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Fractional Order SIR Model of Buruli Ulcer Disease

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Abstract: Mycobacterium ulcerans (MU) has been recognized to be the cause of Buruli ulcer (BU). The association between the ulcer and environmental exposure is identified as a potential factor of spreading BU. The invariant region of the model is determined. In this paper, we explored the power of fractional order in BU SIR model. We applied the Adams-Bashforth predictor corrector method to the proposed model. Numerical simulations are presented to illustrate the benefit of introducing a fractional model.
Keywords: Buruli ulcer; Invariant region; Adams- Bashforth predictor corrector method; Fractional differential.

1. Introduction

Buruli ulcer(BU) disease is caused by infection with the environmental pathogen *Mycobacterium ulcerans* (M. ulcerans) and largely affects the skin, often progressing without pain or fever to the patient [1]. The possibility of BU being transmitted from person-to-person is not properly investigated, however infection is based on direct or indirect contact with *M. ulcerans* in the environment. Studies have commonly implicated *M. ulcerans* with aquatic environments [2, 3]; however, little is available on the ecology of the pathogen and its geographical distribution in the environment [4]. Portaels, *et al.* [5] proposed an interesting hypothesis for a possible mode of transmission to humans through water – filtering hosts (fish, mollusks) that cluster the *MU* bacteria available in water or mud and then discharge them again to the environment. They are then ingested by aquatic predators including water bugs which eventually transmit the disease through biting of humans [6]. The temperate and tropical environments provide the best condition for water bugs to strive in freshwater. They prey, based on their size, on snails, small fishes, mollusks and larvae of some insects they obtain with their raptorial front legs and bite using their rostrum. Studies conducted in West Africa and other places have associated certain aquatic insects as probable candidate in the transmission of *MU* from a natural source to humans [7, 8]. In the endemic areas in Ghana, water bugs are in abundance particularly in swamps and rivers in which human activities including farming, fishing, bathing occur [6]. The effect of BU on affected communities is disturbing since BU brings about permanent disabilities relatively over 25% of its victims [9, 10].

Mathematical modeling in epidemiology offers new phase in understanding the spread of diseases, and it gives suggestions how disease should be controlled [11]. A well-constructed mathematical model is capable of providing a deeper insight into the process of disease transmission [12, 13]. Fractional calculus provides a broad frame work in modeling technique in the context of epidemiology. For some recent work on fractional differential equations (see [14, 15][. Currently, it has been established that several phenomena in diverse fields can be explained successfully by the models applying fractional order differential equations [16]. Main assertion is that a fractional model can bring about a more realistic explanation to natural phenomena.

In this paper, we take into account the fractional order SIR model which is connection with the evolution of Buruli ulcer disease in human population. Qualitative dynamics of the model is determined and studied including the basic reproduction number, R_0. We present a detailed analysis for the asymptotic stability of disease-free and positive fixed points. Numerical simulations are provided to authenticate the obtained results.

2. Model Derivation

In recent times, many definitions have come up of fractional derivatives [17, 18]. The Riemann- liouville derivative is possibly considered as most common. This derivative of order α is expressed as

$$ {}_{RL}D^{\alpha}_{0+} f(t) = \frac{1}{\Gamma(n-\alpha)}\left(\frac{d}{dt}\right)^n \int_0^t \frac{f(t)}{(t-s)^{\alpha-n+1}} ds, \quad n = [\alpha]+1, $$

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Where Γ is the gamma function and n represents an integer. Following, Riemann- liouville derivative an attractive definition was introduced by Caputo which is stated as

$$D_t^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^n(s)}{(t-s)^{\alpha-n+1}} ds.$$

The widely used definition is the one by Caputo due to its convenience for initial conditions of the differential equations and widely application of real life situations. The unique characteristic is that Caputo's derivative are in similar form as the integer – order differential equations exhibit and its real life applications.

We state Grunwald- Letnikov (GL) definition as

$${}_{GL}D_t^{\alpha} f(t) = \lim_{h \to 0} h^{-\alpha} \sum_{j=0}^{[(t-\alpha)/h]} (-1)^j \binom{\alpha}{j} f(t-jh)$$

By algebraic manipulation the formula be simplified as

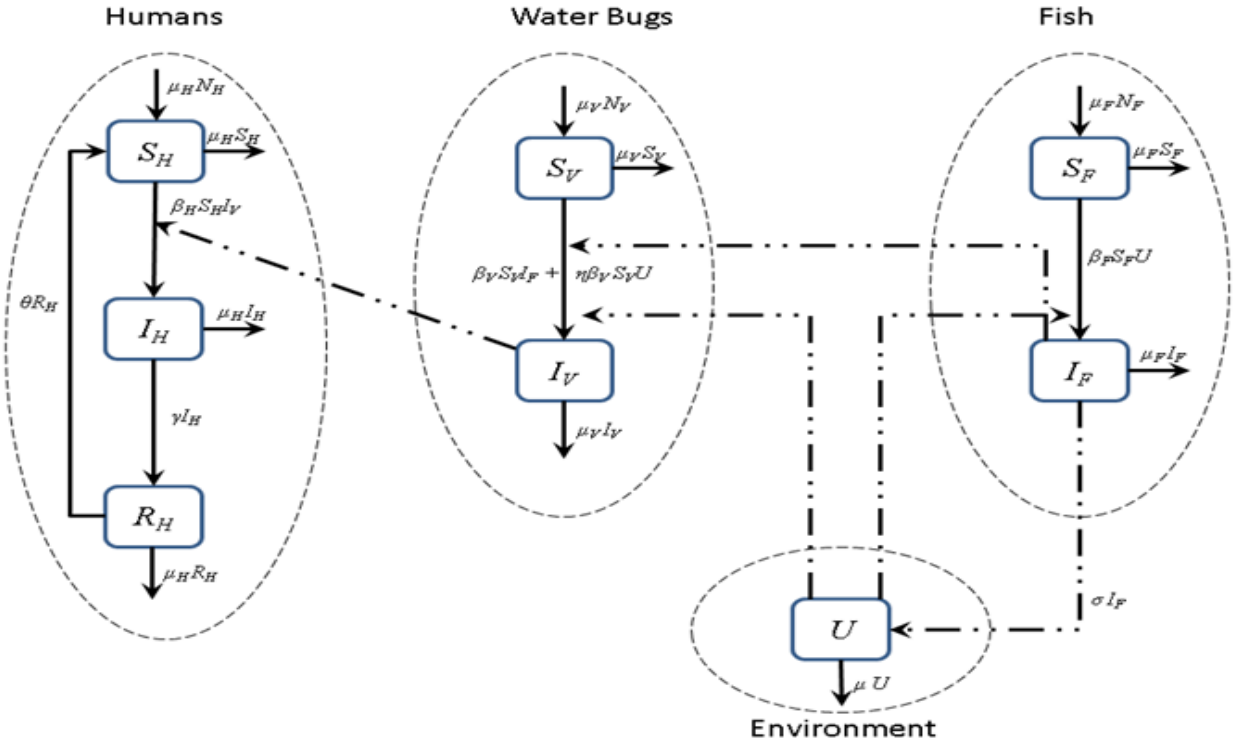
$${}_0D_t^{\alpha} y(t_m) = h^{-\alpha} \sum_{j=0}^{m} w_j^{(\alpha)} y_{m-j,}$$

h is the time step and also w_j^{α} are Grunwald - Letnikov coefficients established as $w_j^{\alpha} = (1-(1+\alpha)/j) w_{j-1,}^{\alpha} \quad j = 0,1,2,...,$ and $w_0^{\alpha} = h^{-\alpha}$. The model that we examine in this paper is a fractional order SIR epidemic model with vertical transmission

Based on the described transmission dynamics of the Buruli ulcer, we take into consideration a constant human population NH(t), the vector population of water bugs NV (t) and the fish population NF (t) at any time t. The total human population NH(t) is divided into three compartments which are susceptible, infectious and recovered, with each size denoted by SH(t), IH(t) and RH(t), respectively. Total population of vector (water bug) at any time t is partitioned into two subclasses which are susceptible water bugs SV (t) and infected water bug IV (t). The total population reservoir of small fish is also partitioned into two compartments of susceptible fish SF (t) and infected fish IF (t). We also take into account the role of the environment and introducing a compartment U that represents the density of *Mycobacterium ulcerans* in the environment.

The possible interrelations between humans, the water bug, fish and density of MU in environment is represented by the schematic diagram Figure 1.

Figure-1. Proposed transmission dynamics of the Buruli ulcer among fish, the water bug and humans.



The dynamics of the ulcer can be explained by the following set of nonlinear differential equations:

$$\left.\begin{aligned}
\frac{dS_H}{dt} &= \mu_H N_H + \theta R_H - \beta_H \frac{S_H I_V}{N_H} - \mu_H S_H, \\
\frac{dI_H}{dt} &= \beta_H \frac{S_H I_V}{N_H} - (\mu_H + \gamma) I_H, \\
\frac{dR_H}{dt} &= \gamma I_H - (\mu_H + \theta) R_H, \\
\frac{dS_V}{dt} &= \mu_V N_V - \beta_V \frac{S_V I_F}{N_V} - \eta\beta_V \frac{S_V U}{K} - \mu_V S_V, \\
\frac{dI_V}{dt} &= \beta_V \frac{S_V I_F}{N_V} + \eta\beta_V \frac{S_V U}{K} - \mu_V I_V, \\
\frac{dS_F}{dt} &= \mu_F N_F - \beta_F \frac{S_F U}{K} - \mu_F S_F, \\
\frac{dI_F}{dt} &= \beta_F \frac{S_F U}{K} - \mu_F I_F, \\
\frac{dU}{dt} &= \sigma I_F - \mu_E U
\end{aligned}\right\} \quad (1)$$

We assume that all the model parameter are positive and the initial conditions of the model system (1) are stated as

$S_H(0) = S_H(0) > 0, I_H(0) = I_H(0) > 0, R_H(0) = R_H(0) > 0$

$S_V(0) = S_V(0) > 0, I_V(0) = I_V(0) > 0, S_F(0) = S_F(0) > 0, I_F(0) = I_F(0) > 0, U(0) > 0$.

We can non-dimensionalise system (1) by letting

$s_h = \frac{S_H}{N_H}, i_h = \frac{I_H}{N_H}, r_h = \frac{R_H}{N_H}, i_v = \frac{I_V}{N_V}, s_f = \frac{S_F}{N_F}, \ u = \frac{U}{K}, m_1 = \frac{N_V}{N_H}$ and $m_2 = \frac{N_F}{N_V}$.

Given that $s_h + i_h + r_h = 1, s_v + i_v = 1, s_f + i_f = 1, 0 \le u \le 1,$ system (1) can be simplified to the following system of equations:

$$\left.\begin{aligned}
\frac{ds_h}{dt} &= (\mu_h + \theta)(1 - s_h) - \theta i_h - m_1\beta_h s_h i_v, \\
\frac{di_h}{dt} &= m_1\beta_h s_h i_v - (\mu_h + \gamma) i_h, \\
\frac{di_v}{dt} &= m_1\beta_v (1 - i_v) i_f + \eta\beta_v (1 - i_v) u - \mu_V i_v, \\
\frac{di_f}{dt} &= \beta_f (1 - i_f) u - \mu_f i_f, \\
\frac{du}{dt} &= \tilde{\sigma} i_f - \mu_e u,
\end{aligned}\right\} \quad (2)$$

Where $\tilde{\sigma} = \frac{\sigma N_F}{K}$. The parameter β_h is the effective contact rate between the infective water bugs (vector) and susceptible humans, β_v is the effective contact rate between infected fish and susceptible water bugs (vectors) and β_f is the effective contact rate between the susceptible fish and *Mycobacterium ulcerans.* The human recovery rate is denoted by γ, θ is the rate of loss of human immunity, σ is also the rate of shedding of *Mycobacterium ulcerans* into the environment. The natural mortality of human, water bugs, fish and MU in environment are denoted by μ_h, μ_v, μ_f and μ_e respectively. K is the environmental carrying capacity of the bacteria population and η is the relative infectivity potential of the fish.

In this paper, we discuss the model comprising fractional system of equations which is obtained by just substituting an integer order derivative by a fractional derivative of order $0 < \alpha \leq 1$.
In recent times, some great break- through have been made to the models of FDEs in diverse area of scientific world. The nonlocal property of FDEs is the most significant property which does not appear to exist in the integer order differential operators. By this property we stipulate that the subsequent state of a model rely not only upon its present state but also upon all of its historical states. Now we establish the presence of fractional order into the ODE model of the system (2). The fractional order introduced into system (2) is described by the following set of fractional order differential equations:

$$\begin{aligned} D_t^\alpha s_h &= (\mu_h + \theta)(1 - s_h) - \theta i_h - m_1 \beta_h s_h i_v, \\ D_t^\alpha i_h &= m_1 \beta_h i_v - (\mu_h + \gamma) i_h, \\ D_t^\alpha i_v &= m_1 \beta_v (1 - i_v) i_f + \eta \beta_v (1 - i_v) u - \mu_V i_v, \\ D_t^\alpha i_f &= \beta_f (1 - i_f) u - \mu_f i_f, \\ D_t^\alpha u &= \tilde{\sigma} i_f - \mu_e u, \end{aligned} \tag{3}$$

where D_t^α is the Caputo fractional derivative. Due to the fact that model (3) examines the dynamics of human populations, all the parameters are presumed to be nonnegative. In addition, it can be proved that all state variables of the model are nonnegative for all time $t \geq 0$ (see, for example, Elbasha, *et al.* [19]).

Lemma 1. *The closed set*

$$\Omega = \left\{ (s_h, i_h, i_v, i_f, u) \in \mathbb{R}_+^5 \,\middle|\, s_h, i_h, i_v, i_f, u \geq 0, s_h + i_h = 1, 0 \leq i_v \leq 1, 0 \leq i_f \leq 1, 0 \leq u \leq \frac{\tilde{\sigma} N_f}{\mu_f} \right\}$$

is positively invariant with respect to model (3)

Proof. The fractional derivative of the total population, obtained by adding all the equations of model (3), is given by

$$D_t^\alpha N(t) = \mu_h - \mu_h N(t) \tag{3.1}$$

The solution to Eq. (3.1) is given by $N\, N(t) = N(0) E_{\alpha,1}(-\mu t^\alpha) + t^\alpha E_{\alpha,1+1}(-\mu t^\alpha)$, where $E_{\alpha,\beta}$ is the Mittag-Leffler function. Therefore, all solutions of the model with initial conditions in Ω remain in Ω for all t > 0. Thus, region Ω is positively invariant with respect to model (3).

3. Numerical Methods and Simulations

In view of the fact that most of the fractional-order differential equations exact analytic solutions cannot be determined, approximation and numerical techniques must be employed. Numerous analytical and numerical methods have been constructed to find solution to fractional order differential equations. For numerical solutions of system (3), one can apply the generalized Adams-Bashforth-Moulton method. In order to obtain the approximate solution by using this algorithm, we take into account the following nonlinear fractional differential equation [20, 21]

$$\begin{aligned} D_t^\alpha y(t) &= f(t, y(t)), \qquad 0 \leq t \leq T, \\ y^{(k)}(0) &= y_0^k, \qquad \text{k=0,1,2,3,....m-1,} \qquad \text{where } m = [\alpha], \end{aligned}$$

This equation is equivalent to the Volterra integral equation

$$y(t) = \sum_{k=0}^{m-1} y_0^{(k)} \frac{t^k}{k!} + \frac{1}{\Gamma(\alpha)} \int_0^t (t - s)^{\alpha - 1} f(s, y(s)) ds \tag{4.1}$$

Diethelm et al. used the predictor-correctors scheme [20, 21], based on the Adams-Bashforth- Moulton algorithm to integrate Eq. (4.1). By applying this scheme to the fractional-order model for childhood diseases, and setting $h = \frac{T}{N}$, $t_n = nh$, $n = 0,1,2,,..., N \in Z^+$, system (3) can be discretized as follows [22, 23]:

$$s_{h(n+1)} = s_{h0} + \frac{h^\alpha}{\Gamma(\alpha+2)}\left((\mu_h+\theta)(1-s^p_{h(n+1)}) - \theta i^p_{h(n+1)} - m_1\beta_h s^p_{h(n+1)} i^p_{v(n+1)}\right) + \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{j=0}^{n} a_{j,n+1}\left((\mu_h+\theta)(1-s_{h_j}) - \theta i_{h_j} - m_1\beta_h s_{h_j} i_{v_j}\right),$$

$$i_{h(n+1)} = i_{h0} + \frac{h^\alpha}{\Gamma(\alpha+2)}\left(m_1\beta_h s^p_{h(n+1)} i^p_{v(n+1)} - (\mu_h+\gamma) i^p_{v(n+1)}\right) + \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{j=0}^{n} a_{j,n+1}\left(m_1\beta_h s_{h_j} i_{v_j} - (\mu_h+\gamma) i_{h_j}\right),$$

$$i_{v(n+1)} = i_{v0} + \frac{h^\alpha}{\Gamma(\alpha+2)}\left(m_1\beta_v(1-i^p_{v(n+1)}) i^p_{f(n+1)} + \eta\beta_v(1-i^p_{v(n+1)}) u^p_{(n+1)} - \mu_V i^p_{v(n+1)}\right) + \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{j=0}^{n} a_{j,n+1}\left(m_1\beta_v(1-i_{v_j}) i_{f_j} + \eta\beta_v(1-i_{v_j}) u_j - \mu_V i_{v_j}\right),$$

$$i_{f(n+1)} = i_{f0} + \frac{h^\alpha}{\Gamma(\alpha+2)}\left(\beta_f(1-i^p_{f(n+1)}) u^p_{(n+1)} - \mu_f i^p_{f(n+1)}\right) + \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{j=0}^{n} a_{j,n+1}\left(\beta_f(1-i_{f_j}) u_j - \mu_f i_{f_j}\right),$$

$$u_{(n+1)} = u_0 + \frac{h^\alpha}{\Gamma(\alpha+2)}\left(\tilde{\sigma} i^p_{f(n+1)} - \mu_e u^p_{(n+1)}\right) + \frac{h^\alpha}{\Gamma(\alpha+2)}\sum_{j=0}^{n} a_{j,n+1}\left(\tilde{\sigma} i_{f_j} - \mu_e u_j\right),$$

Where

$$s^p_{h(n+1)} = s_{h0} + \frac{1}{\Gamma(\alpha)} b_{j,n+1}\left((\mu_h+\theta)(1-s_{h_j}) - \theta i_{h_j} - m_1\beta_h s_{h_j} i_{v_j}\right),$$

$$i^p_{h(n+1)} = i_{h0} + \frac{1}{\Gamma(\alpha)} b_{j,n+1}\left(m_1\beta_h s_{h_j} i_{v_j} - (\mu_h+\gamma) i_{h_j}\right),$$

$$i^p_{v(n+1)} = i_{v0} + \frac{1}{\Gamma(\alpha)} b_{j,n+1}\left(m_1\beta_v(1-i_{v_j}) i_{f_j} + \eta\beta_v(1-i_{v_j}) u_j - \mu_V i_{v_j}\right),$$

$$i^p_{f(n+1)} = i_{f0} + \frac{1}{\Gamma(\alpha)} b_{j,n+1}\left(\beta_f(1-i_{f_j}) u_j - \mu_f i_{f_j}\right),$$

$$u_{(n+1)} = u_0 + \frac{1}{\Gamma(\alpha)} b_{j,n+1}\left(\tilde{\sigma} i_{f_j} - \mu_e u_j\right)$$

$$a_{j,n+1} \begin{cases} n^{\alpha+1} - (n-\alpha)(n+1), & j = 0 \\ (n-j+2)^{\alpha+1} + (n-j)^{\alpha+1} - 2(n-j+1)^{\alpha+1} & 1 \le j \le n, \\ 1 & j = n+1, \end{cases}$$

$$b_{j,n+1} = \frac{h^\alpha}{\alpha}\left((n-j+1)^\alpha - (n-j)^\alpha\right),\ 0 \le j \le n.$$

4. Discussion

In this paper, we take into account the fractional order model for Buruli ulcer. We have determined a stability condition for both disease free and endemic equilibrium. We have also presented a numerical example and validated our results. One should observe that although the disease free and endemic equilibrium are identical for integer order and fractional order models, the solution of the fractional order model incline to the fixed point over a longer period of time during simulations. Mention should be made again that when considering real life issues, the order of the system can be obtained by utilizing the collected data. The conversion of a classical model into a fractional one leads it to be very sensitive to the order of differentiation α : a very small alter in α may result in a huge change in the final result.

Following [24], Figure 2 indicates that s_h drops substantially in a relatively small period of time. Figure 3 depicts that a small change in α exhibits a vast change in the result. In Figure 4, 5 and 6 also show similar results that even a simple fractional model would lead to better result. Figure 7 shows $i_h(t), i_v(t), i_f(t)$ of model (3) at α.=1, and they form the infective compartments together with the environment.

Figure-2. $S_h(t)$ for $\alpha = 1.0, 0.9, 0.8$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95;$ $m_2 = 0.95; \beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$

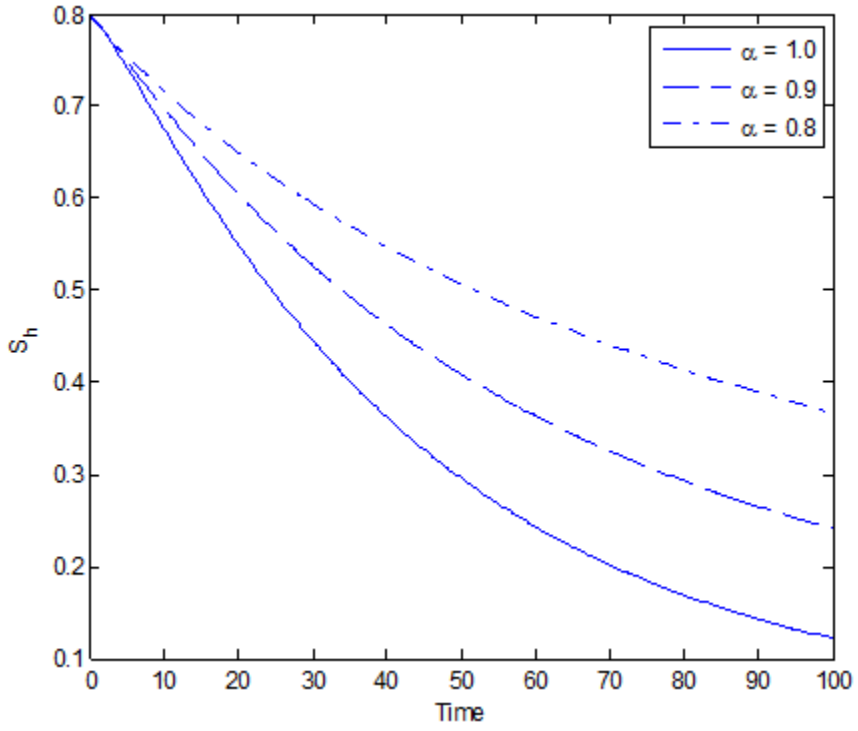


Figure-3. $i_h(t)$ for $\alpha = 1.0, 0.9, 0.8$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95;$ $m_2 = 0.95; \beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$

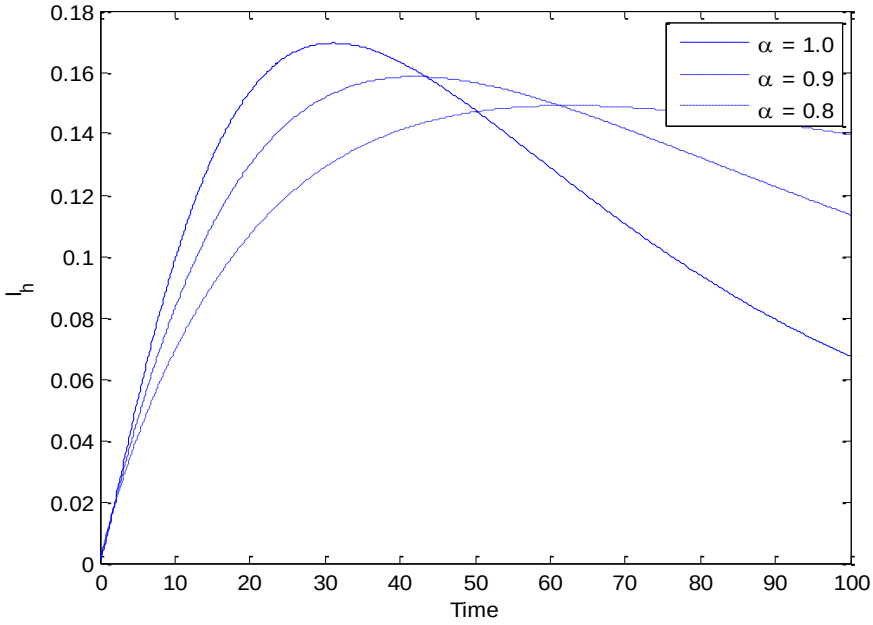


Figure-4. $i_v(t)$ for $\alpha = 1.0, 0.9, 0.8$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95;$ $m_2 = 0.95; \beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$

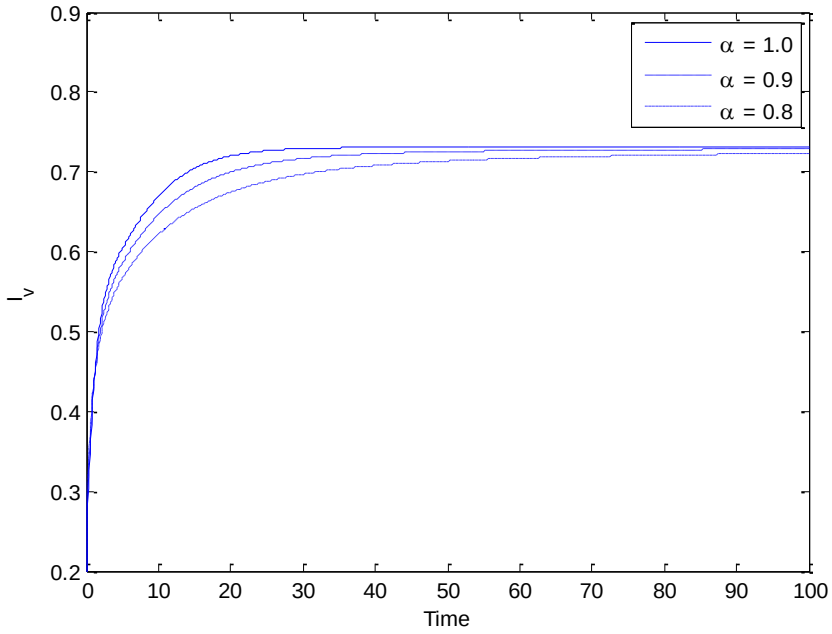


Figure-5. $i_f(t)$ for $\alpha = 1.0, 0.9, 0.8$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95;$ $m_2 = 0.95; \beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$

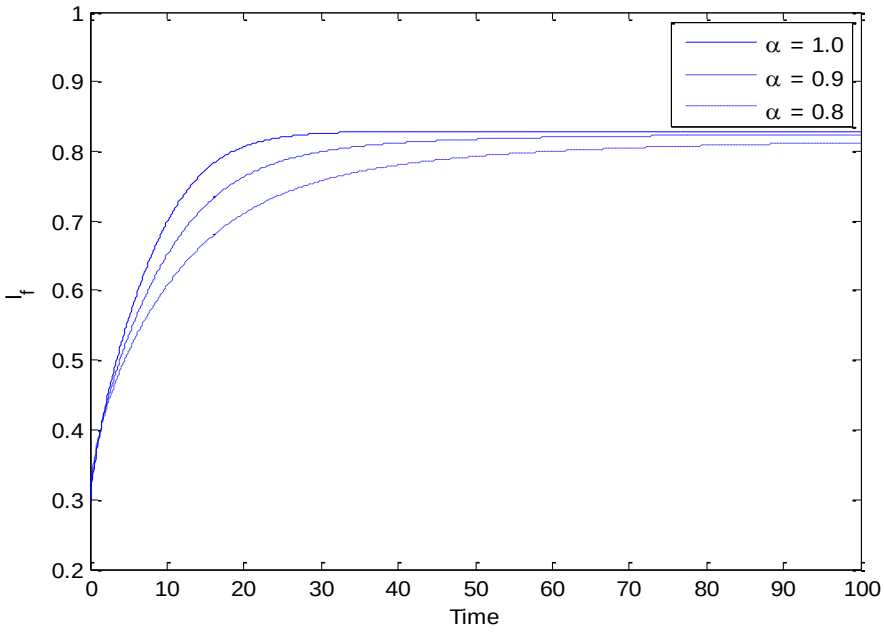


Figure-6. $u(t)$ for $\alpha = 1.0, 0.9, 0.8$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95; m_2 = 0.95$; $\beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$.

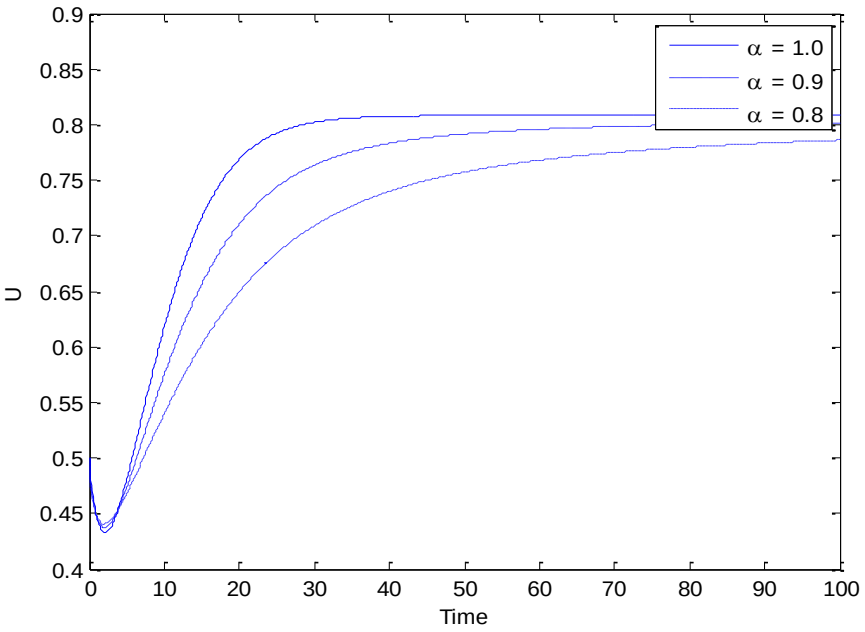
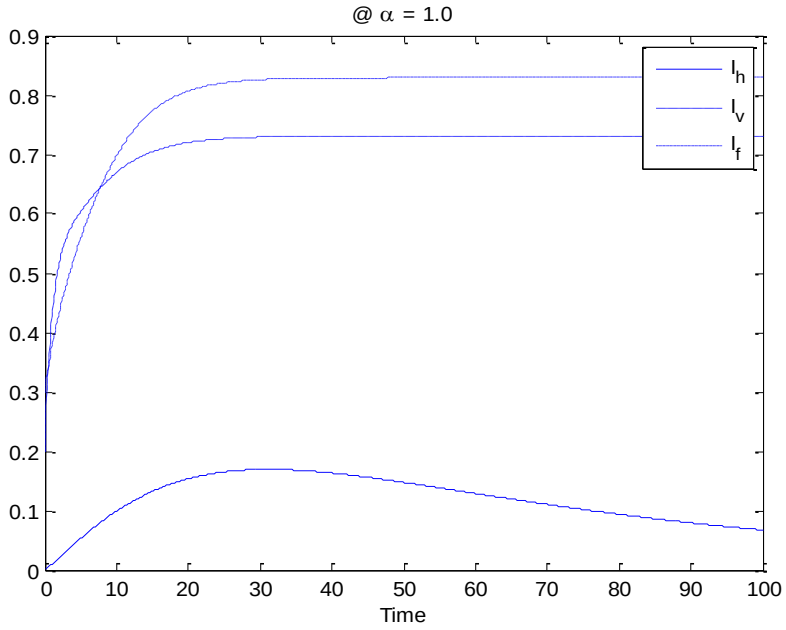


Figure-7. $i_h(t), i_v(t), i_f(t)$ for $\alpha = 1.0$ and $u_h = 4.5\times10^{-5}; \gamma_h = 1.6\times10^{-5}; \theta_h = 1\times10^{-2}; m_1 = 1.95;$ $m_2 = 0.95; \beta_h = 0.75; \beta_v = 0.86; \eta = 2.62; \beta_f = 0.83; u_f = 3\times10^{-4}; u_v = 0.93; \tilde{\sigma} = 0.68; u_v = 0.90$



Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.

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References

[1] World Health Organization, 2007. "Buruli ulcer disease (Mycobacterium ulcerans infection) Fact Sheet Nu199.World Health Organization." Available: http://www.who.int/mediacentre/factsheets/fs199/en/

[2] Brou, T., Broutin, H., Elguero, E., Asse, H., and Guegan, J.-F., 2008. "Landscape diversity related to buruli ulcer disease in Coˆte d'Ivoire." *PLoS Neglected Tropical Diseases*, vol. 2, p. e271. Available: http://dx.plos.org/10.1371/journal.pntd.0000271

[3] Marsollier, L., Severin, T., Aubry, J., Merritt, R. W., and Saint, A. J. P., 2004. "Aquatic snails, passive hosts of Mycobacterium ulcerans." *Applied and Environmental Microbiology*, vol. 70, pp. 62-96.

[4] Merritt, R. W., Walker, E. D., Small, P. L. C., Wallace, J. R., and Johnson, P. D. R., 2010. "Ecology and transmission of buruli ulcer disease: A systematic review." *PLoS Neglected Tropical Diseases*, vol. 4, p. e911. Available: http://dx.plos.org/10.1371/journal.pntd.0000911

[5] Portaels, F., Chemlal, K., Elsen, P., Johnson, P. D., Hayman, J. A., Hibble, J., Kirkwood, R., and Meyers, W. M., 2001. "Mycobacterium ulcerans in wild animals." *Rev. Sci. Tech. Off. Int. Epizoot.*, vol. 20, pp. 252-264.

[6] Silva, M. T., Portaels, F., and Pedrosa, J., 2007. "Aquatic insects and Mycobacterium ulcerans: An association relevant to buruli ulcer control?" *PLoS Med*, vol. 4, p. e63.

[7] Marsollier, L., Robert, R., Aubry, J., Andre, J. S., Kouakou, H., Legras, P., Ma, A., Mahaza, C., and Carbonelle, B., 2002. "Aquatic insects as a vector for macobacterium ulcerans." *Applied and Environmental Microbiology*, vol. 68, pp. 4623–4628.

[8] Stinear, T. P., Hong, H., Frigui, W., Pryor, M. J., Brosch, R., Garnier, T., Leadley, P. F., and Cole, S. T., 2005. "Common evolutionary origin for the unstable virurence plasmid PMUM found in geographically diverse strains of Mycobacterium ulcerans." *Journal of Bacteriology*, vol. 18, pp. 1668-1676.

[9] Dobo, K. M., Spotts, E. A., Marston, B. J., Horsburgh, C. R. J., and King, H. K., 2000. "Serologic response to culture filtrate antigens of Mycobacterium ulcerans during buruli ulcer disease." *Emerging Infectious Diseases*, vol. 6, pp. 158-164.

[10] Horsburgh, C. R. J. and Meyers, W. M., 1997. *Buruli Ulcer. In: C.R. Horsburgh Jr. and A.M. Nelson (Eds.) Pathology of emerging infections*. Washington: American Society for Microbiology Press. pp. 119-126.

[11] Brauer, F. and Castillo-Chavez, C., 2001. *Mathematical models in population biology and epidemiology*. New York: Springer-Verlag.

[12] Thom´e, R. C., Yang, H. M., and Esteva, L., 2010. "Optimal control of Aedes aegypti mosquitoes by the sterile insect technique and insecticide." *Math. Biosci.*, vol. 223, pp. 12–23.

[13] Otero, M., Schweigmann, N., and Solari, H. G., 2008. " A stochastic spatial dynamical model for Aedes aegypti." *Bull. Math. Biol.*, vol. 70, pp. 1297-1325.

[14] Houben, R. M., Dowdy, D. W., and Vassall, A., 2014. "How can mathematical models advance tuberculosis control in high HIV prevalence settings?" *Int J Tuber Lung Dis.*, vol. 18, pp. 509-514.

[15] Huppert, A. and Katriel, G., 2013. "Mathematical modelling and prediction in infectious disease epidemiology." *Clin Microbiol Infect*, vol. 19, pp. 999-1005.

[16] Ahmed, E., El-Sayed, A. M. A., and El-Saka, H. A. A., 2006. "On some Routh–Hurwitz conditions for fractional order differential equations and their applications in Lorenz, R¨ossler, Chua and Chen systems." *Physics Letters*, vol. A 358, pp. 1–4.

[17] Kilbas, A. A., Srivastava, H. M., and Trujillo, J. J., 2006. "Theory and applications of fractional differential equations." Available: https://www.elsevier.com/books/theory-and-applications-of-fractional-differential-equations/kilbas/978-0-444-51832-3

[18] Podlubny, 1999. *Fractional differential equations*. New York: Academic Press.

[19] Elbasha, E. H., Podder, C. N., and Gumel, A. B., 2011. "Analyzing the dynamics of an SIRS vaccination model with waning natural and vaccine-induced immunity." *Nonlinear Analysis*, vol. 12, pp. 2692-2705.

[20] Diethelm, K. and Ford, N. J., 2002. "Analysis of fractional differential equations." *J Math Anal Appl*, vol. 256, pp. 229–248.

[21] Diethelm, K., Ford, N. J., and Freed, A. D., 2003. "A predictor-corrector approach for the numerical solution of fractional differential equations." *Nonlinear Dyn*, vol. 29, pp. 3-22.

[22] Diethelm, K., 2013. "A fractional calculus based model for the simulation of an outbreak of dengue fever." *Nonlinear Dyn*, vol. 71, pp. 613–619.

[23] Li, C. C. and Tao, C., 2009. "On the fractional Adams method." *Computers and Mathematics with Applications*, vol. 58, pp. 1573-1588.

[24] Soewono, E. and Supriatna, A. K., 2001. "ATwo-dimensionalmodel for transmission of dengue fever disease." *Bulletin of the Malaysian Mathematical Sciences Society*, vol. 24, pp. 48-57.