# **CHAPTER-3**

Study of Stability Analysis and Synchronization of Fractional-Order Biological Models Using Active Control Method

# Chapter 3.1

Stability analysis of fractional-order generalized chaotic susceptible-infected-recovered epidemic model and its synchronization using active control method

# 3.1.1 Introduction

Mathematical modeling is widely used to analyze and gain inside into the process of spread of infectious diseases which can eventually be used to predict the future course of outbreak and to evaluate strategies to control an epidemic. Mathematical epidemiology and population dynamics are some of the suitable forms of describing the biological systems using the language of dynamical systems theory. In this connection we may refer to Buonomo et al. (2008), who stated the usefulness of mathematical epidemiology in revealing valuable information regarding the spread and control of infectious diseases. In a given model a person contracting the disease and then becoming immune to future infection after recovery is called susceptible-infectedrecovered (SIR). This phenomenon of biological systems is directly connected to time evolution of population density of interacting species or individuals in different states (say, susceptible and infected). Due to complexity of interactions amongst species, it is difficult to solve the models describing such systems analytically. Therefore, once model is formulated mathematically, it is necessary to solve numerically using computer simulations to predict the response of biological systems. The study of dynamic implications of information dependent vaccination for SIR vaccine used in preventable childhood diseases can be found in the article of D'Onofrio et al. (2007a).

The contents of this chapter have been accepted in *Pramana - Journal of Physics*.

Simple epidemiological models with information dependent vaccination functions gives rise to sustained oscillations via Holf bifurcation as found in D'Onofrio *et al.* (2007). Recently, the local and global stability of epidemic equilibrium has been well studied by Kar and Mondal (2011).

Fractional order modeling has been an active field of research nowadays both from the theoretical and the applied perspectives. A wide range of problems in different branches of engineering and biology have already been studied by a number of researchers from different parts of the world to explore the potential of the fractional derivative. The usage of first order time derivative with a fractional order time derivative is not only applicable for non-Gaussian but also for Non-Markovian systems. Fractional SIR epidemic model equations are obtained from the classical SIR epidemic equations in mathematical modeling by replacing first order derivatives by fractional derivative of order  $\alpha$  (0 <  $\alpha \le 1$ ). Several universal phenomena can be modeled to a great degree of accuracy by using the property of these evolution equations. In contrast to integer order differential operators, which are local operator, a fractional order differential operator is non-local in the sense that it takes into account the fact that the future state not only depends upon the present state but also upon all of the history of its previous states. For this realistic property, the usage of fractional order systems is becoming popular to model the behaviour of real systems in various fields of science and engineering. It is to be noted that the present states of any real life dynamic system are dependent upon the history of its past states. Such circumstances have motivated the author to study the SIR epidemic model which has a great physical relevance from the perspective of public health policies and its consideration as fractional order system in allied problems is valid.

Synchronization is a vital phenomenon of chaos that may occur when two or more chaotic systems are coupled with or one chaotic system derives the other. Synchronization between two structurally identical/non-identical systems with different initial conditions have attracted a great deal of interest in various fields due to its important applications in secure communication, system identification, pattern reorganization, vibration technology, economical system, ecological system, biology and biotechnology. After the pioneering investigation of Pecore and Corrall (1990), that chaotic systems can be made to synchronize by linking them with common signals, lot of research works for synchronizing chaotic systems using linear and nonlinear feedback control, adaptive control, active control, time delay feedback control, tracking control, sliding mode control methods have already been done (Ho and Hung (2002), Park (2006), Vincent (2008) Ansari and Das (2013), Yassen (2005)), out of which active control method is very efficient and easy to use for the synchronization of a pair of identical or non-identical chaotic systems. Synchronization of fractional order chaotic system has been first studied by Li at al. (2003). Synchronization between fractional order chaotic systems is also being widely investigated in (Deng and Li (2005), Li and Yan (2007), Yan and Li (2007), Agrawal and Das (2013), Srivastava et al. (2012), Srivastava et al. (2014)). In 2012, Agrawal et al. (2012) have successfully applied the active control method for synchronization of different pair of fractional order chaotic systems. To the best of author's knowledge the occurrences of chaotic attractors for a fractional order SIR model have not yet been explored by any researcher.

In this chapter the synchronization between a pair of identical fractional order chaotic SIR model using the active control method is studied. Numerical simulations have been carried out for different order fractional derivatives close to the standard one which are depicted through graphs for different particular cases. The aim of the study is to investigate the minimum time required for synchronization when the fractional order time derivative approaches the standard order.

## 3.1.2 System description

Let us consider the following SIR epidemic model

$$D_{t}^{\alpha}S = rS(1 - \frac{S}{k}) - \frac{\beta SI}{1 + aS}$$

$$D_{t}^{\alpha}I = \frac{\beta IS}{1 + aS} - \mu I - \gamma I$$

$$D_{t}^{\alpha}R = \gamma I - \lambda R, \quad 0 < \alpha \le 1,$$
(3.1.1)

where  $0 < \alpha \le 1$ , S is the density of susceptible within the population, *I* is the density of infected within the population, *R* is the density of recovered within the population, and the parameters viz., *r* is the intrinsic growth rate of susceptible, *k* is the carrying capacity of susceptible, *a* is the saturation factor that measures the inhibitory effect,  $\beta$  is the transmission or contact rate,  $\gamma$  is the rate of recovery from infection,  $\mu$  and  $\lambda$  are the death rates. We consider a new variable *Z*,

$$Z(t) = \int_{-\infty}^{t} g(S(\tau), I(\tau)) K(t-\tau) d\tau , \qquad (3.1.2)$$

known as information variable (Buonomo *et al.* (2008), D'Onofrio *et al.* (2007a, 2007), Kar and Mondal (2011)), which depends on current values of state variables and also summarizes information about past values of state variables. Here  $K(t-\tau)$  is the delaying kernel,  $\tau$  is the distributed delay with  $\tau \leq t$ . Assuming that g(S, I) = S and  $K(t-\tau) = \frac{1}{T} \exp(-\frac{1}{T}(t-\tau))$ , where *T* is the average delay of the collected information on the disease, as well as the

average length of the historical memory concerning the disease, the model (3.1.1) reduces to

$$D_{t}^{\alpha} S = rS(1 - \frac{S}{k}) - \frac{\beta SI}{1 + aS}$$

$$D_{t}^{\alpha} I = \frac{\beta IZ}{1 + aZ} - \mu I - \gamma I$$

$$D_{t}^{\alpha} Z = \frac{1}{T} (S - Z)$$

$$D_{t}^{\alpha} R = \gamma I - \lambda R.$$
(3.1.3)

The last equation of (3.1.3) can be ignored since here the dynamics of *R* depends only on the dynamics of *I*. Therefore, we will concentrate in the study of the following fractional order nonlinear system:

$$D_{t}^{\alpha}S = rS(1-\frac{S}{k}) - \frac{\beta SI}{1+aS}$$

$$D_{t}^{\alpha}I = \frac{\beta IZ}{1+aZ} - \mu I - \gamma I$$

$$D_{t}^{\alpha}Z = \frac{1}{T}(S-Z),$$
(3.1.4)

where r, *k*, *a*,  $\beta$ ,  $\mu_1$ ,  $\gamma$ , *T* > 0.

### 3.1.2.1 Equilibrium points and their asymptotic stability

To evaluate the equilibrium points, let

$$D_t^{\alpha} S = 0, D_t^{\alpha} I = 0, D_t^{\alpha} Z = 0.$$
 (3.1.5)

The system (3.1.4) has trivial equilibrium at  $E_0 = (0, 0, 0)$ , disease free equilibrium at  $E_1 = (k, 0, k)$  and endemic equilibrium at point  $E_2 = (\overline{S}, \overline{I}, \overline{Z})$ , where

$$\overline{S} = \frac{\mu + \gamma}{\beta - a(\mu + \gamma)}$$

$$\overline{I} = \frac{r}{\beta k} (1 + a\overline{S})(k - \overline{S})$$

$$\overline{Z} = \frac{\mu + \gamma}{\beta - a(\mu + \gamma)}.$$
(3.1.6)

Jacobian Matrix of the system (3.1.4) is given by

$$J = \begin{pmatrix} r - \frac{2rS}{k} - \frac{\beta I}{(1+aS)^2} & \frac{-\beta S}{1+aS} & 0\\ 0 & \frac{\beta Z}{1+aZ} - \mu - \gamma & \frac{\beta I}{(1+aZ)^2}\\ \frac{1}{T} & 0 & \frac{-1}{T} \end{pmatrix}.$$
 (3.1.7)

The eigenvalues of the equilibrium points can be determined by solving the characteristic equation

$$\left|J - \lambda I\right| = 0 \tag{3.1.8}$$

i.e., 
$$\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0$$
. (3.1.9)

The equilibrium point is locally asymptotically stable if all the eigenvalues  $\lambda$  of Jacobian matrix J(E) satisfy the following condition (Matignon (1996), Ahmed *et al.* (2007))

$$\left|\arg(\lambda)\right| > \frac{\alpha \pi}{2}.$$
 (3.1.10)

At trivial equilibrium point  $E_0 = (0, 0, 0)$ , the jacobian matrix is given by

$$J(E_0) = \begin{pmatrix} r & 0 & 0 \\ 0 & -(\mu + \lambda) & 0 \\ \frac{1}{T} & 0 & -\frac{1}{T} \end{pmatrix}.$$

Characteristic equation is given by

 $\sim 63 \sim$ 

$$\lambda^{3} + \lambda^{2} \{ \gamma + \mu + \frac{1}{T} - r \} + \lambda \{ \frac{1}{T} (\gamma + \mu) - r(\gamma + \mu + \frac{1}{T}) \} - \frac{r}{T} (\gamma + \mu) = 0.$$

At disease free equilibrium point  $E_1 = (k, o, k)$ ,

$$J(E_1) = \begin{pmatrix} -r & \frac{-\beta k}{1+ak} & 0\\ 0 & -(\gamma + \mu - \frac{\beta k}{1+ak}) & 0\\ \frac{1}{T} & 0 & -\frac{1}{T} \end{pmatrix}.$$

Characteristic equation is given by

$$\lambda^{3} + \lambda^{2} \left( r + \frac{1}{T} + M \right) + \lambda \left\{ \frac{r}{T} + M \left( r + \frac{1}{T} \right) \right\} + \frac{Mr}{T} = 0,$$

where  $M = (\gamma + \mu - \frac{\beta k}{1 + ak})$ .

At endemic equilibrium point  $E_2 = (\overline{S}, \overline{I}, \overline{Z})$ ,

$$J(E_2) = \begin{pmatrix} r - \frac{2r\overline{S}}{k} - \frac{\beta I}{(1 + a\overline{S})^2} & \frac{-\beta \overline{S}}{1 + a\overline{S}} & 0\\ 0 & \frac{\beta \overline{Z}}{1 + a\overline{Z}} - \mu - \gamma & \frac{\beta \overline{I}}{(1 + a\overline{Z})^2}\\ \frac{1}{T} & 0 & \frac{-1}{T} \end{pmatrix},$$

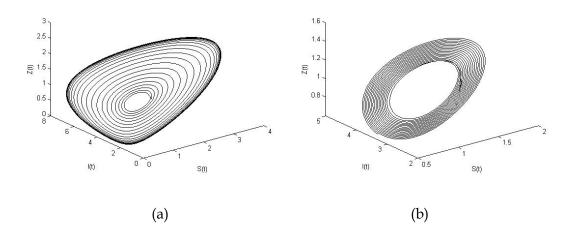
where 
$$\overline{S} = \frac{\mu + \gamma}{\beta - a(\mu + \gamma)}$$
,  $\overline{I} = \frac{r}{\beta k} (1 + a\overline{S})(k - \overline{S})$ ,  $\overline{Z} = \frac{\mu + \gamma}{\beta - a(\mu + \gamma)}$ .

Characteristic equation is given by

$$\lambda^3 + \lambda^2 (M + N + \frac{1}{T}) + \lambda (MN + \frac{M}{T} + \frac{N}{T}) + \frac{MN}{T} + \frac{\beta^2 \overline{S} \overline{I}}{T(1 + a\overline{S})(1 + a\overline{Z})^2} = 0,$$

where 
$$M = (\gamma + \mu - \frac{\beta \overline{Z}}{1 + a\overline{Z}})$$
,  $N = (\frac{\beta \overline{I}}{(1 + a\overline{S})^2} + \frac{2r\overline{S}}{k} - r)$ .

It is known that an equilibrium point E is said to be a saddle point of index 1 if the jacobian matrix at E has one eigen value with a non-negative real part and a saddle point of index 2 if the jacobian matrix at E has two unstable eigenvalues. It is noticed that the scrolls are generated only around the saddle points of index 2, whereas saddle points of index 1 are responsible only for connecting scrolls (Chua et al. (1986), Silva (1993), Cafagna and Grassi (2003), Lu et al. (2004), Tavazoei and Haeri (2007)). With this idea we proceed to find the eigen values for predicting that a point to be stable or unstable taking the parameters as r = 2, k = 5, a = 0.01,  $\beta = 0.5$ ,  $\mu = 0.3$ ,  $\gamma = 0.2$ , T = 0.58. It is seen that at the point  $E_0 = (0, 0, 0)$ , the eigen values are 2, -0.5000, -1.7647, which clearly shows that the point  $E_0$  is unstable. At point  $E_1 = (k, 0, k) = (5, 0, 5)$ , the eigen values are 1.8810, -1.7647, -2, which implies that the point  $E_1$  is unstable. At the point  $E_2 = (\overline{S}, \overline{I}, \overline{Z}) = (1.0101, 3.22416, 1.0101)$ , the eigen values are -1.63339, 0.03442 + 0.75354i, 0.03442 - 0.75354i, which clearly exhibits that  $E_2$  is a saddle point of index 2 for  $\alpha > 0.9704$  satisfying the condition (3.1.10). At this point the chaotic attractors of the system (3.1.4) for different values of  $\alpha$  are depicted through Fig. 3.1.1.



**Fig.3.1.1.** The chaotic attractors of the generalized SIR model (3.1.4): (a) for fractional order  $\alpha = 0.99$ ; (b) for  $\alpha = 0.975$ .

# 3.1.3 Synchronization of the fractional order SIR model using active control method

Here the drive system is described by equation (3.1.4) as

$$D^{\alpha}S_{1} = rS_{1}(1 - \frac{S_{1}}{k}) - \frac{\beta S_{1}I_{1}}{1 + aS_{1}}$$

$$D^{\alpha}I_{1} = \frac{\beta I_{1}Z_{1}}{1 + aZ_{1}} - \mu I_{1} - \gamma I_{1}$$

$$D^{\alpha}Z_{1} = \frac{1}{T}(S_{1} - Z_{1}),$$
(3.1.11)

and the response system as

$$D^{\alpha}S_{2} = rS_{2}(1 - \frac{S_{2}}{k}) - \frac{\beta S_{2}I_{2}}{1 + aS_{2}} + \mu_{1}(t)$$

$$D^{\alpha}I_{2} = \frac{\beta I_{2}Z_{2}}{1 + aZ_{2}} - \mu I_{2} - \gamma I_{2} + \mu_{2}(t)$$

$$D^{\alpha}Z_{2} = \frac{1}{T}(S_{2} - Z_{2}) + \mu_{3}(t),$$
(3.1.12)

where  $\mu(t) = [\mu_1(t), \mu_2(t), \mu_3(t)]^T$  is the controller to be designed. To investigate the synchronization of systems (3.1.11) and (3.1.12), the error states are defined as  $e_1 = S_2 - S_1$ ,  $e_2 = I_2 - I_1$ ,  $e_3 = Z_2 - Z_1$ . The corresponding error dynamics system can be obtained by subtracting equation (3.1.11) from equation (3.1.12), which is given by

$$\frac{d^{\alpha}e_{1}}{dt^{\alpha}} = re_{1} - \frac{r}{k}(S_{2}^{2} - S_{1}^{2}) - \frac{\beta S_{2}I_{2}}{1 + aS_{2}} + \frac{\beta S_{1}I_{1}}{1 + aS_{1}} + \mu_{1}(t)$$

$$\frac{d^{\alpha}e_{2}}{dt^{\alpha}} = -\mu_{1}e_{2} - \gamma e_{2} + \frac{\beta Z_{2}I_{2}}{1 + aZ_{2}} - \frac{\beta Z_{1}I_{1}}{1 + aZ_{1}} + \mu_{2}(t)$$

$$\frac{d^{\alpha}e_{3}}{dt^{\alpha}} = \frac{1}{T}(e_{1} - e_{3}) + \mu_{3}(t).$$
(3.1.13)

The two systems (3.1.11) and (3.1.12) are realized to synchronize if the system (3.1.13) is globally asymptotically stable under a suitable controller. Defining active control functions  $\mu_i(t)$  (i = 1, 2, 3) as

$$\mu_{1}(t) = V_{1}(t) + \frac{r}{k} (S_{2}^{2} - S_{1}^{2}) + \frac{\beta S_{2}I_{2}}{1 + aS_{2}} - \frac{\beta S_{1}I_{1}}{1 + aS_{1}}$$

$$\mu_{2}(t) = V_{2}(t) - \frac{\beta Z_{2}I_{2}}{1 + aZ_{2}} + \frac{\beta Z_{1}I_{1}}{1 + aZ_{1}}$$

$$\mu_{3}(t) = V_{3}(t),$$
(3.1.14)

which leads to the error functions as

$$\frac{d^{\alpha} e_{1}}{dt^{\alpha}} = re_{1} + V_{1}$$

$$\frac{d^{\alpha} e_{2}}{dt^{\alpha}} = -\mu_{1}e_{2} - \gamma e_{2} + V_{2}$$

$$\frac{d^{\alpha} e_{3}}{dt^{\alpha}} = \frac{1}{T}(e_{1} - e_{3}) + V_{3},$$
(3.1.15)

where  $V_1(t)$ ,  $V_2(t)$ ,  $V_3(t)$  are the linear control inputs chosen such that the system (3.1.15) becomes stable. Now, consider

$$\begin{bmatrix} V_1(t) \\ V_2(t) \\ V_3(t) \end{bmatrix} = M \begin{bmatrix} e_1 \\ e_2 \\ e_3 \end{bmatrix},$$

where *M* is a 3×3constant matrix. In order to make the closed loop system stable, the matrix *M* should be selected in such a way that the error dynamical system has eigenvalues  $\lambda_i$  which satisfy the condition  $|\arg(\lambda_i)| > \frac{\pi \alpha}{2}, i = 1,2,3$ . There is not a unique choice for such matrix *M*, a good choice can be as fallows

$$M = \begin{bmatrix} -(r+1) & 0 & 0 \\ 0 & (\mu_1 + \gamma - 1) & 0 \\ -\frac{1}{T} & 0 & \frac{1}{T} - 1 \end{bmatrix},$$

which reduces the error system to

$$\frac{d^{\alpha}e_{1}}{dt^{\alpha}} = -e_{1}$$

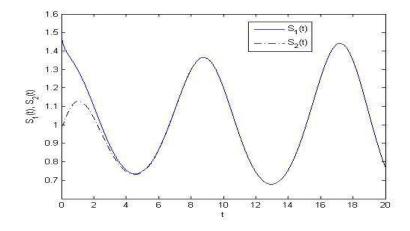
$$\frac{d^{\alpha}e_{2}}{dt^{\alpha}} = -e_{2}$$

$$\frac{d^{\alpha}e_{3}}{dt^{\alpha}} = -e_{3}.$$
(3.1.16)

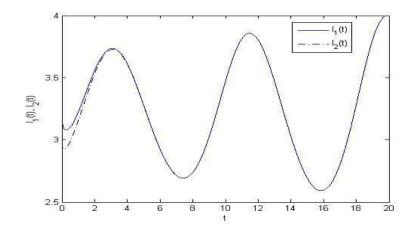
Here all eigenvalues  $\lambda_i$  are -1, which satisfy the condition  $|\arg(\lambda_i)| > \alpha \pi/2$ , for  $0 < \alpha \le 1$ . Therefore, the linear system (3.1.16) is stable and thus the required synchronization is obtained.

#### 3.1.3.1 Numerical simulations and results

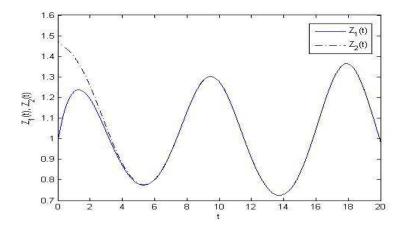
In numerical simulation the parameters of the SIR system are taken as  $r = 2, k = 5, a = 0.01, \beta = 0.5, \mu_1 = 0.3, \gamma = 0.2, T = 0.58$ . Time step size is taken as 0.005. State trajectories of drive system (3.1.11) and response system (3.1.12) are shown in the Fig 3.1.2 and Fig 3.1.3 demonstrate that the systems are synchronized after small duration of time for the considered fractional order time derivatives  $\alpha = 0.99$  and  $\alpha = 0.975$ . It is also seen from the figures that the time taken for synchronization of system decreases with the increase of fractional order approaching towards standard order system.



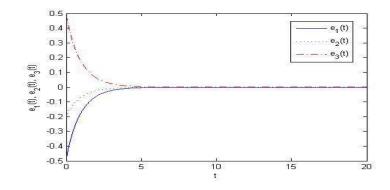
(a) State trajectories between  $S_1$  and  $S_2$ .



(b) State trajectories between  $I_1$  and  $I_2$ .

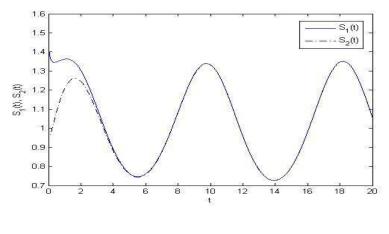


(c) State trajectories between  $Z_1$  and  $Z_2$ .

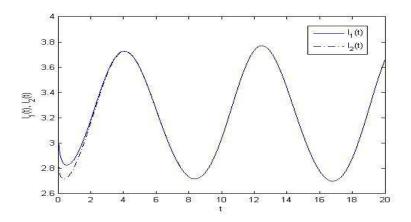


(d) The evolution of the error states  $e_1(t), e_2(t), e_3(t)$ .

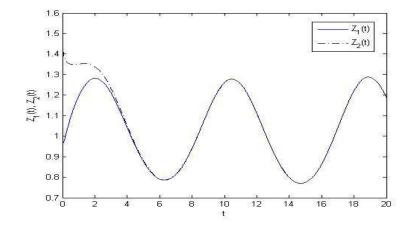
**Fig.3.1.2** The state trajectories of systems (3.1.11) and (3.1.12) between state vectors and evolution of error vectors for the fractional-order  $\alpha$ =0.99.



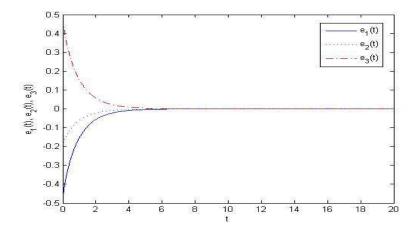
(a) State trajectories between  $S_1$  and  $S_2$ .



(b) State trajectories between  $I_1$  and  $I_2$ .



(c) State trajectories between  $Z_1$  and  $Z_2$ .



(d) The evolution of the error states  $e_1(t), e_2(t), e_3(t)$ .

**Fig.3.1.3.** The state trajectories of systems (3.1.11) and (3.1.12) between state vectors and evolution of error vectors for the fractional-order  $\alpha$ =0.975

# 3.1.4 Conclusion

In a nut shell, the author has achieved four important goals in the present article. First one is the study of stability analysis of fractional order SIR model. Second one is the study of dynamical behavior of two identical fractional order chaotic systems. Third one is the successful implementation of the powerful active control method which provides us a simple way to synchronize coupled chaotic systems. Fourth, the observation that the synchronization time increases when the system pair approaches the standard order from fractional order, is a major outcome of the study. Numerical simulation results demonstrate the ease of implementation, the reliability and effectiveness of the proposed control technique even for the synchronization of fractional order chaotic systems. The author believes that the present study will be appreciated and can be utilized by the researchers involved in the field of mathematical modeling of fractional order dynamical systems.

# Chapter 3.2

# Stability analysis of fractional-order water-borne epidemic model

# 3.2.1 Introduction

Modelling of biological systems is a significant task of systems biology and mathematical biology. Mathematical language is very helpful in designing the precise description of the complicated systems arising in biological sciences. Biological systems are inherently complex. Interaction of numerous molecular components, biochemical reactions, cell structure and compartmentalization of random effect makes the system more complicated. Due to these complexities, it is hard to solve the models describing biological systems analytically. Therefore, once model is formulated mathematically, it is needed to solve numerically using computer simulations and predict the response of biological system.

Water-Borne diseases have become one of the attractive areas of research in epidemiology and public health. The diseases are caused by bacteria, virus and parasites. The most serious disease of public health concern observed in the developing countries and treated as the universally infectious one is the Diarrhoea, one of the important forms due to infection from water borne pathogens. Other severe water borne diseases named as Zimbabwe cholera epidemic causes lot of casualties every year (WHO (2009)). Therefore, prevention of long range spreading of these infectious diseases is challenging. Nowadays, mathematicians are involved to present some physical models

The contents of this chapter have been published in **Communication in Fractional Calculus**, 4(1) (2013) 25-31.

through which it can be understood how various transmission pathways affect disease dynamics by extending classical epidemic theory to SIR type model which include a water compartment. The SIR (Susceptible-Infected-Recovered) model is on described by first order ordinary differential equations and has been used in several epidemiological diseases (Hethcote (2000), Lu et al. (2002), Piccolo and Billings (2005), Smith (1983), Misra and Singh (2004)). For diseases with long incubation and infectivity times, the host populations' vital dynamics, i.e., birth and death rates have to be taken into account. This has been shown to change the system behavior qualitatively. While in the classic Kermack and McKendrick's model (Kermack and McKendrick (2006)), the disease dies out or become endemic if the basic reproductive ratio is less or greater than one, respectively. The infection can also become endemic in models incorporating vital dynamics Brauer and Castillo-Chavez (2001). Makinde (2007) used Adomian decomposition method successfully to solve a SIR epidemic model with constant vaccination strategy. In 2010, Tien and Earn (2010) considered a simple extension of the classical SIR model which is globally stable. They have also shown how the parameters viz., reproduction number, epidemic growth rate and final outbreak depend upon the transmission parameter for the different pathways. This has motivated the authors to present a model introducing a new information variable to the existing standard SIR model in fractional order system. To the best of the author's knowledge, this Water-Borne disease model in fractional order system has not yet been done by any researcher.

Fractional order differential equations which are generalizations of classical differential equations (Podlubny (1999)) have gained popularity in the investigation of dynamical systems since they allow greater flexibility in the model and have ability to provide an exact description of different nonlinear phenomena. It has been extensively applied for modelling of many real

problems such as viscoelasticity, power-law phenomenon in fluid mechanics, dielectric polarization, electromagnetic waves, quantum evolution of complex systems, fractional kinetics, etc. and also in biology, ecology, and medical sciences (Saichev and G.M. Zaslavsky (1997), He (1998), Chechkin *et al.* (2003), Das (2009), Das *et al.* (2010), Wu (2011), Das and Kumar (2011), Wu (2012)). In last few decades, fractional order modelling has been an active field of research both from the theoretical and the applied perspectives since they are naturally related to the systems with memory effects which prevail for most of the physical and scientific system models. The fractional order derivative of a function depends on the values of the function over the entire interval. Thus it is suitable for modelling of the systems with long range interaction both in space and time. In general, fractional calculus is considered as a super set of integer order calculus. Sabatier *et al.* (2007) have rightly stated that fractional calculus has the potential to accomplish what integer order calculus can not.

In this chapter the stability analysis of fractional order SIWR model is studied. The intention of the author is to show numerically the variations of considered variables viz., susceptible (S), infected (I) and pathogen concentration compartment (W) due to the introduction of information variable (Z) for different fractional Brownian motions and also for standard motion. The numerical results are depicted through figures for different values of parameters.

# 3.2.2 Mathematical model

Firstly, the fractional order integro differential operator which is the extension of integer order integro differential operator is defined. There are mainly three ways to define fractional integral and derivative namely GrÄunwald-Letnikov derivative, Riemann Liouville Fractional derivative and

the Caputo derivative. In the present article, the Caputo's definition is used which has the advantage to deal with initial value problems. The Caputo derivative of  $\alpha$  order is given by

$$D_{t}^{\alpha}f(t) = \frac{1}{\Gamma(1-\alpha)} \int_{0}^{t} \frac{f'(s)}{(t-s)^{\alpha}} ds, \qquad 0 < \alpha \le 1.$$
(3.2.1)

Fractional calculus is more suitable in describing complex adaptive system since they naturally represent fractals, memory and non locality effects.

Let us consider the fractional SIR model with the assumption of constant population size and a compartment W that measures pathogen concentration in a water source. Infectious agents have a number of options for their transmission. In general these are either by contact with infected individuals or spread via contamination of the water. Infected individuals can in turn contaminate the water compartment by shedding the pathogen into water source. Thus an infected individual originates secondary infections in two ways: through direct contact with susceptible individuals and by shedding the pathogen into the water compartment which susceptible individuals subsequently come into contact with it. The following differential equations are used to describe the Water-bone disease dynamics in fractional order system.

$$D_{t}^{\alpha}S = \mu - \beta_{W}WS - \beta_{I}SI - \mu S$$

$$D_{t}^{\alpha}I = \beta_{W}WS + \beta_{I}SI - \mu I - \gamma I$$

$$D_{t}^{\alpha}R = \gamma I - \mu R$$

$$D_{t}^{\alpha}W = \xi(I - W),$$
(3.2.2)

where *S*, *I* and *R* are respectively the densities of susceptible, infected and recovered within the population, *W* represents the pathogen concentration in water reservoir and the parameters viz.,  $\beta_W$  and  $\beta_I$  are the transmission rate

parameters for person-water-person and person-to-person contact respectively,  $\mu$  is birth and non-disease-related death rate,  $\gamma$  represents disease induced death rate of human population and  $\xi$  shows the decay rate of pathogen in the water.

Let us introduce a new variable Z,

$$Z(t) = \int_{-\infty}^{t} g(S(\tau), I(\tau)) K(t - \tau) d\tau, \qquad (3.2.3)$$

known as information variable (Buonomo *et al.* (2008), D'Onofrio *et al.* (2007a, (2007), Kar and Mondal (2011)), which summarizes information about current values of state variables and past values of state variables. Here  $K(t - \tau)$  is the delaying kernel,  $\tau$  is the distributed delay with  $\tau \leq t$ . Assuming that  $g(S(\tau), I(\tau)) = S(t)$  and  $K(t - \tau) = \frac{1}{T}(-\frac{1}{T}\exp(t - \tau))$ , where *T* is the average delay of the collected information on the disease, as well as the average length of the historical memory concerning the disease, the model (3.2.2) reduces to

$$D_{t}^{\alpha}S = \mu - \beta_{W}WS - \beta_{I}SI - \mu S$$

$$D_{t}^{\alpha}I = \beta_{W}WZ + \beta_{I}ZI - \mu I - \gamma I$$

$$D_{t}^{\alpha}R = \gamma I - \mu R$$

$$D_{t}^{\alpha}W = \xi(I - W)$$

$$D_{t}^{\alpha}Z = \frac{1}{T}(S - Z).$$
(3.2.4)

The third equation of system (3.2.4) can be ignored since here the dynamics of R depends only on the dynamics of I. Therefore, we will concentrate on the following fractional order nonlinear system

$$D_{t}^{\alpha}S = \mu - \beta_{W}WS - \beta_{I}SI - \mu S$$
  

$$D_{t}^{\alpha}I = \beta_{W}WZ + \beta_{I}ZI - \mu I - \gamma I$$
  

$$D_{t}^{\alpha}W = \xi(I - W)$$
  

$$D_{t}^{\alpha}Z = \frac{1}{T}(S - Z).$$
(3.2.5)

# 3.2.3 Equilibrium points and their asymptotic stability

To evaluate the equilibrium points, let

$$D_t^{\ \alpha} S = 0, \ D_t^{\ \alpha} I = 0, \ D_t^{\ \alpha} W = 0, \ D_t^{\ \alpha} Z = 0.$$
 (3.2.6)

The system (3.2.4) has disease free equilibrium at  $E_0 = (1, 0, 0, 1)$  and endemic equilibrium at point  $E_1 = (\overline{S}, \overline{I}, \overline{W}, \overline{Z})$ , where

$$\overline{S} = \frac{\mu + \gamma}{\beta_W + \beta_I}$$
$$\overline{I} = \mu(\frac{1}{\mu + \gamma} - \frac{1}{\beta_W + \beta_I})$$
$$\overline{W} = \mu(\frac{1}{\mu + \gamma} - \frac{1}{\beta_W + \beta_I})$$
$$\overline{Z} = \frac{\mu + \gamma}{\beta_W + \beta_I}.$$

Jacobian Matrix of the system (3.2.5) is given by

$$J = \begin{pmatrix} -(\mu + \beta_{W}W + \beta_{I}I) & -\beta_{I}S & -\beta_{W}S & 0\\ 0 & \beta_{I}Z - \mu - \gamma & \beta_{W}Z & (\beta_{W}W + \beta_{I}I)\\ 0 & \xi & -\xi & 0\\ \frac{1}{T} & 0 & 0 & -\frac{1}{T} \end{pmatrix}.$$
 (3.2.7)

The eigenvalues of these equilibrium points can be calculated by solving the characteristic equation

$$P(\lambda) = \lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4 = 0,$$

where the coefficients can be determined as

$$\begin{split} a_{1} &= \mu + \beta_{W}W + \beta_{I}I - \beta_{I}Z + \mu + \gamma + \xi + \frac{1}{T}, \\ a_{2} &= -(\mu + \beta_{W}W + \beta_{I}I)(\beta_{I}Z - \mu - \gamma) - \xi(\beta_{I}Z - \mu - \gamma) + \xi(\mu + \beta_{W}W + \beta_{I}I) - \beta_{W}Z\xi \\ &- \frac{1}{T}(-\mu - \beta_{W}W - \beta_{I}I + \beta_{I}Z - \mu - \gamma - \xi), \\ a_{3} &= -(\mu + \beta_{W}W + \beta_{I}I)(\beta_{I}Z - \mu - \gamma)\xi - (\mu + \beta_{W}W + \beta_{I}I)\xi\beta_{W}Z - (\beta_{W}W + \beta_{I}I)\beta_{I}S\frac{1}{T} \\ &+ \frac{1}{T}(-(\mu + \beta_{W}W + \beta_{I}I)(\beta_{I}Z - \mu - \gamma) - (\mu + \beta_{W}W + \beta_{I}I)\xi + (\mu + \beta_{W}W + \beta_{I}I)\xi - \beta_{W}Z\xi), \\ a_{4} &= \frac{1}{T}(\mu + \beta_{W}W + \beta_{I}I)(-(\beta_{I}Z - \mu - \gamma)\xi - \beta_{W}Z\xi) + \frac{1}{T}(\beta_{W}W + \beta_{I}I)(\xi\beta_{I}S + \xi\beta_{W}S). \end{split}$$

The equilibrium point is locally asymptotically stable if all the eigenvalues  $\lambda$  of Jacobian matrix *J*(*E*) satisfy the following condition (Matignon (1996))

$$\left|\arg(\lambda)\right| > \frac{\alpha\pi}{2}.$$
 (3.2.9)

Now we discuss the local stability of fractional order water borne system for both the disease free and endemic steady state. For the disease free equilibrium at  $E_0 = (1, 0, 0, 1)$ , the characteristic equation is given by

$$(\frac{1}{T}+\lambda)(\mu+\lambda)(\lambda^{2}+\lambda(\gamma+\mu+\xi-\beta_{I})-\xi(\beta_{W}+\beta_{I}-\gamma-\mu)).$$
(3.2.10)

Thus all the eigenvalues for disease free equilibrium are

$$\begin{split} \lambda_1 &= -\frac{1}{T} \\ \lambda_2 &= -\mu \\ \lambda_{3,4} &= \frac{1}{2} \left( -(\gamma + \mu + \xi - \beta_I) \pm \sqrt{(\gamma + \mu + \xi - \beta_I)^2 + 4\xi (\beta_W + \beta_I - \gamma - \mu)} \right). \end{split}$$

From the above results, it is clear that for each value of  $\alpha$ , disease free equilibrium is locally asymptotically stability for  $R_0 < 1$  and  $\mu > 0$ , where  $R_0 = \frac{\beta_W + \beta_I}{\gamma + \mu}$  is basic reproduction number. So it can be concluded that if

 $R_0 < 1$ , then the disease will die out. Finally, if  $R_0 > 1$ , the disease free equilibrium is unstable and the disease persists in the population and becomes endemic.

At endemic equilibrium point  $E_1 = (\overline{S}, \overline{I}, \overline{W}, \overline{Z})$  with the parameter values  $\mu = 1, \beta_W = 0.6217, \beta_I = 0.6217, \gamma = 0.1340, \xi = 0.333, T = 0.7$ , the Jacobian matrix J(E) has all the eigenvalues with negative real part (-1.57394, - 0.906396±0.179585*i*, -0.0400016). Thus we can conclude from the result of Matignon that the endemic equilibrium at point  $E_1 = (\overline{S}, \overline{I}, \overline{W}, \overline{Z})$  is asymptotically stable for every  $0 < \alpha \le 1$ .

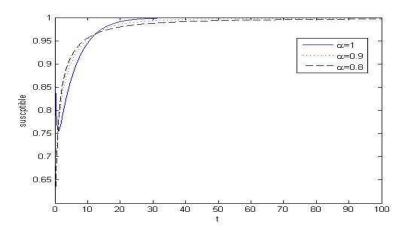
### 3.2.4 Numerical results and discussion

In this section numerical results are carried out using Adams-Bashforth-Moulton algorithm (Diethelm *et al.* (2004a, 2004)) to find the probability density functions of susceptible, infected populations for different fractional Brownian motions  $\alpha = 0.8, 0.9$ , for the standard motion  $\alpha = 1$  for various values of the parameters  $\mu = 1, 2$ ,  $\beta_W = 0.6217$ ,  $\beta_I = 0.6217$ ,  $\gamma = 0.1340$ ,  $\xi = 0.333$ , T = 0.7 and  $\alpha = 0.8, 0.9, 1$  in Fig. 3.2.1 and Fig. 3.2.2.

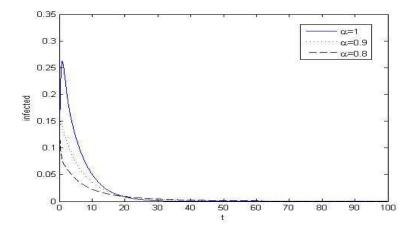
Figs. 3.2.1 – 3.2.2 illustrate the typical behavior of susceptible population, infected population and pathogen concentration in water reservoir. If  $R_0 < 1$ , then the levels of infected population and water pathogen concentration will monotonically decrease and ultimately become zero. It is clear from the figures that in order to control the disease, we have to reduce reproduction number below than one. It is also observed from the figures that the disease will take more time to die out as the order of the fractional derivatives decreases.

In case when some population are infected and pathogen concentration is present in water reservoir, the infected population and water pathogen concentration will monotonically decrease and the trajectories of system approach to steady state and disease become stable after a certain time, which are clearly observed from Fig. 3.2.2. The effect of fractional order exhibits the fact that the realistic biphasic decline behavior of model but at a slower rate.

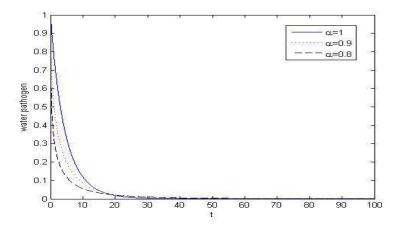
Therefore it is clear from graphical presentations that even for fractional orders the considered model is similar to classical epidemiological model having two steady states where for uninfected steady state, infected population and pathogen concentration in water reservoir disappear but for infected one i.e., for endemically infected steady state it becomes different where susceptible, infected population and pathogen concentration density functions of the model are maintained.



(a) The trajectories of susceptible with respect to time *t* at the equilibrium point  $E_0$  for the fractional order  $\alpha = 0.8, 0.9, 1$ .

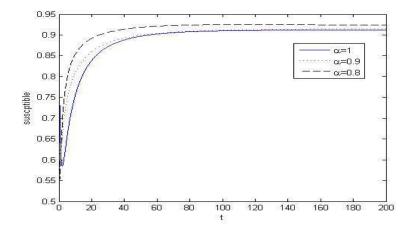


(b) The trajectories of infected with respect to time *t* at the equilibrium point  $E_0$  for the fractional order  $\alpha = 0.8, 0.9, 1$ .

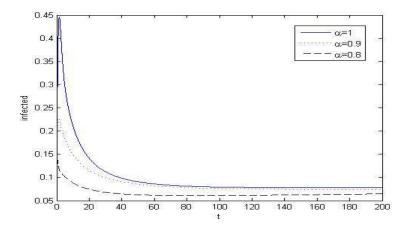


(c) The trajectories of pathogen concentration with respect to time *t* at the equilibrium point  $E_0$  for the fractional order  $\alpha = 0.8, 0.9, 1$ .

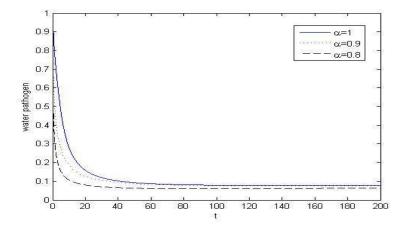
**Fig. 3.2.1.** The trajectories of susceptible, infected and pathogen concentration with respect to time *t* for  $\mu = 2$ ,  $\beta_W = 0.6217$ ,  $\beta_I = 0.6217$ , and  $\alpha = 0.8, 0.9, 1$ .



(a) The trajectories of susceptible with respect to time *t* at the equilibrium point  $E_0$  for the fractional order  $\alpha = 0.8, 0.9, 1$ .



(b) The trajectories of infected with respect to time *t* at the equilibrium point  $E_0$  for the fractional order  $\alpha = 0.8, 0.9, 1$ .



(c) The trajectories of pathogen concentration with respect to time *t* at the equilibrium point E<sub>0</sub> for the fractional order  $\alpha = 0.8, 0.9, 1$ .

**Fig. 3.2.2.** The trajectories of susceptible, infected and pathogen concentration with respect to time *t* for  $\mu = 1$ ,  $\beta_W = 0.6217$ ,  $\beta_I = 0.6217$ ,  $\gamma = 0.1340$ ,  $\xi = 0.333$ , T = 0.7 and  $\alpha = 0.8, 0.9, 1$ .

### 3.2.5 Conclusions

Three important goals are obtained during the present study. First one is the modification of a simple ODE Water-borne model suggested by Tien and Earn (2010) into a system of fractional order differential equations. Second one is the stability analysis of the fractional order water borne disease model with a new information variable. It is shown that the evolution model exhibits two equilibria, namely, disease free equilibrium and the endemic equilibrium points. The disease free equilibrium point is locally asymptotically stable for  $R_0 < 1$  and for  $R_0 > 1$ , a unique endemic equilibrium point exists and is asymptotically stable under certain conditions of the parameters. The most remarkable part of the study is the analysis of time requirements for the stability of the different population density functions as the system approaches to the standard order from the fractional order.