$T_I(t)$	Number of treated individuals from infected individuals at time t	28
$T_L(t)$	Number of treated individuals from latent individuals at time t	27
$T_C(t)$	Number of treated child individuals from infected child individuals at time t	5
B	New recruitments	20
β_1 and β_2	Disease transmission rate by contacts of primary and secondary infected individuals with susceptible individuals due to heterosexual activities	β_1=0.8 β_2=0.4
β_3 and β_4	Disease transmission rate by contacts of primary and secondary infected individuals with susceptible individuals due to MSM sexual activities	β_3=0.7 β_4=0.3
P_1	Probability of new child birth from mother having primary stage infection	0.25
P_2	Probability of new child birth from mother having secondary stage infection	0.25
P_3	Probability of new child birth from mother having primary latent stage	0.25
P_4	Probability of new child birth from mother having late latent stage	0.25
q	Probability of new child birth having neonatal (vertical) infection	0.5
δ	Number of new child birth	4
μ	Mortality rate	0.4
α	Disease induced death rate	0.7
σ	Transmission rate from exposed class to primary infected class	0.8
η_1	Transmission rate from primary infected class to secondary infected class	0.5
η_2	Transmission rate from secondary infected class to early latent class	0.4
ψ_1	Transmission rate from early latent class to late latent class	0.15
ψ_2	Transmission rate from late latent class to tertiary class	0.15
ψ_C	Transmission rate from child infected class to tertiary class	0.2
θ_I	Rate of loss of immunity from treated infected class	28
θ_L	Rate of loss of immunity from treated latent class	27
θ_C	Rate of loss of immunity from child treated infected class	5
γ_1	Treatment rate from primary infected class	0.5
γ_2	Treatment rate from secondary infected class	0.4
γ_3	Treatment rate from early latent class	0.3
γ_4	Treatment rate from late latent class	0.3
γ_C	Treatment rate from child infected class	0.2
γ_S	Force of infection due to sexual activities	Model parameters
γ_V	Force of infection due to vertical transmission	

Figure 1. Disease dynamics of individuals in different compartments



If treated, late latent individual will join treated latent compartment at rate γ_4, or moves toward tertiary (extreme latent) compartment at rate ψ_2.

A foetus of women from either infected (both primary and secondary) or latent (both early and late) or susceptible compartment is considered as susceptible foetus which enters in model population with either a new child birth as susceptible, which will join susceptible compartment at rate $(1-q)\lambda_V$ or get infection at birth time and join infected child compartment at rate $q\lambda_V$. Infected child either joins treated child compartment at rate γ_C or joins tertiary (extreme latent) compartment at rate ψ_C if not treated.

Treated individuals (infected treated, latent treated, child infected treated) loss immunity and re-join susceptible compartment at rates θ_I, θ_L and θ_C respectively. Natural death is taken in account at rate μ from each compartment. Disease induced deaths are also encountered from tertiary compartment at rate α. Thus, the dynamics of the disease can be expressed by the system of non-linear differential equations as

$$\frac{dS(t)}{dt} = B - \lambda_S S + (1-q)\lambda_V S + \theta_I T_I + \theta_L T_L + \theta_C T_C - \mu S \tag{1}$$

$$\frac{dE(t)}{dt} = \lambda_S S - \sigma E - \mu E \tag{2}$$

$$\frac{dI_1(t)}{dt} = \sigma E - \eta_1 I_1 - \gamma_1 I_1 - \mu I_1 \tag{3}$$

$$\frac{dI_2(t)}{dt} = \eta_1 I_1 - \eta_2 I_2 - \gamma_2 I_2 - \mu I_2 \tag{4}$$

$$\frac{dI_C(t)}{dt} = q\lambda_V S - \psi_C I_C - \gamma_C I_C - \mu I_C \tag{5}$$

$$\frac{dL_1(t)}{dt} = \eta_2 I_2 - \psi_1 L_1 - \gamma_3 L_1 - \mu L_1 \tag{6}$$

$$\frac{dL_2(t)}{dt} = \psi_1 L_1 - \psi_2 L_2 - \gamma_4 L_2 - \mu L_2 \tag{7}$$

$$\frac{dT_I(t)}{dt} = \gamma_1 I_1 + \gamma_2 I_2 - \theta_I T_I - \mu T_I \tag{8}$$

$$\frac{dT_L(t)}{dt} = \gamma_3 L_1 + \gamma_4 L_2 - \theta_L T_L - \mu T_L \tag{9}$$

$$\frac{dT_C(t)}{dt} = \gamma_C I_C - \theta_C I_C - \mu T_C \tag{10}$$

$$\frac{dL_T(t)}{dt} = \psi_2 L_2 + \psi_C I_C - \alpha L_T - \mu L_T \tag{11}$$

with,

$$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + I_C(t) + L_1(t) + L_2(t) + L_T(t) + T_I(t) + T_L(t) + T_C(t) \tag{12}$$

Adding (1) to (12), $\frac{dN(t)}{dt} = B + \lambda_V S - \mu N - \alpha L_T \le B - \mu N$,

Hence $\limsup_{t\to\infty} N(t) \le \frac{B}{\mu}$

The feasible region is

$$\Omega = \left\{ \begin{array}{l} (S,E,I_1,I_2,I_C,L_1,L_2,T_I,T_L,T_C,L_T) \in R_{11}{}^{+} / S+E+I_1+I_2+I_C+L_1+L_2+T_I+T_L+T_C+L_T \le \frac{B}{\mu} \\ \& S>0, E\ge 0, I_1\ge 0, I_2\ge 0, I_C\ge 0, L_1\ge 0, L_2\ge 0, T_I\ge 0, T_L\ge 0, T_C\ge 0, L_T\ge 0 \end{array} \right\}$$

EQUILIBRIUM AND STABILITY

Disease Free Equilibrium (DFE)

DFE of system is given by, $D=(S_0,0,0,0,0,0,0,0,0,0,0)$ where, $S_0 = \frac{B}{\mu}$.

Basic Reproduction Number

Using next generation method suggested by: let $X_0 = (S,E,I_1,I_2,I_C,L_1,L_2,T_I,T_L,T_C,L_T)$,

$$\therefore X_0{}' = \frac{dX_0}{dt} = \Im(X_0) - v(X_0),$$

where,

$$\Im(X) = \begin{pmatrix} (1-q)\lambda_V S \\ \lambda_S S \\ 0 \\ 0 \\ q\lambda_V S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \text{ and } \upsilon(X) = \begin{pmatrix} -B+\lambda_S S-\theta_I T_I-\theta_L T_L-\theta_C T_C+\mu S \\ \sigma E+\mu E \\ -\sigma E+\eta_1 I_1+\gamma_1 I_1+\mu I_1 \\ -\eta_1 I_1+\eta_2 I_2+\gamma_2 I_2+\mu I_2 \\ \psi_C I_C+\gamma_C I_C+\mu I_C \\ -\eta_2 I_2+\psi_1 L_1+\gamma_3 L_1+\mu L_1 \\ -\psi_1 L_1+\psi_2 L_2+\gamma_4 L_2+\mu L_2 \\ -\gamma_1 I_1-\gamma_2 I_2+\theta_I T_I+\mu T_I \\ -\gamma_3 L_1-\gamma_4 L_2+\theta_L T_L+\mu T_L \\ -\gamma_C I_C+\theta_C I_C+\mu T_C \\ -\psi_2 L_2-\psi_C I_C+\alpha L_T+\mu L_T \end{pmatrix}$$

Using, $F = \left[\frac{\partial \Im_i (X_0)}{\partial X_j} \right]$ and

$V = \left[\frac{\partial \upsilon_i (X_0)}{\partial X_j} \right]$ *for* $i, j = 1,2,3,4,5,6,7,8,9,10,11$

Therefore, we have

$$F = \begin{pmatrix} 0 & 0 & \frac{(1-q)\delta P_1 S_0}{N} & \frac{(1-q)\delta P_2 S_0}{N} & \frac{(1-q)\delta P_3 S_0}{N} & \frac{(1-q)\delta P_4 S_0}{N} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \left(\frac{\beta_1}{N} + \frac{\beta_3}{N}\right) S_0 & \left(\frac{\beta_2}{N} + \frac{\beta_4}{N}\right) S_0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{q\delta P_1 S_0}{N} & \frac{q\delta P_2 S_0}{N} & \frac{q\delta P_3 S_0}{N} & \frac{q\delta P_4 S_0}{N} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \mu & 0 & \left(\frac{\beta_1}{N} + \frac{\beta_3}{N}\right) S_0 & \left(\frac{\beta_2}{N} + \frac{\beta_4}{N}\right) S_0 & 0 & 0 & 0 & -\theta_I & -\theta_L & -\theta_C & 0 \\ 0 & A_{10} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\sigma & A_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\eta_1 & A_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & A_5 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\eta_2 & 0 & A_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\psi_1 & A_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma_1 & -\gamma_2 & 0 & 0 & 0 & A_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\gamma_3 & -\gamma_4 & 0 & A_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_C & 0 & 0 & 0 & 0 & A_8 & 0 \\ 0 & 0 & 0 & 0 & -\psi_C & 0 & -\psi_2 & 0 & 0 & 0 & A_9 \end{pmatrix}$$

where,

$$A_1=\eta_1+\gamma_1+\mu,\ A_2=\eta_2+\gamma_2+\mu,\ A_3=\psi_1+\gamma_3+\mu,\ A_4=\psi_2+\gamma_4+\mu, A_5=\psi_C+\gamma_C+\mu,$$
$$A_6=\theta_I+\mu,\ A_7=\theta_L+\mu,\ A_8=\theta_C+\mu,\ A_9=\alpha+\mu, A_{10}=\sigma+\mu$$

The basic reproduction number of system is $R_0=R_H+R_V$, where R_H and R_V are eigenvalues of matrix FV^{-1} evaluated as,

$$R_H=\frac{(\beta_1+\beta_3)\sigma S_0}{N(\sigma+\mu)(\eta_1+\gamma_1+\mu)}+\frac{(\beta_2+\beta_4)\sigma\eta_1 S_0}{N(\sigma+\mu)(\eta_1+\gamma_1+\mu)(\eta_2+\gamma_2+\mu)} \quad \text{and}$$
$$R_V=\frac{q\delta P_3 S_0}{N(\psi_C+\gamma_C+\mu)}.$$

STABILITY AT DFE

Local Stability at Disease Free Equilibrium

Jacobian of the system described by equations (1) to (12) at DFE is,

$$J=\begin{pmatrix}
-\mu & 0 & J_{13} & J_{14} & 0 & J_{16} & J_{17} & \theta_I & \theta_L & \theta_C & 0\\
0 & -A_{10} & J_{23} & J_{24} & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & \sigma & -A_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \eta_1 & -A_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & J_{53} & J_{54} & -A_5 & J_{56} & J_{57} & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \eta_2 & 0 & -A_3 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \psi_1 & -A_4 & 0 & 0 & 0 & 0\\
0 & 0 & \gamma_1 & \gamma_2 & 0 & 0 & 0 & -A_6 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \gamma_3 & \gamma_4 & 0 & -A_7 & 0 & 0\\
0 & 0 & 0 & 0 & \gamma_C & 0 & 0 & 0 & 0 & -A_8 & 0\\
0 & 0 & 0 & 0 & \psi_C & 0 & \psi_2 & 0 & 0 & 0 & -A_9
\end{pmatrix}$$

where,

$$J_{13} = -\left(\frac{\beta_1}{N} + \frac{\beta_3}{N}\right)S_0 + \frac{(1-q)\delta P_1}{N} S_0, J_{14} = -\left(\frac{\beta_2}{N} + \frac{\beta_4}{N}\right)S_0 + \frac{(1-q)\delta P_2}{N} S_0,$$

$$J_{16} = \frac{(1-q)\delta P_3}{N} S_0,\ J_{17} = \frac{(1-q)\delta P_4}{N} S_0, J_{23} = -\left(\frac{\beta_1}{N} + \frac{\beta_3}{N}\right)S_0, J_{24} = -\left(\frac{\beta_2}{N} + \frac{\beta_4}{N}\right)S_0,$$

$$J_{53} = \frac{q\delta P_1}{N} S_0, J_{54} = \frac{q\delta P_2}{N} S_0,\ J_{56} = \frac{q\delta P_3}{N} S_0,\ J_{57} = \frac{q\delta P_4}{N} S_0$$

As $trace(J) = -\mu - \sum_{i=1}^{10} A_i < 0$ and Principal minors $M_1, M_2, \ldots, M_{10}$ are alternately negative and positive respectively with largest order principal minor $M_{11} = \det(J) = \mu(R_H - 1)\prod_{i=1}^{10} A_i$. Thus M_{11}<0 if and only if R_H<1.

Hence, by Routh- Hurwitz's criterion DFE is locally stable if and only if R_H<1.

Global Asymptotic Stability at Disease Free Equilibrium

Let X=(S) and Z=(E,I_1,I_2,I_C). At DFE, X^0=(S_0) and Z^0=(0,0,0,0,0).

Then, $\frac{dX}{dt}\left(=\frac{dS}{dt}\right) = F(X,Z) = B - \mu S$.

Hence, at Z=Z^0 and as $X \to X_0$, $\frac{dX}{dt} \to F(X_0, 0) = B - \mu S_0 = 0$.

Thus, X=X^0 is globally asymptotically stable.

Fromequations(2)to(5), $\frac{dZ}{dt}\left(=\frac{d(E, I_1, I_2, I_C)}{dt}\right) = G(X,Z) = HZ - \hat{G}(X,Z)$

Where M is the Jacobian matrix for the system (2) to (5) at DFE which can be given by

$$H = \begin{pmatrix} -\sigma - \mu & \frac{\beta_1 + \beta_3}{N} & \frac{\beta_2 + \beta_4}{N} & 0 \\ \sigma & -\eta_1 - \gamma_1 - \mu & 0 & 0 \\ 0 & \eta_1 & -\eta_2 - \gamma_2 - \mu & 0 \\ 0 & \frac{q\delta P_1}{N} & \frac{q\delta P_2}{N} & -\psi_C - \gamma_C - \mu \end{pmatrix}$$

and

$$\hat{G}(X,Z)=\begin{pmatrix} \frac{\beta_1+\beta_2+\beta_3+\beta_4}{N}(S-1) \\ 0 \\ 0 \\ 0 \\ \frac{q\delta P_1+q\delta P_2}{N}(S-1) \end{pmatrix}$$

As H is M- matrix (having all off diagonal entries non negative) and $G(X,Z)\geq 0$ in Ω, conditions (H_1) and (H_2) suggested by Castillo-Chavez *et al*., 2002 are satisfied and hence DFE is globally asymptotically stable if $R_H<1$.

Endemic Equilibrium (EE) and its Stability

Endemic Equilibrium

Solving,

$$\left(\frac{dS(t)}{dt}\right)=0,\left(\frac{dE(t)}{dt}\right)=0,\left(\frac{dI_1(t)}{dt}\right)=0,\left(\frac{dI_2(t)}{dt}\right)=0,\left(\frac{dI_C(t)}{dt}\right)=0,$$

$$\left(\frac{dL_1(t)}{dt}\right)=0,\left(\frac{dL_2(t)}{dt}\right)=0,\left(\frac{dT_I(t)}{dt}\right)=0,\left(\frac{dT_L(t)}{dt}\right)=0,\left(\frac{dT_C(t)}{dt}\right)=0$$

and $\left(\frac{dL_T(t)}{dt}\right)=0,$ simultaneously.

Endemic equilibrium point is

$$S^*=\frac{S_0}{R_H},\ I_1^*=b_1E^*,\ I_2^*=b_2E^*,I_C^*=b_3E^*,\ L_1^*=b_4E^*,L_2^*=b_5E^*,T_I^*=b_6E^*,T_L^*=b_7E^*,$$

$$T_C^*=b_8I_C^*,L_T^*=b_9E^*$$

where,

$$b_1=\left(\frac{\sigma}{A_1}\right),\ b_2=\left(\frac{b_1\eta_1}{A_2}\right),\ b_3=\left(\frac{q\delta(b_1P_1+b_2P_2+b_3P_4+b_4P_5)S_0}{NA_5R_H}\right),b_4=\left(\frac{b_2\eta_2}{A_3}\right),b_5=\left(\frac{b_4\psi_1}{A_4}\right),$$
$$b_6=\left(\frac{b_1\gamma_1+b_2\gamma_2}{A_6}\right),b_7=\left(\frac{b_4\gamma_3+b_5\gamma_4}{A_7}\right),b_8=\left(\frac{b_3\gamma_C}{A_8}\right),b_9=\left(\frac{b_5\psi_2+b_3\psi_C}{A_9}\right)$$

Finally, one can get value of E^*as

$$E^*=\left(\begin{array}{c}\dfrac{BA_1A_2A_3A_4A_5A_6A_7A_8N\left(R_H-1\right)}{A_1A_2A_3A_4A_5A_6A_7A_8A_{10}NR_H-\sigma(\theta_C\gamma_CA_5A_8NR_H(\theta_IA_3A_4A_7(\gamma_1A_2+\gamma_2\eta_1)+\theta_LA_6\eta_1\eta_2(\gamma_3A_3+\gamma_4\psi_1)))}\\ -\sigma(\delta S_0A_6A_7(A_5A_8(1-q)+\theta_C\gamma_Cq)(P_1A_2A_3A_4+P_2\eta_1A_3A_4+P_3\eta_1\eta_2A_4+P_4\eta_1\eta_2\psi_1))\end{array}\right)$$

which suggests existence of endemic equilibrium for $R_0>R_H>1$.

Global Asymptotic Stability at Endemic Equilibrium

Consider, Lyapunov function $V:R_+^{11}\to R$, defined as

$$V\left(x\right)=\left[S-S^*-S^*\ln\left(\frac{S}{S^*}\right)\right]+\left[E-E^*-E^*\ln\left(\frac{E}{E^*}\right)\right]+\left[I_1-I_1^*-I_1^*\ln\left(\frac{I_1}{I_1^*}\right)\right]$$
$$+\left[I_2-I_2^*-I_2^*\ln\left(\frac{I_2}{I_2^*}\right)\right]+\left[I_C-I_C^*-I_C^*\ln\left(\frac{I_C}{I_C^*}\right)\right]+\left[T_1-T_1^*-T_1^*\ln\left(\frac{T_1}{T_1^*}\right)\right]$$
$$+\left[T_2-T_2^*-T_2^*\ln\left(\frac{T_2}{T_2^*}\right)\right]+\left[T_C-T_C^*-T_C^*\ln\left(\frac{T_C}{T_C^*}\right)\right]+\left[L_1-L_1^*-L_1^*\ln\left(\frac{L_1}{L_1^*}\right)\right]$$
$$+\left[L_2-L_2^*-L_2^*\ln\left(\frac{L_2}{L_2^*}\right)\right]+\left[L_T-L_T^*-L_T^*\ln\left(\frac{L_T}{L_T^*}\right)\right]$$

where, $x=(S,E,I_1,I_2,I_C,T_1,T_2,T_C,L_1,L_2,L_T)$

Here $V(x)>0$ for $\forall x\in\Omega-\{0\}$, $V(0)=0$ and

$$\frac{dV}{dx}<S_0\left(1-\frac{1}{R_H}\right)+\left(\frac{\delta\left(P_1b_1+P_2b_2+P_3b_4+P_4b_5\right)S_0-\mu NR_H\left(b_1+b_2+b_4+b_5+b_6+b_7+b_8+b_9\right)}{NR_H}\right)E^*\cdot$$

Hence, $\frac{dV}{dx} < 0$ if $R_H > 1$ and $\frac{dV}{dx}(0) = 0$.

Thus, stability criteria of Lyapunov (1992), guarantees that endemic equilibrium is stable when $R_0 > R_H > 1$.

OPTIMAL CONTROL

An optimal control model for a syphilis disease in order to derive optimal treatment for individuals from infected child class and late latent class with minimal implementation cost is formulated.

Defining control variables as $\mu_1(t)=\gamma_1$, $\mu_2(t)=\gamma_2$, $\mu_3(t)=\gamma_3$, $\mu_4(t)=\gamma_4$, and $\mu_5(t)=\gamma_C$, for the developed model, given by equations (1) to (11) and considering the feasible region same as given by Ω.

Consider the following cost-functional.

$$J(u_1,u_2,u_3,u_4,u_5)=\int_0^T \begin{pmatrix} w_{21}(S)^2 + w_{22}(E)^2 + w_{23}(I_1)^2 + w_{24}(I_2)^2 + w_{25}(I_C)^2 + w_{26}(L_1)^2 \\ +w_{27}(L_2)^2 + w_{28}(T_I)^2 + w_{29}(T_2)^2 + w_{19}(T_C)^2 + w_{20}(L_T)^2 \\ +w_{11}(u_1(t))^2 + w_{12}(u_2(t))^2 + w_{13}(u_3(t))^2 + w_{14}(u_4(t))^2 + w_{15}(u_5(t))^2 \end{pmatrix} dt$$

where, $x=(S,E,I_1,I_2,I_C,T_1,T_2,T_C,L_1,L_2,L_T)$, $u=(u_1,u_2,u_3,u_4,u_5)$ and w_{ij} are weights to regularise the optimal control.

Optimal control task reads as $\min_u J(u_1,u_2,u_3,u_4,u_5)$ such that $P(x,u)=0$. where $P(x,u)=0$ denotes the system of equations.

Defining the Hamiltonian H for the control problem as,

$$\begin{aligned} H(x,u,\lambda) = {} & w_{21}(S)^2 + w_{22}(E)^2 + w_{23}(I_1)^2 + w_{24}(I_2)^2 + w_{25}(I_C)^2 + w_{26}(L_1)^2 + w_{27}(L_2)^2 \\ & + w_{28}(T_I)^2 + w_{29}(T_2)^2 + w_{19}(T_C)^2 + w_{20}(L_T)^2 + w_{11}(u_1(t))^2 + w_{12}(u_2(t))^2 + w_{13}(u_3(t))^2 \\ & + w_{14}(u_4(t))^2 + w_{15}(u_5(t))^2 + \lambda_1 \begin{pmatrix} B-\lambda_S S+(1-q)\lambda_V S+\theta_I T_I \\ +\theta_L T_L+\theta_C T_C-\mu S \end{pmatrix} + \lambda_2 \begin{pmatrix} \lambda_S S-\sigma E \\ -\mu E \end{pmatrix} \\ & + \lambda_3 \begin{pmatrix} \sigma E-\eta_1 I_1 \\ -u_1 I_1-\mu I_1 \end{pmatrix} + \lambda_4 \begin{pmatrix} \eta_1 I_1-\eta_2 I_2 \\ -u_2 I_2-\mu I_2 \end{pmatrix} + \lambda_5 \begin{pmatrix} q\lambda_V S-\psi_C I_C \\ -u_5 I_C-\mu I_C \end{pmatrix} + \lambda_6 \begin{pmatrix} \eta_2 I_2-\psi_1 L_1 \\ -u_3 L_1-\mu L_1 \end{pmatrix} \\ & + \lambda_7 \begin{pmatrix} \psi_1 L_1-\psi_2 L_2 \\ -u_4 L_2-\mu L_2 \end{pmatrix} + \lambda_8 \begin{pmatrix} u_1 I_1+u_2 I_2 \\ -\theta_I T_I-\mu T_I \end{pmatrix} + \lambda_9 \begin{pmatrix} u_3 L_1+u_4 L_2 \\ -\theta_L T_L-\mu T_L \end{pmatrix} + \lambda_{10} \begin{pmatrix} u_5 I_C-\theta_C I_C \\ -\mu T_C \end{pmatrix} \\ & + \lambda_{11} \begin{pmatrix} \psi_2 L_2+\psi_C I_C \\ -\alpha L_T-\mu L_T \end{pmatrix} \end{aligned}$$

Using Pontrayagin's maximum (minimum) principle,

Let $(u^*_1, u^*_2, u^*_3, u^*_4, u^*_5)$ be optimal solution of an optimal control problem then there exists a nontrivial vector function

$$\lambda(t) = \left(\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t), \lambda_5(t), \lambda_6(t), \lambda_7(t), \lambda_8(t), \lambda_9(t), \lambda_{10}(t), \lambda_{11}(t)\right)$$

satisfying following equations.

(1) The state equation $\dfrac{dx}{dt} = \dfrac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, \lambda(t))}{\partial \lambda}$

(2) The optimality condition $0 = \dfrac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, \lambda(t))}{\partial u}$

(3) The adjoint equation $\dfrac{d\lambda}{dt} = -\dfrac{\partial H(t, u_1^*, u_2^*, u_3^*, u_4^*, u_5^*, \lambda(t))}{\partial x}$.

Solving optimality conditions for optimal control and the property of control space give,

$$u_1^*(t) = \max\left\{\min\left\{1, \frac{1}{2w_{11}}\left[\left(\lambda_3 - \lambda_8\right) I_1\right]\right\}, 0\right\},$$

$$u_2^*(t) = \max\left\{\min\left\{1, \frac{1}{2w_{12}}\left[\left(\lambda_4 - \lambda_8\right) I_2\right]\right\}, 0\right\},$$

$$u_3^*(t) = \max\left\{\min\left\{1, \frac{1}{2w_{13}}\left[\left(\lambda_6 - \lambda_9\right) L_1\right]\right\}, 0\right\},$$

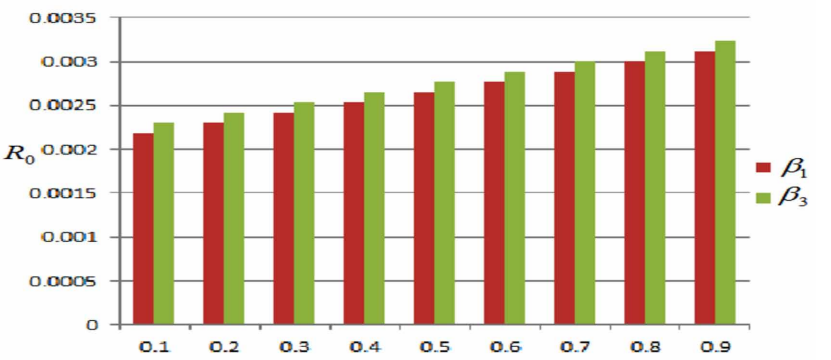
$$u_4^*(t) = \max\left\{\min\left\{1, \frac{1}{2w_{14}}\left[\left(\lambda_7 - \lambda_9\right) L_2\right]\right\}, 0\right\},$$

$$u_5^*(t) = \max\left\{\min\left\{1, \frac{1}{2w_{15}}\left[\left(\lambda_5 - \lambda_{10}\right) I_C\right]\right\}, 0\right\}.$$

Numerical Simulation and Observations

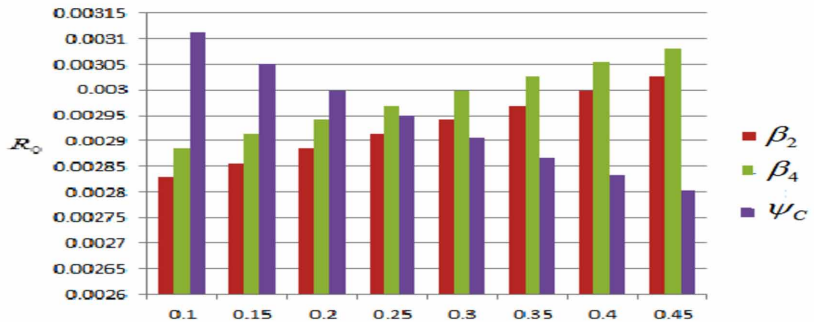
Numerical simulation is carried out with the parametric values given in Table 1. Sensitivity of R_0 with different disease parameters is verified to understand disease spread possibilities.

Figure 2. Effect of change in contact rates β_1 and β_3 on R_0



In figure 2, the effects of change in contact rates β_1 and β_3 on R_0 are shown. It is observed that increase in the heterosexual contact rates will also increase R_0.

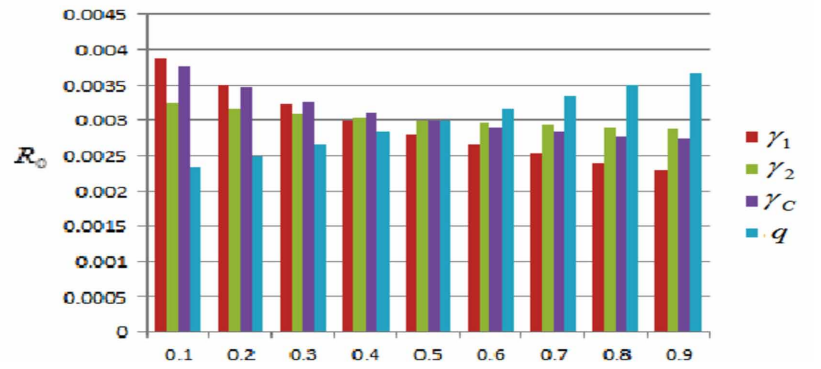
Figure 3. Effect of change in contact rates β_2, β_4 and rate of infected children joining late latent classs ψ_C on R_0



In figure 3, the effects of change contact rates β_2, β_4 and rate ψ_C of infected children joining latent classs on R_0 are described. Comparing figure 2 and figure 3, it can be observed that MSM activities spread disease faster than heterosexual

activities. From figure 3 it can be observed that increase in ψ_C controls the disease as individuals moves to tertiary class which involves disease induced deaths.

Figure 4. Effect of change in γ_1, γ_2, γ_C and q on R_0



In figure 4, the effects of changes in parameters γ_1, γ_2, γ_C and q on R_0 are plotted. It can be observed that increase in probability of a new born child having disease at birth time makes system unstable. It also indicates that increment in various treatment rates, with the strategy of keeping treatment rate of infected child higher than other treatment rates helps to control disease spread.

Figure 5. Effect of change in η_1, η_2 and σ on R_0

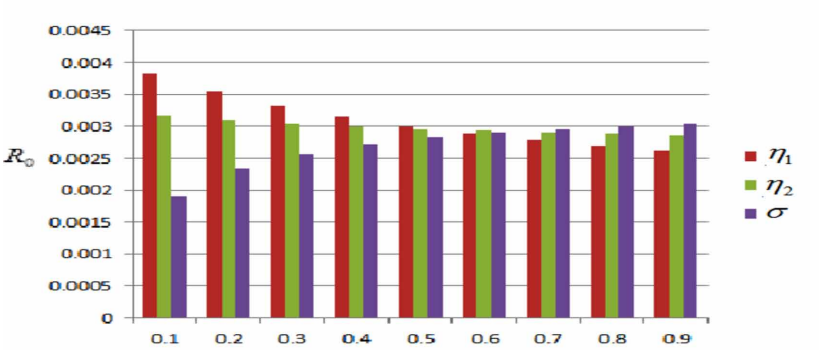
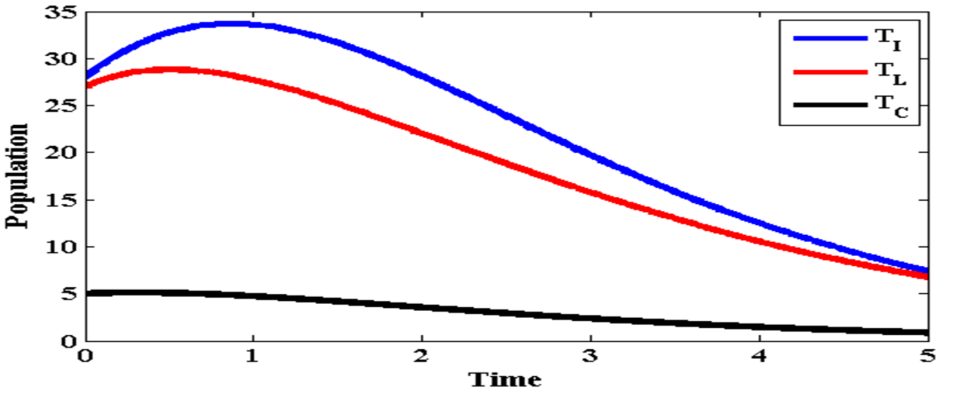


Figure 5, indicates the effects of changes in parameters η_1, η_2 and σ on R_0. It can be visualised that increment in rate of individual's movement towards primary infected compartment increases R_0, while movement of individual toward secondary

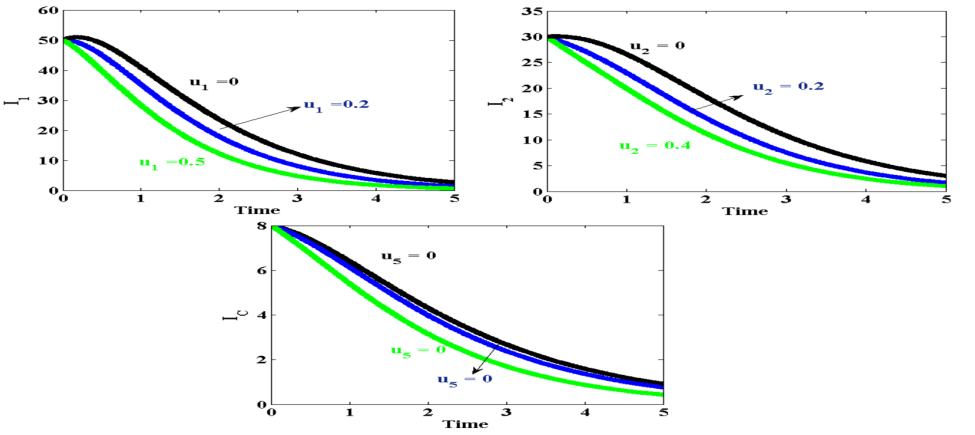
infected compartment and early latent compartment keeps system stable due to high treatment applied in initial stage.

Figure 6. Population dynamics of treated compartments



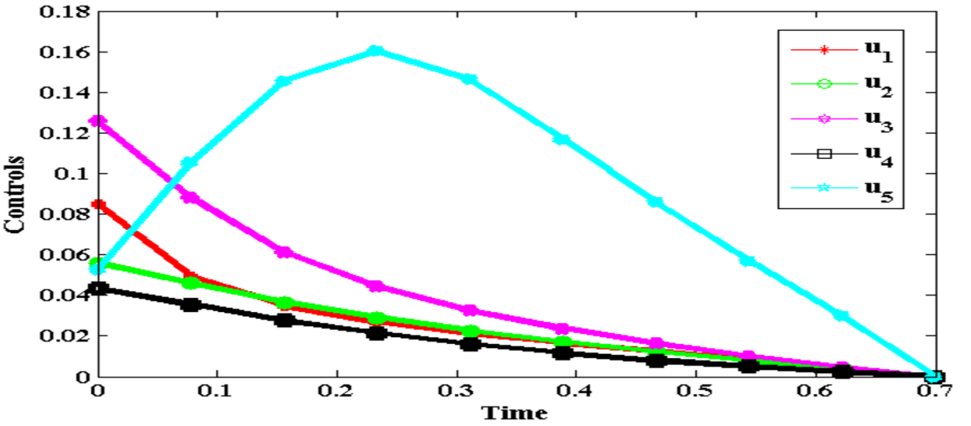
In figure 6, population dynamics of treated class is shown. It can be noted that initial high treatment increases treated class population and then after they moves towards stability.

Figure 7. Effects of control treatments on respective infected compartments



From figure 7, it can be visualised that treatment at proper rate applied on time would stop disease spread effectively.

Figure 8. Controls



To minimise the total effective cost to control disease spray by applying control treatments, the policy is to be designed in a way that initially, 6% treatment should be applied to infected children with increased up to 16% till third week of infection along with 9% treatment to primary infected, 5% to secondary infected, 13% to early latent and 4% to late latent individuals.

CONCLUSION

In this chapter, vertical transmission of Syphilis with control treatment is studied. Mathematical model using system of non linear differential equations is suggested. The basic reproduction number is calculated using the next generation matrix method. Stability at the equilibrium states for model parameters along with numerical simulation is carried out. Results suggests that treatment given at earlier stage of infection is effective way to get cured faster. Treatment of infected child at birth is essential and should be applied immediaetly after birth. Safe sexual activities should be adopted to control disease spread.

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Chapter 12
Mathematical Model to Analyze Effect of Demonetization

Nita H. Shah
https://orcid.org/0000-0003-1605-4778
Department of Mathematics, Gujarat University, Ahmedabad, India

Bijal M. Yeolekar
Department of Mathematics, Gujarat University, Ahmedabad, India

Zalak Ashvinkumar Patel
L. D. College of Engineering, India

ABSTRACT

Demonetization is a fundamental regulatory act of stripping in which a currency unit's status as an exchange is professed worthless. Generally, it is done whenever there is a change of national currency, often to be replaced of the old notes or coins with a new one. Sometimes, a country totally replaces the old currency with new currency. For example, in India recently the government demonetized RS. 500 and 1000 notes. So, one has to deposit their cash within limited time in the banks. The demonetization affects individuals mildly or potentially, which in turn affects banking sector. So, SMPB-model is proposed and analyzed for demonetization. The SMP-model is formulated with the system of nonlinear differential equations. The effect of demonetization is studied by calculating threshold using next generation matrix. The local and global stability for demonetization free equilibrium and demonetization equilibrium is worked out. The existence of the equilibrium is investigated. The model is validated with numerical simulation.

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INTRODUCTION

Demonetization is the act of banning a specific form of currency notes from circulation by the government. Due to demonetization, a currency is blocked or the old currencies changed by the new currency. There are several reasons for demonetizing currency for nations: some reasons include combating the inflation, to control the corruption and crime, to discourage a cash-dependent and to promote the cashless economy for getting transparency in all the modes of legal transaction in the country.

In India, the first time in 1946 that the government demonetized of high value currency notes of Rs. 1000 and Rs. 10000 and then introduced higher quantity bank note of Rs. 1000, Rs.5000 and Rs. 10000 currency notes in 1954. After that in 1978, Prime Minister Shri Morarji Desai demonetized these high value notes. After 38 years again demonetization of currency note bans of Rs. 500 and Rs. 1000 in November 8, 2016 and this time old currency of Rs. 500 notes were replaced with new designed and Rs. 1000 notes vanished and the first time introduced Rs. 2000 value note. The objective of the demonetization was to curb the disease of corruption and black money and curtail the circulation of fake notes in the system.

Demonetization is the interruption of current currency and replaces the old currency units with new currency units. It is a significant decision and it disappointed all the individuals as unexpectedly all the money individuals have become a piece of paper. The old money has no value if one cannot exchange it with new currency units or deposited it in the banks within a given time by the government and use maximum the online services at that time. Online banking mentioned as using internet for different banking amenities extending from bill payments to savings [Pikkarainen *et al.* (2014)]. Previously banks used to deliver information for their products on respective bank websites and with time they have given chance to the clients for making monetary transaction like bill payments and money transfers etc. [Chong *et al.* (2010)]. Online banking is classification into an online account with direct money transfer in the account [Chiles (2013)]. From mid ninety's internet banking had started in India. ICICI was implemented internet banking in 1998 [Kesharwani and Sainti (2012)]. The several reasons like compatibility, importance, involvedness, ability, apparent risk and were measured customer attitude towards internet banking [Ndubisi *et al.* (2006)]. For understanding internet banking usage Yoon and Steege (2013) observed some features like usability, personality, security and social stimulus of internet banking which are beneficial for banks as well as customers and also it made possible cost savings and ease. Internet banking adoption professed some factors like usefulness, ease of use, social influence and self-efficacy have an influence on customer's attitude in urban area [Sharma and Govindaluri (2014)]. According to survey of Internet and Mobile Association of India (IAMAI), the safety apprehensions and less knowledge about online transactions are the main

blocks to adopt online or internet banking. Rathore (2016) studied that digital wallets are speedily becoming conventional approach of e-payment. Sellers and buyers both are accepting digital wallets at rapid pace, because of its handiness and ease of usability. These all suggested studying the demonetization effect and net banking.

In this chapter, the effect of demonetization in India using digital payment method is explored with mathematical formulation. For the proposed model, notations and parametric values with schematic diagram of the transmission is formulated. The nonlinear differential equations for demonetization model are formulated in with the existence of the solution of system and boundness of the solution of the system. The calculation of the basic reproduction number at the demonetization free equilibrium point is calculated using the next generation matrix. We discussed the stability of the system, and prove the local stability and global stability. The demonetization model is validated through parametric values. This chapter ends up with the conclusion.

NOTATIONS

In this chapter, we formulate mathematical model for the individuals who are affected by demonetization. The model is formulated using the notations and parametric values are given in the Table1 as follows. The following notations and parametric values in table 1 are taken into consideration.

Using these notations, we formulate a transfer diagram for demonetization effect in following figure 1.

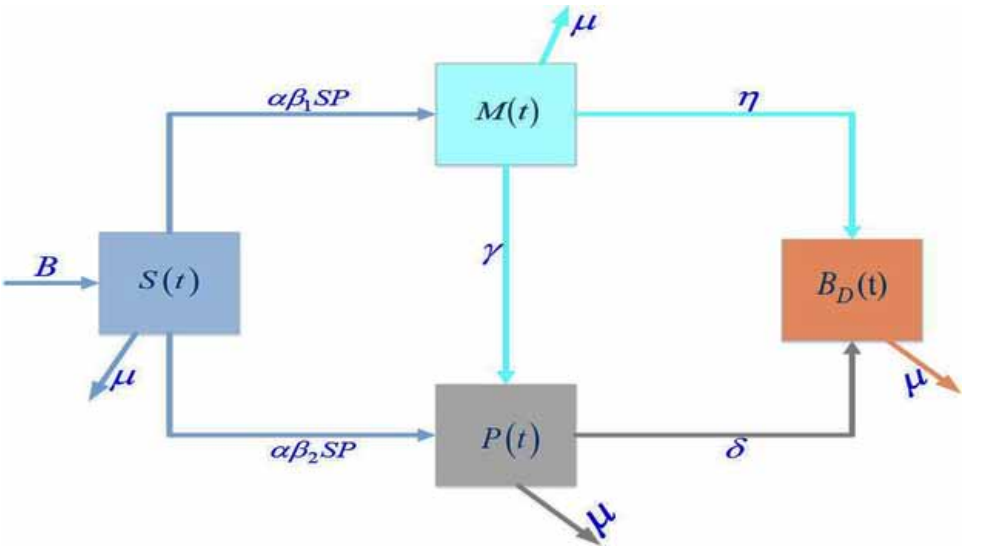
MATHEMATICAL MODEL

The nonlinear mathematical model for demonetization effect is formulated. In this model, total population is divided in to four compartments viz. the class of susceptible who are not yet suffering from demonetization $S(t)$, if any currency ban or demonetization is declared than some individuals are mildly affected that comes in $M(t)$ class and some of them are potentially affected that are potentially affected class $P(t)$ and these individuals who tries to deposit their liquid amount in banks that are considered as a bank depositor class $B_D(t)$. The total population at time t denoted by $N(t)$. Based on the abovementioned modeling assumptions, the proposed compartmental deterministic demonetization system is governed by the following system of ordinary non-linear differential equations from the schematic diagram given in Figure1.

Table 1. Notations and Values of parameters

Parameter		Value
$S(t)$	Number of individuals who may have cash of old notes after declaration of demonetization	12
$M(t)$	Number of individuals who are mildly affected with demonetization	5
$P(t)$	Number of individuals who are potentially (highly) affected with demonetization	3
$B_D(t)$	Number of individuals who had deposited their old cash in bank	6
B	New recruitment rate	10
μ	Rate at which individuals opts for digital payment	0.6
α	Contact rate of potentially affected individual with susceptible per unit time	2
β_1	Fraction of susceptible individuals in mildly affected compartment	0.03
β_2	Fraction of susceptible individuals in potentially affected compartment	0.02
γ	Rate at which mildly affected individuals moves to potentially affected individual	0.02
δ	Potential individuals deposit their old cash in bank	0.8
η	Mildly affected individuals deposited their old cash in bank	0.7

Figure 1. Transfer diagram of demonetization effects on individuals



$$\frac{dS(t)}{dt} = B - \alpha SP - \mu S$$

$$\frac{dM(t)}{dt} = \alpha\beta_1 SP - \gamma M - \eta M - \mu M \tag{1}$$

$$\frac{dP(t)}{dt} = \alpha\beta_2 SP + \gamma M - \delta P - \mu P$$

$$\frac{dB_D(t)}{dt} = \eta M + \delta P - \mu B_D$$

with initial conditions,

$$S(0) = S_0 > 0,\ \mathrm{M}(0) = M_0 \geq 0,\ \mathrm{P}(0) = P_0 \geq 0,\ \mathrm{B}_D(0) = B_{D0} \geq 0,$$

and the total population is, $N(t) = S(t)+M(t)+P(t)+B_D(t)$.

Adding all the equations of system (1), we have

$\frac{dN}{dt} \leq B - \mu\left(S + M + P + B_D\right)$ which gives, $\frac{dN}{dt} \leq B - \mu N$.

So, we have $\lim_{t\to\infty} sup\, N \leq \frac{B}{\mu}$.

So, we consider the feasible solution inside the region of the system is

$$\Omega = \left\{ \begin{array}{c} (S, M, P, B_D) : S + M + P + B_D \leq \frac{B}{\mu}, 0 < S \leq S(t), \\ 0 \leq M \leq M(t), 0 \leq P \leq P(t), 0 \leq M \leq M(t) \end{array} \right\} \tag{2}$$

The existence and uniqueness of solutions and in continuation results hold in the system (1). We study the system and privilege that the region Ω is bounded and positively invariant with respect to the proposed system for effects of demonetization.

Positivity of Solution of System of Demonetization

Theorem 1 Given

$$S(0) = S_0 > 0, \mathrm{M}(0) = M_0 > 0, \mathrm{P}(0) = P_0 > 0, \mathrm{B}_D(0) = B_{D0} > 0,$$

the solutions (S,M,P,B_D) of the system are positively invariant for all $t>0$.

Proof Let $Z_1 = \sup\{t > 0 : S > 0, \mathrm{M} > 0, \mathrm{P} > 0, \mathrm{B}_D > 0\}$, for the first equation,

$$\frac{dS}{dt} = B - \alpha SP - \mu S = B - (\alpha P + \mu) S \tag{3}$$

The integrating factor (I.F.) is $e^{\int_0^t (\alpha P + \mu) dS}$. Multiply integrating factor with equation (3) and we have

$$\frac{dS(t)}{dt}\left[e^{\int_0^t (\alpha PSds) + \mu t} \right] \geq Be^{\int_0^t (\alpha PSds) + \mu t}.$$

Now, solving the inequality and we get

$$S(t) e^{\int_0^t (\alpha PSdS) + \mu t} - S(0) \geq \int_0^t \left[Be^{\int_0^t (\alpha PSdS) + \mu t} \right] dk.$$

Therefore, $S(t)$ becomes

$$S(t) \geq S(0) e^{\int_0^t (\alpha PSdS) + \mu t} + e^{-\int_0^t (\alpha PSdS) + \mu t} \times \int_0^t \left[Be^{\int_0^t (\alpha PSdS) + \mu t} \right] dk > 0.$$

Hence, we proved that $S(t)>0$. Similarly, we can prove for all the other compartments respectively. This theorem is very important to founds that population size cannot be negative.

Boundness of The Solution of System

Theorem 2 All solutions ($S(t),M(t),P(t),B_D(t)$) of the system of demonetization effects are bounded.

Proof System refers that the total population size suffers from demonetization of the model equation,

we obtain

$$\frac{dN}{dt} = B - \mu\left(S + M + P + B_D\right) \le B - \mu N \text{ . Then } \lim_{t\to\infty} sup\, N \le \frac{B}{\mu}. \tag{4}$$

Therefore, all solution of model (1) are bounded. The feasible region for the total population Ω in equation (2).

We defined Ω as the positively invariant region with respect to the model equation (3), therefore for any initial point $\left(S_0 > 0, M > 0, P > 0, B_D > 0\right) \in R_4^+$, the trajectories lies in the feasible region Ω and thus the model equation (3) is mathematically and epidemiologically well posed in Ω. Let Ω denote the interior of Ω.

Theorem 3 The region $\Omega \subset R_+^4$ is positively invariant for the model (1) with multiple involvement of effects of demonetization with non-negative initial condition in R_+^4.

Basic Reproduction Number

In this section, we calculate Basic reproduction number of system (1) using with the next generation matrix method at demonetization free equilibrium $M=P=B_D=0$.

Hence, let $X_0 = \left(\frac{B}{\mu}, 0, 0, 0\right)$ be demonetization free equilibrium point of system.

Using next generation method, let $X' = \left(M, P, B_D, S\right)'$, where dash denotes derivative where,

$$\Im = \begin{bmatrix} \alpha\beta_1 SP \\ \alpha\beta_2 SP \\ 0 \\ 0 \end{bmatrix} \text{and } \upsilon = \begin{bmatrix} (\gamma+\eta+\mu)\,\mathrm{M} \\ -\gamma M + (\mu+\delta)\,\mathrm{P} \\ -\eta M - \delta P + \mu B_D \\ -B + \alpha SP + \mu P \end{bmatrix}$$

Using,

$$F = \left[\frac{\partial \Im_i(X_0)}{\partial X_j}\right] \text{ and } V = \left[\frac{\partial \upsilon_i(X_0)}{\partial X_j}\right] \quad for\ i, j = 1,2,3,4$$

Therefore, we have

$$F = \begin{bmatrix} 0 & \frac{\alpha\beta_1 B}{\mu} & 0 & 0 \\ 0 & \frac{\alpha\beta_1 B}{\mu} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{and} V = \begin{bmatrix} A & 0 & 0 & 0 \\ -\gamma & \mu+\delta & 0 & 0 \\ -\eta & -\delta & \mu & 0 \\ 0 & \frac{\alpha B}{\mu} & 0 & \mu \end{bmatrix}, \text{ where } A=\gamma+\eta+\mu. \tag{5}$$

The basic reproduction number R_0 is spectral radius of matrix FV^{-1} which is threshold of the system (1).

$$R_0 = \frac{\alpha B(\beta_1\gamma + \beta_2\,\mathrm{A})}{A\mu(\mu+\delta)}. \tag{6}$$

The basic reproduction number or threshold is computed by simply imposing the non-negativity condition on the demonetization affected individual compartment. The basic reproduction number is the average number of secondary contagions produced when one single affected individual is presented into a host population where everyone is susceptible (vander *et al*. (2002), Heffernan (2005),Hethcote (2000)). Here, the basic reproduction number mainly depends on the potentially affected individual with susceptible, recruitment rate, susceptible individuals in mildly affected, susceptible individuals in potentially affected, mildly affected individuals moves to potentially affected individual and potential individuals deposit their old cash in bank.

EQUILIBRIA OF THE SYSTEM (1)

In this section, we discuss the positive (demonetization exist) equilibrium $X^*=(S^*,M^*,P^*,B_D{}^*)$ of the system (1) by putting zero at the right-hand side of the equations (1) to (4), we have

$$S^* = \frac{B}{\alpha P^* + \mu} \tag{7}$$

$$M^* = \frac{\alpha\beta_1 S^* P^*}{A} \tag{8}$$

$$B_D{}^* = \frac{\eta M^* + \delta P^*}{\mu} \tag{9}$$

where the value of A is in equation (5). Putting (7) and (8) in equation (3) of system (1), we obtain nontrivial solution

$$P^* = \frac{\alpha\beta\left(A\beta_2 + \gamma\beta_1\right) - A\mu\left(\mu+\delta\right)}{\alpha A\left(\mu+\delta\right)} = \frac{\alpha\beta\left(A\beta_2 + \gamma\beta_1\right)}{\alpha A\left(\mu+\delta\right)} - \frac{\mu}{\alpha} \tag{10}$$

Thus, $P^* = -\frac{\mu}{\alpha}\left(1-R_0\right)$.

Theorem 4 The system (1) has two equilibrium points Demonetizations free equilibrium (DFE) $X_0 = \left(\frac{B}{\mu}, 0, 0, 0\right)$ is always exist and Demonetization exist equilibrium (DEE) $X^*=(S^*,M^*,P^*,B_D{}^*)$ does not exist if $R_0<1$.

STABILITY ANALYSIS

In this section, we discuss the local stability and global stability of the system at the demonetization free equilibrium points X_0 and demonetization exist equilibrium points X^*.

Local Stability

Here, we investigate the local stability of the Demonetization Free Equilibrium point X_0 and Demonetization Exist Equilibrium point X^*.

Theorem 5 (Local stability at X_0) The demonetization free equilibrium point X_0 is locally asymptotically stable if $R_0<1$, X_0 locally stable if $R_0=1X_0$ and unstable if $R_0>1$.

Proof Linearizing system (1) by Linearized method (Perko, (2013)) and by using matrix analysis (Anton (2010)), we obtained the Jacobian matrix at demonetization free equilibrium points X_0 is as

$$J=\begin{bmatrix} -\mu & 0 & -\frac{\alpha\beta}{\mu} & 0 \\ 0 & -\mu-\eta-\gamma & \frac{\alpha\beta_1 B}{\mu} & 0 \\ 0 & \gamma & \frac{\alpha\beta_2 B}{\mu}-\mu-\delta & 0 \\ 0 & \eta & \delta & -\mu \end{bmatrix} \tag{11}$$

Clearly the two eigenvalues of the Jacobian matrix are $\lambda_{1,2}=-\mu$ and the remaining characteristic equation is

$$\lambda^2+a_1\lambda+a_2=0 \tag{12}$$

where $a_1=\eta+\gamma-\frac{\alpha\beta_2 B}{\mu}+2\mu+\delta)$ and

$$a_2=(\eta+\gamma+\mu)\left(\frac{\alpha\beta_2 B}{\mu}-\mu-\delta\right)-\frac{\alpha\gamma\beta_1 B}{\mu}.$$

Now, from Routh -Hurwitz Criteria (Edelstein- Keshet (2005)), Jacobian in (11) of the system has two negative eigenvalues and the remaining eigenvalues from equation (12) are negative if $a_1>0$ and $a_2>0$.

If $R_0<1$, then

$$\alpha B(\beta_1\gamma + \beta_2 \mathrm{A}) < A\mu(\mu+\delta) \Rightarrow \alpha B\beta_2 \mathrm{A} < A\mu(\mu+\delta)$$

$$\Rightarrow \frac{\alpha B\beta_2}{\mu} < (\mu+\delta) < \eta + \gamma + 2\mu + \delta = a_1 > 0.$$

For a_2, we have

$$a_2 = -A\left(\frac{\alpha\beta_2 B}{\mu} - \mu - \delta\right) - \frac{\alpha\gamma\beta_1 B}{\mu}$$

$$= A(\mu+\delta)\left(1 - \frac{\alpha A\beta_2 B + \alpha\gamma\beta_1 B}{A\mu(\mu+\delta)}\right) = A(\mu+\delta)(1-R_0)$$

So, if $R_0<1$ then only $a_2>0$. Therefore, we say that the Jacobian matrix has all the eigenvalues with negative real part if $R_0<1$. Hence X_0 is locally asymptotically stable if $R_0<1$. Now, for $R_0=1$, $a_1>0$ and $a_2=0$ which shows X_0 is locally stable. If $R_0>1$ then $a_2<0$, then X_0 is unstable.

Theorem 6 (Local stability at X^***)** The demonetization existence equilibrium point (DEE) X^* is locally asymptotically stable if $R_0<1$.

Proof Linearizing system (1) by linearized method (Perko, (2013)) and by using matrix analysis (Anton (2010)), we obtained the Jacobian matrix at X^* is as

$$J = \begin{bmatrix} -\mu - \alpha P^* & 0 & -\alpha S^* & 0 \\ \alpha\beta_1 P^* & -\mu - \eta - \gamma & \alpha\beta_1 S^* & 0 \\ \alpha\beta_2 P^* & \gamma & \alpha\beta_2 S^* - \mu - \delta & 0 \\ 0 & \eta & \delta & -\mu \end{bmatrix}$$

Clearly, one eigenvalue of the Jacobian matrix is λ_1=-μ and for the remaining eigenvalues, the characteristic equation is $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$ where, $a_1 = \alpha P^* + A + \mu + (\mu + + \delta - \alpha\beta_2 S^*)$

$$a_2 = \alpha P^*[\mathrm{A} + \mu + \delta] + \alpha\beta_1\gamma S^* + A\mu + (\mu + A)[\mu + \delta - \alpha\beta_2 S^*]$$

$$a_3 = \alpha\beta_1 S^*\gamma(2\alpha P^* + \mu) + A\mu[\mu + \delta - \alpha\beta_2 S^*] + A\alpha P^*(\mu + \delta)$$

From equation (3) of the model (1), we get $\mu+\delta-\alpha\beta_2 S^* = \frac{\gamma M^*}{P^*} > 0$ and So, we say that $a_1>0$, $a_2>0$ and $a_3>0$.

$$\begin{aligned}
a_1a_2 - a_3 &= \left(\alpha P^* + A + 2\mu + \delta - \alpha\beta_2 S^*\right)\left(\alpha P^*(\mathrm{A}+\mu+\delta) + \alpha\beta_1\gamma S^* + A\mu + (\mu+A)(\mu+\delta-\alpha\beta_2 S^*)\right) \\
&\quad - \left(\alpha\beta_1 S^*\gamma(2\alpha P^* + \mu) + A\mu(\mu+\delta-\alpha\beta_2 S^*) + A\alpha P^*(\mu+\delta)\right) \\
&= \alpha^2 P^{*2}(\mathrm{A}+\mu+\delta) + \alpha P^*\alpha\beta_1\gamma S^* + \alpha P^*(\mu+A)(\mu+\delta-\alpha\beta_2 S^*) + (A+\mu)(\alpha P^*\ \mathrm{A}) \\
&\quad + A\alpha\beta_1\gamma S^* + A(\mu+A)(\mu+\delta-\alpha\beta_2 S^*) + (\mu+\delta-\alpha\beta_2 S^*)\begin{pmatrix}\alpha P^*(\mathrm{A}+\mu+\delta)+\alpha\beta_1\gamma S^* \\ +(\mu+A)(\mu+\delta-\alpha\beta_2 S^*)\end{pmatrix} \\
&\quad + \mathrm{A}\,\mu(A+\mu) + \alpha^2\beta_1 S^*\gamma P^* + \alpha P^* A\mu
\end{aligned}$$

Here, $\mu+\delta-\alpha\beta_2 S^* = \frac{\gamma M^*}{P^*} > 0$ therefore, we get $a_1a_2 - a_3>0$. Hence, by Routh-Hurwitz Criteria [Anton], $a_1>0$, $a_3>0$ and $a_2a_2 - a_3>0$ then all eigenvalues of Jacobian matrix have negative real parts. Thus, DEE X^* is locally asymptotically stable.

Global Stability

In this section, we examined the global stability at Demonetization Free Equilibrium(DFE) point X_0 and Demonetization Exist Equilibrium(DEE) point X^*.

Theorem 7 Suppose $R_0<1$, then the Demonetization Free Equilibrium point X_0 is globally asymptotically stable.

Proof Here, we used the method of Castillo-Chavez (2002) to prove global stability of Demonetization Free Equilibrium.

Let $Y=B_D$ and $Z=(M,P)$ with $A_0=(Y_0,Z_0)$, where $Y_0=(0)$ and $Z_0=(0,0)$. We have

$$\frac{dY}{dt} = g(Y,Z) = \eta M + \delta P - \mu B_D$$

At $Z=Z_0$, $\frac{dY}{dt} = G(Y,0) = -\mu B_D$ as $\mathrm{t}\to\infty, \mathrm{Y}\to\mathrm{Y}_0$.

Hence, $Y=Y_0=(B_{Do}=0)$ is globally asymptotically stable. From system (1), we get

$$\frac{dZ}{dt} = H(Y,Z) = F_1 Z - H^*(Y,Z)$$

where $H^*(Y,Z) = \begin{bmatrix} \alpha\beta_1 SP \\ \alpha\beta_2 SP \end{bmatrix}$ and $F_1 = \begin{bmatrix} \gamma+\mu+\eta & 0 \\ -\gamma & \mu+\delta \end{bmatrix}$

It is clear that F_1 is an M-matrix. Here, $S>0$ and $P>0$ and also α, β_1 and β_2 are non-negative. So, we have $H^*(Y,Z)\geq 0$. Hence, Demonetization free equilibrium is globally asymptotically stable if $R_0<1$.

Theorem 8 If $R_0>1$, then the Demonetization Exist Equilibrium point X^* is globally asymptotically stable.

Proof In this section, we explored the possible endemic equilibrium forX^* of the system (1). Let $X^*= (S^*,M^*,P^*,B_D{}^*)$, from equation (10), we have $P^* = -\frac{\mu}{\alpha}(1-R_0)$ which is positive only if $R_0>1$. Now, if we substitute P^* into equation (7), we get

$$S^* = \frac{A(\mu+\delta)}{\alpha(\mathrm{A}\,\beta_2+\gamma\beta_1)}. \tag{13}$$

It follows from (7) to (10) (both P^* and S^* are positive if $R_0>1$) that $X^* \in R_4^+$ when $R_0>1$. Thus, the following theorem is established.

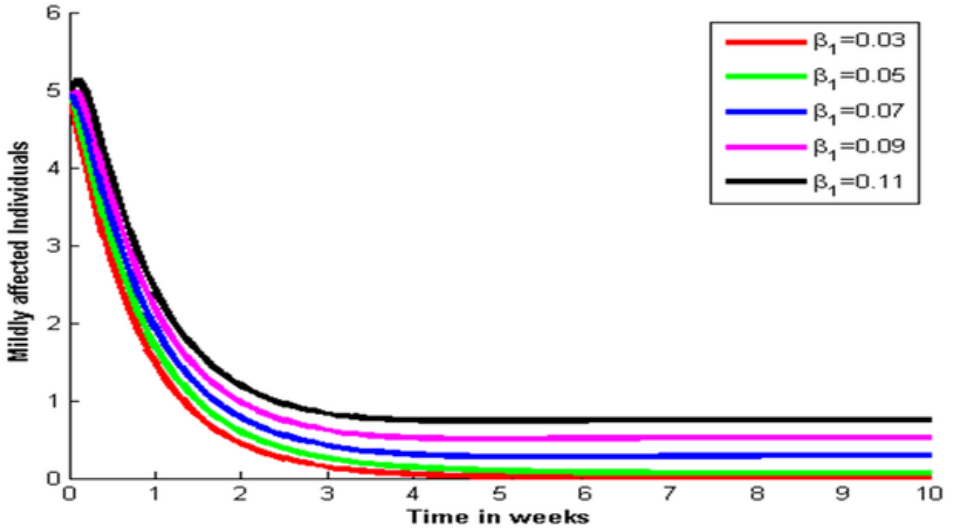
Theorem 9 The system (1) has a unique endemic (positive) equilibrium is given by X^*, whenever $R_0>1$.

NUMERICAL SIMULATION

In this section, we have validated our numerical data in table 1 with MATLAB simulation using Runge-Kutta forth order iterative method. Here, we discussed the effect of the affected rates for the demonetization. The system stability for $R_0<1$ and $R_0>1$ is also discussed.

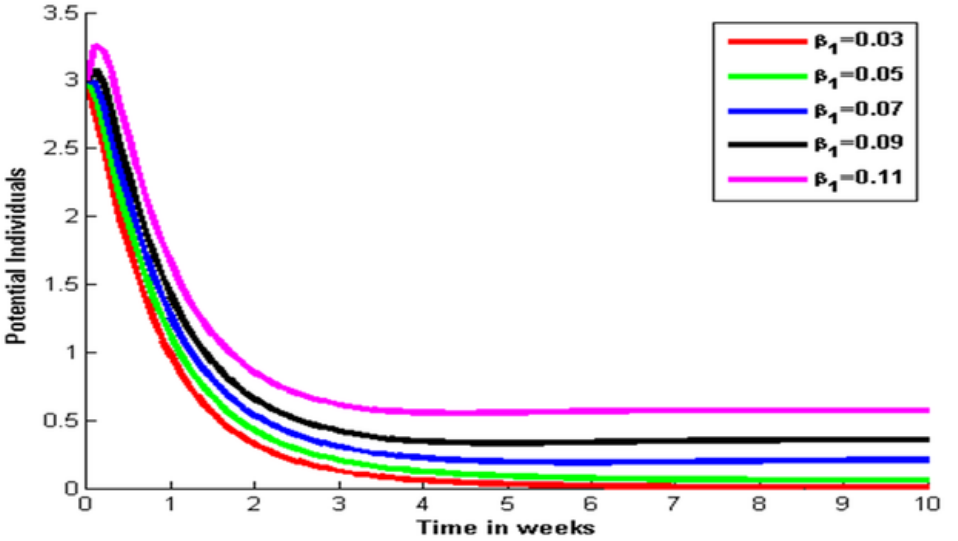
The figure 2a and figure 2b shows that the transmission rate from susceptible to demonetization affected individuals; is increasing then affected individuals are increasing means affected individuals and transmission rate are propositional. If is increasing from 3% to 11% then affected individuals increases in 3 weeks.

Figure 2. (a) Effect of β_1 on mildly affected individuals and (b) Effect of β_1 on potential individuals



In figure 3, we observed the effect of basic reproduction number. If basic reproduction less than unity then susceptible individuals goes to equilibrium point $\frac{B}{\mu}$, that is 16.66 and other demonetization affected individuals converges to zero

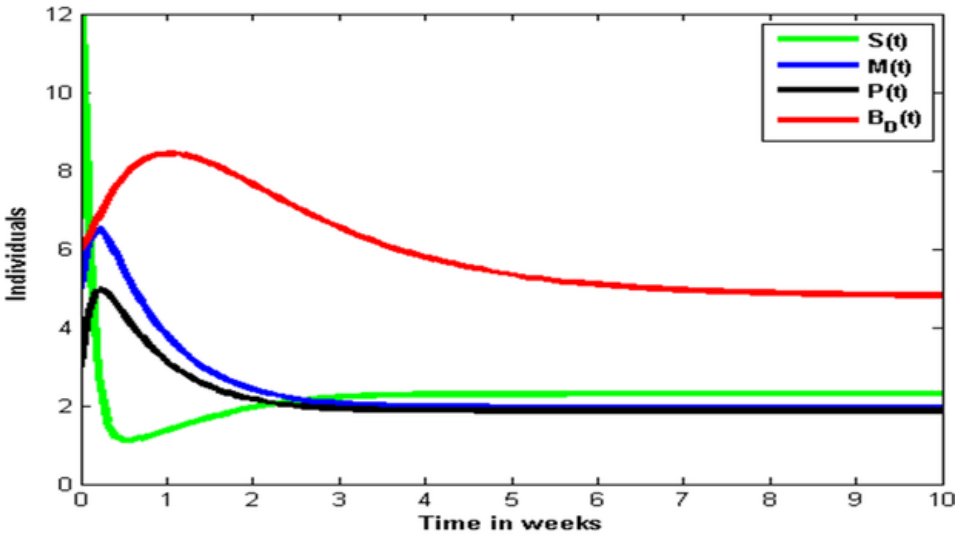
Figure 3. Compartments with R_0<1



after seven weeks. Bank depositors are increasing in the beginning and then decreasing after 5 weeks and reaches to zero. This shows that if $R_0<1$, then the system is stable after 3 weeks.

Figure 4 shows that if $R_0>1$ then individuals in affected compartment increases in the beginning then after decreases but not convergent to zero. If currency ban is declared then individuals have deposited their cash in the banks in first 3 weeks. But after that currency becomes piece of paper. So, affected individuals converge to zero which is not observed in figure 4 that proves that if $R_0>1$, system is unstable.

Figure 4. Compartments with $R_0>1$



CONCLUSION

In this paper, the dynamical model for effect of demonetization is proposed and mathematical analysis of the nonlinear differential equations are carried out to get insight into the qualitative dynamics in presence of demonetization effect. The effects of demonetization model depend on time interval for epidemic spreading in the society is formulated. The prevalent threshold R_0 of the proposed model is calculated by next generation matrix method. The demonetization free equilibrium point (DFE) and demonetization exist equilibrium point (DEE) are worked out. The model has a locally–asymptotically stable at demonetization free equilibrium point whenever the associated effective threshold is less than unity. Stability analysis for $R_0<1$ and

R_0>1 are studied theoretically and numerically for the demonetization model. This study suggests that for curbing corruption and black money use digital payment.

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About the Contributors

Nita H. Shah is a professor in the Department of Mathematics, Gujarat University, Ahmedabad, India. She received her Ph.D. in inventory control management, operations research. Currently, she is engaged in research in inventory control and management, supply chain management, forecasting and information technology and information systems, neural networks, sensors and image processing. She has more than 275 papers published in international and national journals. She is author of four books. She is serving as a member of the editorial board of Investigation Operational, Journal of Social Science and Management, International Journal of Industrial Engineering and Computations and Mathematics Today.

Mandeep Mittal started his carrier in the education industry in 2000 with Amity Group. Currently, he is working as an Assistant Professor in the Department of Mathematics, Amity Institute of Applied Sciences, Amity University Noida. He earned his Post Doctorate from Hanyang University, South Korea, 2016, Ph.D. (2012) from University of Delhi, India, and Post-graduation in Applied Mathematics from IIT Roorkee, India (2000). He has published more than 50 research papers in the International Journals and International Conferences. He authored one book with Narosa Publication on C language, and edited three Research books with IGI Global and Springer. He has been awarded Best Faculty Award by the Amity School of Engineering and Technology, New Delhi for the year 2016-2017. He also served as Dean of Students Activities at Amity School of Engineering and Technology, Delhi for nine years and as a Head, Department of Mathematics in the same institute for one year. He actively participated as a core member of organizing committees in the International Conferences in India and outside India.

* * *

Sumit Kaur Bhatia has obtained his PhD from IIT Delhi. Currently, she is the Assistant Professor in the Department of Applied Mathematics, Amity Institute of Applied Sciences, Amity University. She has published more than 25 research

papers in the areas of mathematical modelling in eco-toxicology and epidemiology. Se has guided M.Sc. Dissertations in Applied mathematics. Her research interests include mathematical modelling in dynamical system.

Prianka Bose is a Graduate Teaching Assistant (GTA) in Department of Mathematical Sciences, New Jersey Institute of Technology, Newark,NJ. Did her masters from Amity Institute of Applied Sciences, Amity University, Sector-125, Noida.

D. K. Chaturvedi is working in Dept. of Elect. Engg, Faculty of Engg, D.E.I., Dayalbagh, Agra since 1989. Presently he is Professor. He did his B.E. from Govt. Engineering College Ujjain, M.P. then he did his M.Tech. and Ph.D. from D.E.I. Dayalbagh. He is gold medalist and received Young Scientists Fellowship from DST, Government of India in 2001-2002 for post doctorial research at Univ. of Calgary, Canada. Also, he had research collaboration with different organizations at national and international level. He is the Fellow - The Institution of Engineers (India), Fellow - Aeronautical Society of India, Fellow - IETE, Sr. Member IEEE, USA and Member of many National and International professional bodies such as IET, U.K., ISTE, Delhi, ISCE, Roorkee, IIIE, Mumbai and SSI etc. The IEE, U.K. recognized his work in the area of Power System Stabilizer and awarded honorary membership to him in 2006. He did many R&D projects of MHRD, UGC, AICTE etc. and consultancy projects of DRDO. He contributed in the national mission of ICT of Govt. of India as Virtual Power Lab Developer. He has guided 10 Ph.Ds., 65 M.Tech. Dissertations and published more than 300 International and National Papers. He has chaired and Co-Chaired many International and National Conferences. He is referee of many International Journals including IEE Proceedings and IEEE Transactions. He is Head of Dept. of Footwear Technology, Convener, Faculty Training and Placement Cell, and Advisor, IEI Students' Chapter (Elect. Engg.), D.E.I. Dayalbagh, Agra.

Kuldeep Chaudhary obtained his Ph.D. in Operational Research from University of Delhi and M.Sc. from Indian Institute of Technology(IIT), Roorkee respectively. Currently, he is the Assistant Professor in the Department of Applied Mathematics, Amity Institute of Applied Sciences, Amity University. He has published more than 25 research papers in the areas of optimization in software reliability, Marketing and inventory. He has guided M.Sc. Dissertations in Applied mathematics. His research interests include mathematical modelling in optimal control theory and optimization in marketing, Inventory-production and software reliability.

Sudipa Chauhan has obtained his PhD from Jiwaji University, Gwalior in Bio-mathematics. Currently, she is the Assistant Professor in the Department of

Applied Mathematics, Amity Institute of Applied Sciences, Amity University. She has published more than 25 research papers in the areas of mathematical modelling in eco-toxicology and epidemiology. Se has guided M.Sc. Dissertations in Applied mathematics. Her research interests include mathematical modelling in dynamical system.

R. Dhineshbabu is a Research Scholar at Sacred Heart College, Tirupattur, Tamil Nadu, S. India and pursuing his Ph.D in Mathematics at Thiruvalluvar University, Vellore, Tamil Nadu, S. India. He is a graduate in Mathematics from Thiruvalluvar University, Vellore, Tamil Nadu, S. India. His areas of specialization are Applied and Computational Mathematics, Mathematical Modelling, Nonlinear Analysis, Mathematical Biology, Oscillation theory and Boundary value problems.

Priyanshi Garg is pursuing her B.Tech from ABES Engineering College and currently is in 2nd year. She is learning programming and has keen interest to develop the knowledge of Data Structure and Algorithms. She likes to play badminton.

Mayank Gupta is acting as System and IT Analyst in Tata Consultancy services, Noida and expert of Data sciences and Business Analytics. He has skill to visualize the situations from different perspectives and explore the real facts through critical Analysis. He has deep interest in Human health domains. Currently he is working on Japan Projects on Life Sciences.

Ekta N. Jayswal is a research scholar in Department of Mathematics, Gujarat University. She is working in dynamics of pollution and its control. She has published four research articles in Scopus indexed journals.

Purvi M. Pandya is a research scholar in Department of Mathematics, Gujarat University. Her research interest is development of dynamical system for social and health issues. She has published three research articles in Scopus indexed journals.

Zalak Patel is associated with L D College of Engineering. He has 6 publications in Vertical Dynamics of Infectious Diseases.

Mukund Rastogi is Engineering student in AKTU Univ. Presently he is B.Tech. Second Year student of CSE in ABESEC, Ghaziabad. India. He is working presently on Yagya and Mantra therapy and its analysis by Machine Learning. He has keen interest in Google surfing. His hobbies are playing badminton and reading books. He is young, talented, and dynamic.

Rohit Rastogi received his B.E. degree in Computer Science and Engineering from C.C.S. Univ. Meerut in 2003, the M.E. degree in Computer Science from NITTTR-Chandigarh (National Institute of Technical Teachers Training and Research-affiliated to MHRD, Govt. of India), Punjab Univ. Chandigarh in 2010. Currently he is pursuing his Ph.D. In computer science from Dayalbagh Educational Institute, Agra under renowned professor of Electrical Engineering Dr. D.K. Chaturvedi in area of spiritual consciousness. Dr. Santosh Satya of IIT-Delhi and dr. Navneet Arora of IIT-Roorkee have happily consented him to co supervise. He is also working presently with Dr. Piyush Trivedi of DSVV Hardwar, India in center of Scientific spirituality. He is a Associate Professor of CSE Dept. in ABES Engineering. College, Ghaziabad (U.P.-India), affiliated to Dr. A.P. J. Abdul Kalam Technical Univ. Lucknow (earlier Uttar Pradesh Tech. University). Also, He is preparing some interesting algorithms on Swarm Intelligence approaches like PSO, ACO and BCO etc. Rohit Rastogi is involved actively with Vichaar Krnati Abhiyaan and strongly believe that transformation starts within self.

Moksha H. Satia is research scholar in Department of Mathematics, Gujarat University, India. She is working in dynamical systems of eco-friendly society. Her research interest is applications of mathematics in various disciplines. She has published 9+ papers in SCOPUS indexed journals. She has also been awarded the Best Thesis Award of 2019 in mathematical sciences.

Yash Shah is a medical student (completed his M.B.B.S and internship) and his research interest is in analyzing disease spread and factors responsible for the spread. He is preparing for USMLE for post-graduate studies and research.

Nisha Sheoran is a research scholar in Department of Mathematics, Gujarat University.She's working with dynamical system of co-infectious diseases.

Parul Singhal received her B.Tech degree from AKTU Univ. Presently She is M.Tech. Second Year student of CSE in ABESEC, Ghaziabad. She is a Teaching Assistant in the CSE department of ABES Engineering. Ghaziabad, India. She is working presently on data mining (DM) and machine learning (ML). She is also working on Yagopathy. She has keen interest in Google surfing. Her hobbies are playing badminton and reading books. She is young, talented, and dynamic.

Ankush H. Suthar is research scholar in Department of Mathematics, Gujarat University, India. He is working in dynamical systems and mathematical modeling of health problem related to infectious diseases. He has published 2-papers in

JMCS and IJCER on transmission of deadly infectious diseases like Nipah-virus and swine-flu.

Bijal Yeolekar has research interest in dynamical system and its analysis. She has published 10 articles. She is recipient of Best Thesis award.

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