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Research paper

Fractional order models of infectious diseases: a review

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Abstract

The aim of this paper is to present a succinct review on fractional order models of infectious diseases. Fractional order derivative is a potential tool which gives a better understanding of the impact of memory on spread of infectious diseases. This paper reviews different infectious diseases models with constant, variable or complex fractional order. Fractional order models with time delay are presented in this paper as well. We argue that, such models are essential for decision makers in health organizations.

Keywords: *Constant/Variable Fractional Order Models-Models with Complex Fractional Order -Fractional Order Models with Time Delay-Infectious Diseases Models with Memory.*

1. Introduction

Infectious diseases and epidemics have become one of the crucial global issues as they cause of death and disability not only in developing countries, but also worldwide. Every year, HIV, TB and malaria cause 10% of all deaths [37], [28]. Over the last few decades, the number of infectious diseases outbreaks has increased dramatically, resulting in economic crises and millions of disability and deaths [48], [61]. For example, West Africa suffered up to $32 billion loss by 2015 and more than 11,000 deaths during Ebola outbreak [60], [66].
Infectious diseases are spreading around the globe faster than ever before, and new diseases are emerging at a high rate [20]. Speed modern transportation has helped spread of some communicable diseases. International travelling and commerce drive the rapid, global distribution of microbial pathogens and the organisms that harbor them [45]. Recently, the Zika virus is now spreading explosively in the world through modern transportations. There is no, no background immunity in the population, or vaccine currently available [70]. Outbreaks provide an opportunity to collect and analyze initial data. These gathered data are essential to predict the behavior of diseases and to adjust control strategies. But sometimes data collection is impossible due pathological limitations [37], [40], [54]. Also, testing spread of infectious diseases in human societies are unethical or needs big budgets [39], [40], [54]. So, mathematical models of infectious diseases should be used to give a better understanding of spread of diseases spread in human communities [36] and to predict crucial data that should be collected. It is worth mentioning that, mathematical modeling of diseases extends to ancient history. One of the earliest mathematical models in epidemiology was presented by Daniel Bernoulli (1700–1782) [50], [22]. He predicted the impact of immunity with smallpox disease that made the idea of eradication feasible [50], [22]. Nowadays, modern mathematical models of infectious diseases play an increasingly significant role to evaluate the potential impact of eradication and control programs in reducing morbidity and mortality [23], [25], [62], [46]. Such enormous models are significant to link between clinical data for selected subpopulations and population-level used [30]. Mathematical modelling can achieve a better understanding of the indirect protection provided by immunization [29]. For example, the spread of influenza virus in USA were simulated in 2009 from gathered data from different areas in [29]. Gathered data from the H1N1 epidemic have been used to approve SEIR mathematical model. Also, an estimate for the vaccination coverage needed to block the spread of infectious diseases can be obtained from such models after estimating its parameters from available medical and epidemiological data [29].
Mathematical models enable researchers to understand the development of drug resistance throughout therapy [47].
Although many of these models have been proposed in literature, it has been restricted to integer order models [17]. However, integer order systems do not convey any information about prior states [4-11]. Understanding the concept of memory in biological systems can be very essential to predict the future of infectious diseases outbreaks and to control infectious diseases. Studying immunological memory is essential to develop vaccines. Memory and learning process in vector and host are critical in vector-borne disease transmission like Malaria and dengue fever [56]. Learning behavior and memory in vectors like mosquitoes are important in vector borne diseases transmission [56]. Mosquitoes' experience is considered as the ability for mosquitoes to accurately identify their hosts. Also memory and learning behavior is significant in immunological memory which is defined as the potential of the immune system to respond more effectively to threats that have been encountered previously [38]. Fractional order differential equations can be potential flexible tools for modelling epidemiological and biological systems related with memory.
Adding fractional-order parameter enhances the system potential as it adds a new degree of freedom which leads the model to more space [17], [19]. So, the fractional order is supposed to be the memory index [21]. However, if the fractional order value tends to unity, the system

will have short memory dependence. Otherwise, if the fractional order approaches zero, so the system depends strongly on the previous states [28], [38]. So, of fractional-order models are more logical than the integer model [1], [5], [6].
In this paper, we summarize different recent fractional order models of infectious diseases including constant & variable fractional order models, fractional delay models, and fractional complex order models.
The rest of the paper is organized as follows: In section two, some basic definitions of the fractional calculus are presented. Section three introduces discussion about fractional order models of immune system while section four is devoted for the epidemic models with memory. Section five displays the role of mathematical models of with memory of Human immunodeficiency virus based on fractional order models. Two models are discussed in this section; the first is a constant fractional order model of HIV infection of $CD4^+$ T-cells, while the second model is a fractional complex-order model drug-resistance in HIV disease. A study on fractional order models of vector borne diseases is presented in section six. Section seven is devoted for Fractional variable-order model of multi-strain tuberculosis (TB). Fractional order model with time delay of HIV infection.

2. Preliminaries

Firstly, basic definitions of constant & variable fractional-order integration and fractional-order differentiation will be discussed [3], [6], [7], [9], [51], [52], [57], [58].

Definition 1: *The fractional order integral of order of a function $f: R^+ \to R$ is given by*

$$J^{\alpha} f(x) = \frac{1}{\Gamma(\alpha)} \int_0^x (x-t)^{\alpha-1} f(t)\, dt, \alpha > 0, x > 0,$$

Where α & $x \in (0, \infty]$
This integral is called integral with memory.

Definition 2: *The Caputo and Riemann–Liouville fractional order derivatives of order α are given respectively as follows* [46]*:*

$$D^{\alpha} f(x) = D^m\left(J^{m-\alpha} f(x)\right),$$

$$D_*^{\alpha} f(x) = J^{m-\alpha}\left(D^m f(x)\right),$$

Where

$m - 1 < \alpha \leq m, m \in N.$

Definition 3: *Grunwald-Letnikov fractional order derivative of function $f(t)$ is given as follows* [48]*:*

$${}_a^{GL}D_t^{\alpha} f(x) = \lim_{h \to 0} h^{-\alpha} \sum_{k=0}^{n} (-1)^j \binom{\alpha}{k} f(t - kh)$$

Where $nh = x - a$

The variable-order fractional derivative, is a powerful tool to characterize memory that may vary from point to point [57], [58]. Some of the basic definitions of the variable-order fractional derivative are presented as follows:

Definition 4: *(Riemann–Liouville fractional derivatives of order)*

Let $\alpha(t)$ *be a continuous and bounded function, then Riemann–Liouville variable-order fractional derivative of $f(t)$: $[a, b] \to \mathbb{R}$ is* defined as [57]:

Left Riemann–Liouville derivative of order α(t) is defined by

$${}_a^{RL}D_t^{\alpha(t)} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \frac{d}{dt} \int_a^t (t-\tau)^{-\alpha(t)} f(\tau) d\tau,$$

Where $0 < \alpha(t) \leq 1$

Definition 5: *(Caputo fractional derivatives of order $\alpha(t)$)*

Let α(t) be a continuous and bounded function, then the Caputo variable-order fractional derivative of f(t): $[a, b] \to \mathbb{R}$ is defined as [57], [58]:

Left Caputo derivative of order α(t) is defined by

$${}_a^{c}D_t^{\alpha(t)} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \int_a^t (t-\tau)^{-\alpha(t)} f'(\tau) d\tau,$$

Where $0 < \alpha(t) \leq 1$

Definition 6: *(Grünwald-Letnikov fractional derivatives of order $\alpha(t)$)*
Let α(t) be a continuous and bounded function, then the variable-order fractional derivative of $f(t)$: $[a, b] \to \mathbb{R}$ is defined as [52]:

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

Where h is the step size, $n = \frac{t}{h}$, $[n]$ is the integer part of n and $0 < \alpha(t) \leq 1$.

3. Fractional order models with an immune response

All The immune system plays a vital role in human health as it protects the body from all harmful foreign substances like viruses and bacteria [32]. After defeating an infection, B and T cells create memory cells as they hold information about each hazard that attack the body. This creates the memory of immune system which makes it easier to identify and eliminate in the future [14]. There is no alternative to mathematical modeling to understand the behavior of immune system [39]. Numerous mathematical models have been presented to simulate the immune system [32]. The majority of such models proposed in the literature were integer order models. In [2], the authors presented a generalization of hepatitis C virus (HCV) model based on HCV basic model of Perelson et al [16] as follows

$$\begin{aligned} D^{\alpha}(T) &= s - d\,T - (1-\eta)\beta VT, \\ D^{\alpha}(I) &= (1-\eta)\beta VT - \delta\, I\,(1 - I/c_2), \\ D^{\alpha}(V) &= \left(1-\varepsilon_p\right) pI - cV. \end{aligned} \tag{1}$$

Where *T*, *I* and *V* present respectively uninfected hepatocytes, infected hepatocytes and virus density, and $0 < \alpha \leq 1$ is the index of memory. The rate of production of uninfected hepatocytes is s, while their rate of death is d per cell and rate of infection is β. The rate of loss of infected hepatocytes is δ per cell. The rate of production of virions is p per infected hepatocytes and their rate of clearness is c per virion. ε_p and η present respectively the efficacy of treatment in blocking virion production and reducing new infections. The authors show that "fractional order results show the realistic biphasic decline behavior of HCV but at a slower rate".

In [24], another example to show that fractional order models are more significant to model the immune system than classical integer order models. The authors consider two immune effectors y, z attacking an antigen x. As follows:

$$\begin{aligned} D^{\alpha}(x) &= x - axy - bxz, \\ D^{\alpha}(y) &= -cy + xy, \\ D^{\alpha}(z) &= -ez + xz. \end{aligned} \tag{2}$$

Where a, b, c, e are positive constants and $0 < \alpha \leq 1$.

Equilibrium points of (2) are locally asymptotically stable the condition $|arg\ \lambda_i| > \alpha\frac{\pi}{2}$ is satisfied, where λ_i is the eigenvalue of the Jacobian matrix of the system at the equilibrium. It has been proved in [24] that, fractional order model is either stable as classical integer order model or more stable.

4. Epidemic models with memory

An epidemic occurs when an infectious disease transmitted rapidly to many people than what was expected in a region during a given period. The Flu of 1918 was known as one of the most deadly outbreaks in recorded world history. This flu killed more people than World War I (about 20 million victims) [54]. Recently, World Health Organization (WHO) has declared emergency state for Ebola outbreak in West Africa after thousands of deaths because of the virus in 2014. Mathematical models in epidemiology are able to describe and predict highly dynamic outbreaks. For example, the following model [12] presents a fractional order model of Ebola infection using SEIR model as follows:

$$\begin{aligned} D^{\alpha}(S) &= -\frac{\beta S(t)\left(qE(t)+I(t)\right)}{N}, \\ D^{\alpha}(E) &= \frac{\beta S(t)\left(qE(t)+I(t)\right)}{N} - \delta E(t), \\ D^{\alpha}(I) &= \delta E(t) - \gamma I(t), \\ D^{\alpha}(R) &= \gamma I(t). \end{aligned} \tag{3}$$

Where $0 < \alpha \leq 1$, while *S(t)*, *E(t)*, *I(t)*, and *R(t)* are the susceptible, exposed, infected, removed populations respectively at time t and the number of total population *N = S(t) + E(t) + I(t) + R(t)*. The parameter $\beta = pc$ is average number of infected people, p is the probability of infection, c is the per capita contact rate, $q \in [0, 1]$ and γ is the per-capita death rate. The authors have analyzed the WHO data in order to give an accurate prediction of the outbreak in Liberia, Guinea, and Sierra Leone. They achieved a good approximation to real gathered data.

5. Fractional order models of HIV infection

HIV is one of the most deadly viruses. It causes AIDS and destroys the immune system, so the body cannot defend itself against any threat. There were more than 36 million people living with HIV by the end of 2014 [67]. An estimated 21.8 million people have died of AIDS since the start of the epidemic. Mathematical models provide an essential tool to understand the interactions between HIV and the immune system. Fractional order models are helpful to provide a better understanding of the dynamics of HIV infection [19]. An impressive example has been shown in [19], where the authors have introduced a modified model of fractional order based on the ODE model proposed by Culshaw and Ruan [15] into a system of fractional-order as follows:

$$D^{\alpha}(T) = s - \mu_T T + rT\left(1 - \frac{T+1}{T_{max}}\right) - K_1 VT$$

$$D^{\alpha}(I) = K_1' VT - \mu_I I, \tag{4}$$

$$D^{\alpha}(V) = N\mu_b I - K_1 VT - \mu_V V$$

Where $T(t)$ is the concentration of healthy $CD4^{+}T$-cells at time t, $I(t)$ is the concentration of infected $CD4^{+}T$-cells, and $V(t)$ presents the concentration of free HIV at time t. The parameters $s, \mu_T, r \text{ and } T_{max}$ are the source of $CD4^{+}T$-cells, the natural death rate of $CD4^{+}T$-cells, their growth rate and their carrying capacity respectively. K_1 is the infection rate of T-cells with free virus while K_1' presents the rate at which infected cells become actively infected, μ_I is a blanket death term for infected cells, $\mu_b$is the lytic death rate for infected cells, μ_V is the loss rate of virus and N is the number of viral particles. In [19], it is assumed that $0.5 < \alpha \leq 1$. The authors think that fractional derivatives cannot describe accurately the rate of change in number when $0 < \alpha \leq 0.5$. It has been proven that in [19], there is a unique solution $x(t) = (T, I, V)^t$ for on $t \geq 0$ and the solution will remain in R_{+}^{3}. Furthermore, $T(t)$ and $I(t)$ are all bounded by T_{max} . The authors showed that the model established in this paper possesses non negative solutions, as desired in any population dynamics. A restriction on the number of viral particles released per infectious has been obtained cell in [19], to sustain the infection. Another interesting study about a fractional complex-order model for drug resistance in HIV virus during therapy is presented in [44] as follows:

$$\frac{1}{2}\left(D^{\alpha+\beta j} + D^{\alpha-\beta j}\right) T(t) = \lambda - dT - k_s(1 - n_{rt}^{s})V_s(t)T(t) - k_s(1 - n_{rt}^{s})V_r(t)T(t),$$

$$\frac{1}{2}\left(D^{\alpha+\beta j} + D^{\alpha-\beta j}\right) T_s(t) = (1-u)k_s(1 - n_{rt}^{s})V_s(t)T(t) - \delta T_s(t),$$

$$\frac{1}{2}\left(D^{\alpha+\beta j} + D^{\alpha-\beta j}\right) V_s(t) = N_s\delta(1 - n_P^{s})T_s(t) - cV_s(t) \tag{5}$$

$$\frac{1}{2}\left(D^{\alpha+\beta j} + D^{\alpha-\beta j}\right) T_r(t) = uK_s(1 - n_{rt}^{s})V_s(t)T(t) + K_r(1 - n_{rt}^{r})V_r(t)T(t) - \delta T_r(t)$$

$$\frac{1}{2}\left(D^{\alpha+\beta j} + D^{\alpha-\beta j}\right) V_r(t) = N_r\,\delta(1 - n_P^{r})T_r(t) - cV_r(t)$$

Where T is the density of uninfected $CD4^{+}T$-cells with rate of production λ and rate of death d. The parameter K_s is the infection rate of the $CD4^{+}T$-cells with drug-sensitive HIV viruses V_s. The concentration of drug-sensitive infected $CD4^{+}T$ is $T_s(t)$ while the concentration of the drug-resistant infected $CD4^{+}T$ is T_r. The $CD4^{+}T$ -cells may be infected by drug-resistant viruses V_r at a rate K_r. The efficacy rates of RTI for wild type and mutants are n_{rt}^{s} and n_{rt}^{r} respectively (The proportions of eliminated T_s and T_r cells by RTI). The parameter u is the proportion of T_s cells that can become resistant to the drug. The rate of death of $T_r$and T_s is δ while c is the clearness rate of the viruses. V_s and V_r are produced with bursting sizes drug-sensitive strains $N_s$and N_r respectively. The efficacy of PI for wild type strain and mutants are n_P^{s} and n_P^{r} respectively. Based on Grunwald-Letnikov formulation, the authors simulated the model for different values of the fractional derivative of complex order $D^{\alpha+\beta j}$ where $(\alpha + \beta j)\,\epsilon\, C$. If $\alpha = 1$ and $\beta = 0$, the system will be converted to the integer order model. Applying fractional derivative complex-order to the system, results in complex valued outcomes. The authors fixed $\beta = 0.8$ and $0.5 \leq \alpha \leq 1$. Faster declines in the density of viruses have been observed for decreasing values of α [44]. In other words, increasing memory of T-cells leads to faster decreasing in density of HIV.

6. Fractional order vector borne diseases models

Vector-borne disease is a commonly used term that describes a disease transmitted to people by vectors like mosquitos and ticks through feeding activity [63]. Being a vector means that it carries a disease from one host to another. More than 1 million people die every year from mosquito-borne diseases like Malaria and Dengue Fever [65]. An interesting generalization of the basic deterministic ODE Dengue Fever disease model has been presented in [49]. The authors have modified the integer order model in [41] to be fractional order model as follows

$$\begin{aligned}
D^{\alpha}(S_H) &= \mu_H(K - S_H) - \frac{b\beta_1 S_H I_V}{K}, \\
D^{\alpha}(I_H) &= \frac{b\beta_1 S_H I_V}{K} - (\mu_H + \gamma_H)I_H, \\
D^{\alpha}(R_H) &= \gamma_H I_H - \mu_H R_H, \\
D^{\alpha}(I_V) &= \frac{b\beta_2 I_H S_V}{K} - mI_V, \\
D^{\alpha}(S_V) &= A - \frac{b\beta_2 I_H S_V}{K} - mS_V.
\end{aligned} \tag{6}$$

Where $0 < \alpha \leq 1$, S_H, I_H and R_H are the populations of susceptible humans, infected human, and recovered human respectively. S_V and I_V are the populations of susceptible mosquitos, infected mosquitos. The total human population K at time t is denoted by N_H where $N_H = S_H + I_H + R_H$. The authors considered that $N_V = S_V + I_V$. The parameter μ_H is the per capita mortality rate of the humans and m is the corresponding value for the mosquitoes. γ_H is the humans recovery rate while b is the biting rate. The probabilities of transmission from human to mosquito and vice versa are denoted by β_1 and β_2 respectively while A_1 is the recruitment rate of mosquito. Model (6) has some drawback, because the left-hand side of the system (6) has dimension $(time)^{-\alpha}$ while the right-hand has the dimension $(time)^{-1}$. So it is recommended to use the procedure presented in [18,49]. System (6) can be re-written as follows:

$$
\begin{aligned}
D^{\alpha}(S_H) &= \mu_H^{\alpha}(K - S_H) - \frac{b^{\alpha}\beta_1 S_H I_V}{K},\\
D^{\alpha}(I_H) &= \frac{b^{\alpha}\beta_1 S_H I_V}{K} - (\mu_H^{\alpha} + \gamma_H^{\alpha})I_H,\\
D^{\alpha}(R_H) &= \gamma_H^{\alpha} I_H - \mu_H^{\alpha} R_H,\\
D^{\alpha}(S_V) &= A_2 - \frac{b^{\alpha}\beta_2 I_H S_V}{K} - m^{\alpha} S_V,\\
D^{\alpha}(I_V) &= \frac{b^{\alpha}\beta_2 I_H S_V}{K} - m^{\alpha} I_V.
\end{aligned} \tag{7}
$$

Where $A_2 = \mu_m^{\alpha} \times (S_V(0) + I_V(0))$, and μ_m is the birth rate of mosquito. In [48], two different orders $\alpha \in (0,1]$ and $\beta \in (0,1]$ are introduced to the system (6) to be more sensible, so the new system is as follows:

$$
\begin{aligned}
D^{\alpha}(S_H) &= \mu_H^{\alpha}(K - S_H) - \frac{b^{\alpha}\beta_1 S_H I_V}{K}\\
D^{\alpha}(I_H) &= \frac{b^{\alpha}\beta_1 S_H I_V}{K} - (\mu_H^{\alpha} + \gamma_H^{\alpha})I_H\\
D^{\alpha}(R_H) &= \gamma_H^{\alpha} I_H - \mu_H^{\alpha} R_H,\\
D^{\beta}(S_V) &= A_2 - \frac{b^{\beta}\beta_2 I_H S_V}{K} - m^{\beta} S_V\\
D^{\beta}(I_V) &= \frac{b^{\beta}\beta_2 I_H S_V}{K} - m^{\beta} I_V.
\end{aligned} \tag{8}
$$

The basic reproduction number has been derived in [48] for (6) to be

$$R_0 = \sqrt{\frac{b\beta_1}{m} \times \frac{b\beta_2 A}{mK(\mu_H + \gamma_H)}} \tag{9}$$

The infection persists if $R_0 > 1$, and dies out if $R_0 < 1$. Using the next generation matrix approach, the basic reproduction numbers R_0, $\bar{R}_0$and $\bar{\bar{R}}_0$ have been derived for systems (7) and (8) respectively in [49]to be:

$$\bar{R}_0 = \sqrt{\frac{b^{\alpha}\beta_1}{m^{\alpha}} \times \frac{b^{\alpha}\beta_2 A}{m^{\alpha}K(\mu_H^{\alpha} + \gamma_H^{\alpha})}} \tag{10}$$

And

$$\bar{\bar{R}}_0 = \sqrt{\frac{b^{\alpha}\beta_1}{m^{\beta}} \times \frac{b^{\beta}\beta_2 A}{m^{\beta}K(\mu_H^{\alpha} + \gamma_H^{\alpha})}} \tag{11}$$

It is clear that, $\bar{R}_0$ and $\bar{\bar{R}}_0$ carry information about the persistence of the disease. Involvement of the index of human memory (α) and the index of mosquitoes memory (β) in (10) and (11) has some effects on the values of the reproduction numbers $\bar{R}_0$ and $\bar{\bar{R}}_0$. The authors in [49] observed the relation between the memory of mosquitoes and the dengue transmission. Also they studied the effect of the memory of humans on dengue transmission. Increasing human memory ($\alpha \to 0$) will reduce the disease transmission. The authors in [49] presented a strategy for controlling dengue disease through studying the memory effects of the host, and the vector. they believe that, increasing human memory and learning behavior through different kinds of media awareness is essential to reduce the disease transmission. Also the memory of the mosquito (like detecting host sites and suitable locations for their eggs) plays a significant role in the disease transmission. So, Increasing mosquitoes memory and learning behavior ($\beta \to 0$), leads to increasing the disease transmission. The authors observed that the results of (8) have a better agreement with a real measured data in [64]. Such observations support using the models with memory.

7. Fractional variable-order model of multi-strain tuberculosis (TB)

TB is an infectious disease is one of the most dangerous diseases [42], [66]. It is often affect the lungs. According to reports of World Health Organization (WHO) in 2014, 9.6 million people have been infected with TB and 1.5 million died from the disease [69]. TB is one of the major killers of HIV-positive people. In 2015, 1 in 3 HIV deaths was due to TB [67]. Ending the TB epidemic by 2030 is among the World Health Organization (WHO) future plans [69]. Mathematical models help the decision makers to put their plans to control TB diseases and to achieve public health and economic benefits [27]. Anti (TB) drug resistance is one of the most dangerous problems that delayed progress made in TB care [13]. In [52], multi-strain TB model of variable-order fractional derivatives is presented as follows

$$D^{\alpha(t)}(S) = b - dS - \beta_s \frac{SI_s}{N} - \beta_m \frac{SI_m}{N} - \beta_x \frac{SI_x}{N},$$

$$D^{\alpha(t)}(L_s) = \lambda_s\beta_s \frac{SI_s}{N} + \sigma_s\lambda_s\beta_s \frac{RI_s}{N} + \gamma_s I_s - \alpha_{ss}\beta_s \frac{L_s I_s}{N} - \alpha_{sm}\beta_m \frac{L_s I_m}{N} - \alpha_{sx}\beta_x \frac{L_s I_x}{N} - (d + \varepsilon_s + t_{1s})L_s,$$

$$D^{\alpha(t)}(L_m) = \lambda_m\beta_m \frac{SI_m}{N} + \sigma_m\lambda_m\beta_m \frac{RI_m}{N} + \gamma_m I_m + \alpha_{sm}\beta_m\lambda_m \frac{L_s I_m}{N} + (1 - P_1)t_{1s}L_s + (1 - P_2)t_{2s}I_s - \alpha_{mm}\beta_m \frac{L_m I_m}{N} - \alpha_{mx}\beta_x \frac{L_m I_x}{N} - (d + \varepsilon_m)L_m$$

$$D^{\alpha(t)}(L_x) = \lambda_x\beta_x\frac{SI_x}{N} + \sigma_x\lambda_x\beta_x\frac{RI_x}{N} + \gamma_x I_x + \alpha_{sx}\beta_x\lambda_x\frac{L_sI_x}{N} + \alpha_{mx}\lambda_x\beta_x\frac{L_mI_x}{N} + (1-P_3)t_{2m}I_m - \alpha_{xx}\beta_x\frac{L_xI_x}{N} - (d+\varepsilon_x)L_x,$$

$$D^{\alpha(t)}(I_s) = \alpha_{ss}\beta_s\frac{L_sI_s}{N} + (1-\lambda_s)\beta_s\left(\frac{SI_s}{N} + \sigma_s\frac{RI_s}{N}\right) + \varepsilon_s L_s - (d+\delta_s+t_{2s}+\gamma_s)I_s,$$

$$D^{\alpha(t)}(I_m) = \alpha_{mm}\beta_m\frac{L_mI_m}{N} + (1-\lambda_m)\beta_m(\frac{SI_m}{N} + \sigma_m\frac{RI_m}{N}\alpha_{sm}\frac{L_sI_m}{N})\varepsilon_m L_m - (d+\delta_m+t_{2m}+\gamma_m)I_m,$$

$$D^{\alpha(t)}(I_x) = \alpha_{xx}\beta_x\frac{L_xI_x}{N} + (1-\lambda_x)\beta_x\left(\frac{SI_x}{N} + \sigma_x\frac{RI_x}{N} + \alpha_{sx}\frac{L_sI_x}{N} + \alpha_{mx}\frac{L_mI_x}{N}\right) + \varepsilon_x L_x - (d+\delta_x+t_{2x}+\gamma_x)I_x$$

$$D^{\alpha(t)}(R) = P_1t_{1s}L_s + P_2t_{2s}I_s + P_3t_{2m}I_m + t_{2x}I_x - \sigma_s\beta_s\frac{RI_s}{N} - \sigma_m\beta_m\frac{RI_m}{N} - \sigma_x\beta_x\frac{RI_x}{N} - dR \quad (12)$$

Where $0 < \alpha(t) \leq 1$ is the fractional variable order,
$S(t)$ is the susceptible individuals,
$L_s(t)$ is the latently infected individuals with the drug-sensitive TB strain,
$L_m(t)$ is the latently infected population with MDR-TB,
$L_x(t)$ is the latently infected population with XDR-TB,
$I_s(t)$ is the infected individuals with the drug-sensitive TB strain who are infectious to others,
$I_m(t)$ presents the infectious individuals with the MDR-TB strain,
$I_x(t)$ presents the individuals who infectious with the XDR-TB strain,
$R(t)$ is the populations for who have been treated successfully,

$N(t) = S(t) + L_s(t) + L_m(t) + L_x(t) + I_s(t) + I_m(t) + I_x(t) + R(t)$ is the total population

b is the rate of birth/recruitment,
d is per capita natural death rate,
β_r is transmission coefficient for strain r,
λ_r is the proportion of newly infected individuals developing LTBI with strain r,
$1-\lambda_r$ is the proportion of newly infected individuals progressing to active TB with strain r due to fast infection,
ε_r is per capita rate of endogenous reactivation of L_r,
α_{r1}, α_{r2} are the proportion of exogenous reinfection of L_{r2} due to contact with I_{r2},
γ_r is per capita rate of natural recovery to the latent stage L_r,
δ_r is per capita rate of death due to TB of strain r,
t_{1s} is per capita rate of treatment for L_s,
t_{2r} is per capita rate of treatment for I_r,
$1-\sigma_x$ is the efficiency of treatment,
P_1, P_2, and P_3 are the probabilities of treatment success for L_s, I_s, and I_m respectively,
The notation $r_1, r_2, r_3 \in \{s, m, x\}$ is used. This model presents three strains: drug-sensitive, emerging multi-drug resistant (MDR) and extensively drug-resistant (XDR), as an extension for multi-strain TB integer order model which was developed in [13]. From the numerical results obtained in [52], the integer order model can be used to describe the short memory of the model, and the variable-order fractional model can be used to present the variable memory of TB model. The basic reproduction number has been derived in [ ] for (12) to be:

$$R_{0s} = \frac{\beta_s(\varepsilon_s+(1-\lambda_s)(d+t_{1s}))}{(\varepsilon_s+d+t_{1s})(t_{2s}+\delta_s+d)+\gamma_s(t_{1s}+d)}$$

$$R_{0m} = \frac{\beta_m(\varepsilon_m+(1-\lambda_m)d)}{(\varepsilon_m+d)(t_{2m}+\delta_m+d)+\gamma_m d}$$

$$R_{0x} = \frac{\beta_x(\varepsilon_x+(1-\lambda_x)d)}{(\varepsilon_x+d)(t_{2x}+\delta_x+d)+\gamma_x d}$$

8. Fractional order models of infectious diseases with time delay

Many researchers introduce the fractional order derivative into the infectious diseases models for its memory property. Time delay in these models plays a significant role in the process of spreading infectious diseases [34,35,59] as it introduces the dependence of the present state of the model on the past history. This time delay takes into account certain hidden processes like the viral life cycle and duration of immunity to diseases. Time delays increases the risk of epidemics and the difficulty of epidemic control. Fractional order derivative and time delays in infectious diseases models give a better understanding of the dynamics of infectious diseases. Several works have been proposed to study the fractional order models with
time delay of infectious diseases. In this section, three examples of such models will be illustrated.

8.1. Fractional order model of HIV with time delay

The authors in [59] have introduced the following system to model HIV infection of $CD4^+T$-cells with time delay:

$$D^{\alpha}(T) = s - \mu_T T(t) + rT(t)\left(1 - \frac{T(t)+I(t)}{T_{max}}\right) - kT(t)V(t)$$

$$D^{\alpha}(I) = k'T(t-\tau)V(t-\tau) - \mu_I I(t) \quad (13)$$

$$D^{\alpha}(V) = N\mu_b I(t) - kT(t)V(t) - \mu_v V(t)$$

Where

$T(\theta) = T_0, I(0) = 0, V(\theta) = V_0, \theta \in [-\tau, 0]$,

The authors in [34] presents a new fractional order time-delay model which is as an extension of the work in [59]. This model includes full logistic growth terms of both healthy and infected CD4$^+$ T-cells, and cure rate items. The new model is described by the following fractional order differential time-delay model:

$$D^\alpha(T) = s - \mu_T T(t) + rT(t)\left(1 - \frac{T(t)+I(t)}{T_{max}}\right) - kT(t)V(t) + \rho I(t)$$

$$D^\alpha(I) = k'T(t-\tau)V(t-\tau) + rI(t)\left(1 - \frac{T(t)+I(t)}{T_{max}}\right) - (\mu_I + \rho)I(t) \quad (14)$$

$$D^\alpha(V) = N\mu_b I(t) - \mu_v V(t)$$

Where $T(\theta) = T_0, I(0) = 0, V(\theta) = V_0, \theta \in [-\tau, 0], 0 < \alpha \leq 1$, and $\tau \geq 0$ is the length of the delay in days, $T(t)$, $I(t)$, and $V(t)$ represent the concentration of healthy $CD4^+T$ cells, infected $CD4^+T$-cells, and density of HIV RNA in the blood, respectively. The parameters are defined in [1], [2] as follows:
s is the source term for uninfected $CD4^+T$ cells,
μ_T is the natural death rate of $CD4^+T$ cells,
μ_I is the blanket death rate of the infected cells,
μ_v is the death rate of free virus,
μ_b is the rate of lytic death of infected cells,
k is the rate of infectious of $CD4^+T$- cells,
k' is the rate of activation of infected cells,
ρ is the rate of reverting from infected cells to the uninfected state,
r is the growth rate of $CD4^+T$ - cells,
N is the number of virions produced by infected $CD4^+T$- cells,
T_{max} is the maximal population level of $CD4^+T$- cells,
T_0 is the $CD4^+T$ - cells population for HIV-negative persons,
The numerical solutions in [34] indicate that, when α increases, the trajectory of the model is close to the integer-order model and when τ increases, the fluctuation of the trajectory of the model is smaller during the previous period of the time. Also numerical solutions in [34] show that if the cure rate gets large, the HIV infection efficiently can be controlled.

8.2. Fractional order TB model with time delay

In [53], fractional order TB model with time delay is proposed. This model is based on Grünwald–Letnikov fractional order derivative. In this work, the fractional order system which has been presented in [53] as follows:
which has been presented in [53] as follows:

$$D^\alpha(S) = b^\alpha - d^\alpha S - \beta_s^\alpha \frac{SI_s}{N} - \beta_m^\alpha \frac{SI_m}{N} - \beta_x^\alpha \frac{SI_x}{N},$$

$$D^\alpha(L_s) = \lambda_s^\alpha \beta_s^\alpha \frac{SI_s}{N} + \lambda_s^\alpha \sigma_s^\alpha \beta_s^\alpha \frac{RI_s}{N} + \gamma_s^\alpha I_s - \alpha_{ss}^\alpha \beta_s^\alpha \frac{L_s I_s}{N} - \alpha_{sm}^\alpha \beta_m^\alpha \frac{L_s I_m}{N} - \alpha_{sx}^\alpha \beta_x^\alpha \frac{L_s I_x}{N} - (d^\alpha + \varepsilon_s^\alpha + t_{1s}^\alpha)L_s,$$

$$D^\alpha(L_m) = \lambda_m^\alpha \beta_m^\alpha \frac{SI_m}{N} + \lambda_m^\alpha \sigma_m^\alpha \beta_m^\alpha \frac{RI_m}{N} + \gamma_m^\alpha I_m + \alpha_{sm}^\alpha \beta_m^\alpha \lambda_m^\alpha \frac{L_s I_m}{N} + (1 - P_1^\alpha) t_{1s}^\alpha L_s + (1 - P_2^\alpha) t_{2s}^\alpha I_s - \alpha_{mm}^\alpha \beta_m^\alpha \frac{L_m I_m}{N} - \alpha_{mx}^\alpha \beta_x^\alpha \frac{L_m I_x}{N} - (d^\alpha + \varepsilon_m^\alpha)L_m,$$

$$D^\alpha(L_x) = \lambda_x^\alpha \beta_x^\alpha \frac{SI_x}{N} + \sigma_x^\alpha \lambda_x^\alpha \beta_x^\alpha \frac{RI_x}{N} + \gamma_x^\alpha I_x + \alpha_{sx}^\alpha \beta_x^\alpha \lambda_x^\alpha \frac{L_s I_x}{N} + \alpha_{mx}^\alpha \lambda_x^\alpha \beta_x^\alpha \frac{L_m I_x}{N} + (1 - P_3^\alpha) t_{2m}^\alpha I_m - \alpha_{xx}^\alpha \beta_x^\alpha \frac{L_x I_x}{N} - (d^\alpha + \varepsilon_x^\alpha)L_x$$

$$D^\alpha(I_s) = \alpha_{ss}^\alpha \beta_s^\alpha \frac{L_s I_s}{N} + (1 - \lambda_s^\alpha)\beta_s^\alpha \left(\frac{SI_s}{N} + \sigma_s^\alpha \frac{RI_s}{N}\right) + \varepsilon_s^\alpha L_s - (d^\alpha + \delta_s^\alpha + t_{2s}^\alpha + \gamma_s^\alpha)I_s,$$

$$D^\alpha(I_m) = \alpha_{mm}^\alpha \beta_m^\alpha \frac{L_m I_m}{N} + (1 - \lambda_m^\alpha)\beta_m^\alpha \left(\frac{SI_m}{N} + \sigma_m^\alpha \frac{RI_m}{N} + \alpha_{sm} \frac{L_s I_m}{N}\right) + \varepsilon_m^\alpha L_m - (d^\alpha + \delta_m^\alpha + \gamma_m^\alpha)I_m - t_{2m}^\alpha I_m(t-\tau),$$

$$D^\alpha(I_x) = \alpha_{xx}^\alpha \beta_x^\alpha \frac{L_x I_x}{N} + (1 - \lambda_x^\alpha)\beta_x^\alpha \left(\frac{SI_x}{N} + \sigma_x^\alpha \frac{RI_x}{N} + \alpha_{sx}^\alpha \frac{L_s I_x}{N} + \alpha_{mx}^\alpha \frac{L_m I_x}{N}\right) + \varepsilon_x^\alpha L_x - (d^\alpha + \delta_x^\alpha + \gamma_x^\alpha)I_x - t_{2x}^\alpha I_x(t-\tau),$$

$$D^\alpha(R) = P_1^\alpha t_{1s}^\alpha L_s + P_2^\alpha t_{2s}^\alpha I_s + P_3^\alpha t_{2m}^\alpha I_m + t_{2x}^\alpha I_x(t-\tau) - \sigma_s \beta_s^\alpha \frac{RI_s}{N} - \sigma_m^\alpha \beta_m^\alpha \frac{RI_m}{N} - \sigma_x^\alpha \beta_x^\alpha \frac{RI_x}{N} - d^\alpha R \quad (15)$$

The basic reproduction number has been derived in [53] for (15) to be:

$$R_{0s} = \frac{\beta_s^\alpha(\varepsilon_s^\alpha + (1-\lambda_s^\alpha)(d^\alpha + t_{1s}^\alpha))}{(\varepsilon_s^\alpha + d^\alpha + t_{1s}^\alpha)(t_{2s}^\alpha + \delta_s^\alpha + d^\alpha) + \gamma_s^\alpha(t_{1s}^\alpha + d^\alpha)}$$

$$R_{0m} = \frac{\beta_m^\alpha(\varepsilon_m^\alpha + (1-\lambda_m^\alpha)d^\alpha)}{(\varepsilon_m^\alpha + d^\alpha)(t_{2m}^\alpha + \delta_m^\alpha + d^\alpha) + d^\alpha \gamma_m^\alpha}$$

$R_{0x} = \frac{\beta_x^\alpha(\varepsilon_x^\alpha+(1-\lambda_x^\alpha)d^\alpha)}{(\varepsilon_x^\alpha+d^\alpha)(t_{2x}^\alpha+\delta_x^\alpha+d^\alpha)+\gamma_x^\alpha d^\alpha}$

8.3. Fractional order model for malaria transmission with time delay under impact of vaccination control

Malaria disease is caused by the bites of infected Anopheles mosquitoes. It is the one of most fatal vector borne diseases. About 216 million people have been infected with malaria in 2016 [71]. 91% of such cases were in Africa. 445 000 people died in 2016 because of malaria infection [71]. Fractional order models can be used to study the impact of vaccination strategies against malaria transmission. A delayed fractional order model of Malaria transmission is proposed in [43]. The effect of impact of vaccination control is implemented in this model.

$$D^{\alpha_1}(N_h(t)) = \Lambda_h - \xi_h\big(I_h(t) + (1-\theta_2)Y_h(t) + (1-\upsilon)T_h(t)\big) - \mu_h N_h(t),$$

$$D^{\alpha_2}(S_h(t)) = (1-p)\Lambda_h - f_h(t)S_h(t) + r_h\big(I_h(t) + \theta_1 Y_h(t) + \delta T_h(t)\big) + \sigma V_h(t) - \mu_h S_h(t)$$

$$D^{\alpha_3}(V_h(t)) = p\Lambda_h - f_h(t)(1-\gamma)V_h(t) - (\sigma+\mu_h)V_h(t)$$

$$D^{\alpha_4}(I_h(t)) = f_h(t-\tau_h)S_h(t-\tau_h)e^{-\mu_h\tau_h} - (k + r_h + \xi_h + \mu_h)I_h(t)$$

$$D^{\alpha_5}(Y_h(t)) = f_h(t-\tau_h)(1-\gamma)V_h(t-\tau_h)e^{-\mu_h\tau_h} - (k + \theta_1 r_h + (1-\theta_2)\xi_h + \mu_h)Y_h(t)$$

$$D^{\alpha_6}(T_h(t)) = k(I_h(t) + Y_h(t)) - (\delta r_h + (1-\nu)\xi_h + \mu_h)T_h(t)$$

$$D^{\alpha_7}(N_m(t)) = \Lambda_m - \xi_m I_m(t) - \mu_m N_m(t)$$

$$D^{\alpha_8}(S_m(t)) = \Lambda_m - f_m(t)S_m(t) - \mu_m S_m(t)$$

$$D^{\alpha_9}(I_m(t)) = f_m(t-\tau_m)S_m(t-\tau_m)e^{-\mu_m\tau_h} - (\mu_m + \xi_m)I_m(t) + \Lambda_m \quad (16)$$

Given that

$$f_h(t) = \beta_h c(1-bz)\frac{I_m(t)}{N_h(t)},$$

$$f_m(t) = \beta_m c(1-bz)\frac{I_h(t)+(1-\epsilon)Y_h(t)+(1-\eta)T_h(t)}{N_h(t)}$$

Where
$\alpha_i \in [0,1] for\ i = 1,2,3,\dots,9$
$N_h(t)$ is the total human population,
$S_h(t)$ is the number of susceptible human individuals,
$V_h(t)$ is the number of vaccinated human individuals,
$I_h(t)$ is the number of infectious human individuals,
$Y_h(t)$ is the number of infectious vaccinated human individuals,
$T_h(t)$ is the number of treated human individuals,
$N_m(t)$ is the total number of mosquito population,
$S_m(t)$ is the number of susceptible mosquitos,
$I_m(t)$ is the number of infectious mosquitos,
Λ_h is the rate of recruitment of human population,
p is the proportion of vaccinated individuals,
σ is the rate of loss of immunity due to vaccination,
$r_h$is the rate of recovery,
C is the biting rate,
$\beta_h$is the probability of transmission,
b is the personal protection efficacy,
z is the compliance of personal protection,
$\xi_h$is the death rate due to the disease,
μ_h is the human's natural death rate,
γ is the pre-erythrocytic vaccine efficacy,
ϵ is the efficacy of transmission blocking vaccine,
τ_h is the latent period of humans,
k is the Treatment rate,
η is the drug efficacy,
Λ_m is the rate of recruitment of human population,
β_m is the probability of infection transmission to mosquitoes,
μ_m is the natural death rate of mosquitoes,
τ_m is the latent period for mosquitoes,
ξ_m is the rate of death due to presence of parasites,
Infectious vaccinated humans recover at the rate of $\theta_1 r_h$ or die at the rate $(1-\theta_2)\xi_h$, while treated humans recover at the rate of δr_h or die at the rate of $(1-\nu)\xi_h$.

9. Fractional order SIR model based on stochastic process

In [5], a new sophisticated approach has been proposed to derive fractional order SIR model. Instead of replacing first order derivatives in the classical SIR model by a Caputo fractional order derivatives, the authors in [5] developed a new Fractional order SIR recovery model based on physical stochastic process. They follow an interesting technique in their approach by replacing the constant parameters in the classical SIR model with time dependent parameters. In addition, they use a Mittag-Leffler waiting time distribution, to derive the following generalized master SIR model with fractional recovery:

$$\frac{ds(t)}{dt} = \lambda(t) - \omega(t)S(t)I(t) - \gamma(t)S(t),$$

$$\frac{dI(t)}{dt} = \omega(t)S(t)I(t) - \gamma(t)I(t) - \theta(t,0)\left(\mu D_t^{1-\alpha}\left(\frac{I}{\theta(t,0)} - \frac{I_0}{\theta(t,0)}\right) - \frac{d}{dt}\left(\frac{I_0}{\theta(t,0)}\right)\right), \quad (17)$$

$$\frac{dR(t)}{dt} = \theta(t,0)\left(\mu D_t^{1-\alpha}\left(\frac{I}{\theta(t,0)} - \frac{I_0}{\theta(t,0)}\right) - \frac{d}{dt}\left(\frac{I_0}{\theta(t,0)}\right)\right) - \gamma(t)R(t).$$

Where
S is the susceptible population
I is the infected populations
R is the recovered population
$\theta(t,t')$ is the death surviving probability of from t' to time t

$$\theta(t,0) = \theta(t,t')\theta(t',0), for\ 0 < t' < t.$$

$\omega(t)$ is the rate of becoming infected
If the probability of death happens in the interval t to $t+\delta t$ is $\gamma(t)\delta t + o(\delta t)$, then:

$$\theta(t,t') = e^{-\int_{t'}^{t}\gamma(t)ds}$$

10. Conclusion

As the fractional-order dynamical models possess memory, it offers a deep insight into the mathematical modeling of infectious diseases. In this paper, we have provided some fractional order models of infectious diseases to show how memory effect changes the dynamics of diseases. Fractional order models exhibit much more realistic dynamics than integer order models because such models carry information about the memory of living systems and its associative learning mechanisms. The fractional order derivative is the parameter of memory.

References

[1] Cho E. Ahmed, A.M.A. El-Sayed and H.A.A. El-Saka, on some Routh-Hurwitz conditions for fractional order differential equations and their applications in Lorenz, Rossler, Chua and Chen systems, Phys. Lett. A358, 1–4 (2006). https://doi.org/10.1016/j.physleta.2006.04.087.

[2] E. Ahmed and H.A. El-Saka, on fractional order models for Hepatitis C, Nonlinear Biomed. Phys. 4 (2010). https://doi.org/10.1186/1753-4631-4-1.

[3] E. Ahmed, A.M.A. El-Sayed, H.A.A. El-Saka, Equilibrium points, stability and numerical solutions of fractional-order predator–prey and rabies models, J. Math. Anal. Appl. 325 (2007) 542–553. https://doi.org/10.1016/j.jmaa.2006.01.087.

[4] E. Ahmed and A.S. Elgazzar, on fractional order differential equations model for nonlocal epidemics, Physica A 379 (2007) 607–614. https://doi.org/10.1016/j.physa.2007.01.010.

[5] C. N. Angstmann, B. I. Henry, A. V. McGann, A Fractional Order Recovery SIR Model from a Stochastic Process, Bull Math Biol (2016) 78:468–499. https://doi.org/10.1007/s11538-016-0151-7.

[6] A.A.M. Arafa, S.Z. Rida, M. Khalil, A Fractional-Order Model of HIV Infection: Numerical solution and Comparisons with Data of Patients, Int. J. Biomath, 7 (4) (2014). https://doi.org/10.1142/S1793524514500363.

[7] A.A.M. Arafa, S.Z. Rida and M. Khalil, Fractional modeling dynamics of HIV and CD4+ T-cells during primary infection, Nonlinear Biomed. Phys 6(2012). https://doi.org/10.1186/1753-4631-6-1.

[8] A.A.M. Arafa, S.Z. Rida and M. Khalil, The effect of anti-viral drug treatment of human immunodeficiency, Appl. Math. Model, 37 (2013) 2189–2196. https://doi.org/10.1016/j.apm.2012.05.002.

[9] A.A.M. Arafa, S.Z. Rida and M. Khalil, Fractional Order Model of Human T-cell Lymphotropic Virus I (HTLV-I) Infection of CD4+T-cells, Advanced Studies in Biology, 3(2011) 347 – 353. https://doi.org/10.14419/ijbas.v1i1.15.

[10] A.A.M. Arafa, S.Z. Rida, M.Khalil, Solutions of Fractional Order Model of Childhood diseases with Constant Vaccination Strategy, Math. Sci. Lett. No. 1 1(2012) 17-23. https://doi.org/10.12785/msl/010103.

[11] A.A.M. Arafa, S.Z. Rida, M.Khalil, A fractional-order model of HIV infection with drug therapy effect, J. Egyptian Math. Soc., 22(2014) 538–543. https://doi.org/10.1016/j.joems.2013.11.001.

[12] I. Area, H. Batarfi, J. Losada, J. J Nieto, W. Shammakh, A. Torres, On a fractional order Ebola epidemic model, Adv. Difference Equ. 278(2015). https://doi.org/10.1186/s13662-015-0613-5.

[13] J. Arino, I.A. Soliman, A model for the spread of tuberculosis with drug-sensitive and emerging multidrug-resistant and extensively drug resistant strains, in Mathematical and computational modelling with applications in natural and social sciences, engineering and arts, Wiley; 2014. https://doi.org/10.1002/9781118853887.ch5.

[14] S. Crotty, R. Ahmed, Immunological memory in humans, Seminars in Immunology 16 (2004) 197–203. https://doi.org/10.1016/j.smim.2004.02.008.

[15] R.V. Culshaw, S. Ruan, A delay-differential equation model of HIV infection of CD4+ T-cells, Math. Biosci.165 (2000) 27-39. https://doi.org/10.1016/S0025-5564(00)00006-7.

[16] H. Dahari, A. Lo A, R.M. Ribeiro, A.S. Perelson: Modeling hepatitis C virus dynamics: Liver regeneration and critical drug efficacy. J Theoret Biol 2007, 247:371-381. https://doi.org/10.1016/j.jtbi.2007.03.006.

[17] E. Demirci, A. Unal and N. Ozalp, A fractional order SEIR model with density dependent death rate, Hacet. J. Math. Stat. 40 (2011), 287–295.

[18] K. Diethelm, A fractional calculus-based model for the simulation of an outbreak of dengue fever, Nonlinear Dyn, 71(2013), 613–619. https://doi.org/10.1007/s11071-012-0475-2.

[19] Y. Ding, H. Ye, A fractional-order differential equation model of HIV infection of CD4+ T-cells, Math. Comput. Modelling, 50 (2009) 386-392. https://doi.org/10.1016/j.mcm.2009.04.019.

[20] A. O'Dowd, Infectious diseases are spreading more rapidly than ever before, WHO warns. BMJ 335 (2007)418. https://doi.org/10.1136/bmj.39318.516968.DB.

[21] M. Du, Z. Wang and H. Hu, Measuring memory with the order of fractional derivative. Sci. Rep. 3(2013). https://doi.org/10.1038/srep03431.

[22] M. Glomski, E. Ohanian, Eradicating a Disease: Lessons from Mathematical Epidemiology. The College Mathematics Journal, 43.2 (2012): 123-132. https://doi.org/10.4169/college.math.j.43.2.123.

[23] N.C. *Grassly*, C. Fraser, Mathematical models of infectious disease transmission. Nat Rev Microbiol 2008; 6(6):477-487. https://doi.org/10.1038/nrmicro1845.

[24] A.H. Hashish, E. Ahmed, towards understanding the immune system, Theor. Biosci. 126 (2–3) (2007) 61–64. https://doi.org/10.1007/s12064-007-0011-y.

[25] H.W. Hethcote, the Mathematics of Infectious Diseases, SIAM Rev. 42 (2000) 599. https://doi.org/10.1137/S0036144500371907.

[26] G. M. Hwang, P.J. Mahoney, J.H. James, G.C. Lin, A.D. Berro, M.A. Keyb, D.M. Goedecke, J.J. Mathieu, T. Wilson: A model-based tool to predict the propagation of infectious disease via airports. Travel Med Infect Dis. 10(1) (2012)32-42. https://doi.org/10.1016/j.tmaid.2011.12.003.

[27] R. M. G. J. Houben, D. W. Dowdy, A. Vassall, T. Cohen, M. P. Nicol, R. M. Granich, J. E. Shea, P. Eckhoff, C. Dye, M. E. Kimerling, R. G. White, How can mathematical models advance tuberculosis control in high HIV prevalence settings?, International Journal of Tuberculosis and Lung Disease 18 (2014) 509–514. https://doi.org/10.5588/ijtld.13.0773.

[28] J. Huo, H. Zhao, L. Zhu, The effect of vaccines on backward bifurcation in a fractional order HIV model, Nonlinear Anal. Real World Appl. 26 (2015) 289–305 https://doi.org/10.1016/j.nonrwa.2015.05.014.

[29] A. Huppert A, G. Katriel, Mathematical modelling and prediction in infectious disease epidemiology. Clin Microbiol Infect. 2013; 19:999–1005. https://doi.org/10.1111/1469-0691.12308.

[30] M. Jit, M. Brisson, Modelling the epidemiology of infectious diseases for decision analysis: a primer. Pharmaco Economics 29(5), 371–386 (2011). https://doi.org/10.2165/11539960-000000000-00000.

[31] J. CA Jr, et al: The immune system in health and disease. In Immunobiology. Garland Pub, NewYork: 2001.

[32] P. S. Kim, D. Levy, and P. P. Lee, "Modeling and simulation of the immune system as a self-regulating network," *Methods in Enzymology*, vol. 467, no. C, pp. 79–109, 2009. https://doi.org/10.1016/S0076-6879(09)67004-X.

[33] I.K. Kouadio, S. Aljunid, T. Kamigaki, K. Hammad, H. Oshitani, Infectious disease following natural disasters: prevention and control measures. Expert Rev Anti Infect Ther 10(2012); 95–104. https://doi.org/10.1586/eri.11.155.

[34] Z. Liu, P. Lu, Stability analysis for HIV infection of CD4+T-cells by a fractional differential time-delay model with cure rate, *Advances in Difference Equations* 2014, 2014:298. https://doi.org/10.1186/1687-1847-2014-298.

[35] Y. Liu, P. Lu, and I. Szanto, Numerical Analysis for a Fractional Differential Time-Delay Model of HIV Infection of CD4+ T-Cell Proliferation under Antiretroviral Therapy.

[36] S. Mandal, R. R. Sarkar and S. Sinha, Mathematical models of malaria - a review, Malar J, 10 (2011) 10:202. https://doi.org/10.1186/1475-2875-10-202.

[37] A.K. Misra, Anupama Sharma, J.B. Shukla, Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases, Mathematical and Computer Modelling 53 (2011) 1221–1228. https://doi.org/10.1016/j.mcm.2010.12.005.

[38] K. Moaddy, A.G. Radwan, K.N. Salama, S. Momani, I. Hashim, the fractional-order modeling and synchronization of electrically coupled neuron systems, Comput. Math. Appl., 64 (2012) 3329–3339. https://doi.org/10.1016/j.camwa.2012.01.005.

[39] P. A. Morel, S. Ta'asan, B. F. Morel, D. E. Kirschner, and J.L. Flynn, "New insights into mathematical modeling of the immune system," Immunologic Research, vol. 36, no. 1–3, pp.157–165, 2006. https://doi.org/10.1385/IR:36:1:157.

[40] Z. Mukandavire,W. Garira, J.M. Tchuenche, Modelling effects of public health educational campaigns on HIV/AIDS transmission dynamics, Applied Mathematical Modelling, 33(2009) 2084–2095. https://doi.org/10.1016/j.apm.2008.05.017.

[41] H. Nishiura, Mathematical and statistical analyses of the spread of dengue. In: Dengue Bulletin, 30(2006) 51–67.

[42] D. Okuonghae and B. O. Ikhimwin, Dynamics of a Mathematical Model for Tuberculosis with Variability in Susceptibility and Disease Progressions Due to Difference in Awareness Level, Front. Microbiol., (2016). https://doi.org/10.3389/fmicb.2015.01530.

[43] C.M.A. Pinto, J.A. Tenreiro Machado, Fractional model for malaria transmission under control strategies, Comput. Math. Appl. 66 (5) (2013) 908–916. https://doi.org/10.1016/j.camwa.2012.11.017.

[44] C.M.A. Pinto, A.R.M. Carvalho, Effect of drug-resistance in a fractional complex-order model for HIV infection, IFAC, 48(2015) 188–189. https://doi.org/10.1016/j.ifacol.2015.05.162.

[45] D.A. Relman, R.R. Choffnes, A. Mack, Infectious disease movement in a borderless world: workshop summary. The National Academies Press, Washington, DC; 2010.

[46] S.Z. Rida, A.S. Abdel Rady, A.A.M. Arafa, M. Khalil, Approximate solution of a fractional order model of HCV infection with drug therapy effect, International Journal of Applied Mathematical Research, 1 (2) (2012) 108-116. https://doi.org/10.14419/ijamr.v1i2.25.

[47] L. Rong, Z. Feng, A.S. Perelson, Emergence of HIV-1 drug resistance during antiretroviral treatment. Bull Math Biol 2007; 69:2027–2060. https://doi.org/10.1007/s11538-007-9203-3.

[48] M. Salah, Abdelouahab and N.E Hamri, The Grunwald-Letnikov Fractional-Order Derivative with Fixed Memory Length, Mediterr. J. Math., (2015) 1-16. https://doi.org/10.1007/s00009-015-0525-3.

[49] T. Sardar, S. Rana, S. Bhattacharya, K. Al-Khaled, J. Chattopadhyay, A generic model for a single strain mosquito-transmitted disease with memory on the host and the vector, Math. Biosci. 263 (2015) 18–36. https://doi.org/10.1016/j.mbs.2015.01.009.

[50] A. Scherer and A. R. McLean, Mathematical models of vaccination, Brit. Med. Bull., 62 (2002) 187–199. https://doi.org/10.1093/bmb/62.1.187.

[51] H.G. Sun, W. Chen, H. Wei, and Y.Q. Chen, A comparative study of constant-order and variable-order fractional models in characterizing memory property of systems, Eur. Phys. J. Special Topics 193, (2011) 185–192. https://doi.org/10.1140/epjst/e2011-01390-6.

[52] N.H. Sweilam, S. M. AL-Mekhlafi, Numerical study for multi-strain tuberculosis (TB) model of variable-order fractional derivatives, J. Adv. Res. 7(2016) 271–283. https://doi.org/10.1016/j.jare.2015.06.004.

[53] N.H. Sweilam, S.M. Al-Mekhlafi, and T.A.R Assiri, "Numerical Study for Time Delay Multistrain Tuberculosis Model of Fractional Order," Complexity, 2017. https://doi.org/10.1155/2017/1047384.

[54] J.K. Taubenberger, D.M. Morens, 1918 Influenza: the mother of all pandemics. Emerg. Infect. Dis. 12 (2006) 15–22. https://doi.org/10.3201/eid1209.05-0979.

[55] J. M. Tchuenche and C. T. Bauch, Dynamics of an Infectious Disease Where Media Coverage Influences Transmission, ISRN Biomathematics, (2012). https://doi.org/10.5402/2012/581274.

[56] Mm C. Vinauger, E.K. Lutz and J. A. Riffell, Olfactory learning and memory in the disease vector mosquito Aedes aegypti, The Journal of Experimental Biology 217(2014) 2321-2330. https://doi.org/10.1242/jeb.101279.

[57] Y. Xu, Z. He, Existence and uniqueness results for Cauchy problem of variable-order fractional differential equations, J Appl Math Comput, 43(1) (2013) 295-306. https://doi.org/10.1007/s12190-013-0664-2.

[58] M. Xu, J. Yang, D. Zhao and H. Zhao, an image-enhancement method based on variable-order fractional differential operators, Bio-Medical Materials and Engineering 26 (2015) S1325–S1333. https://doi.org/10.3233/BME-151430.

[59] Y. Yan and C. Kou, "Stability analysis for a fractional differential model of HIV infection of CD4+ T-cells with time delay," Mathematics and Computers in Simulation, vol. 82, no. 9, pp. 1572–1585, 2012. https://doi.org/10.1016/j.matcom.2012.01.004.

[60] Africa Renewal: Ebola threatens economic gains in affected countries, 2014. http://www.un.org/africarenewal/magazine/december-2014/ebola-threatens-economic-gains-affected-countries

[61] Global Health Policy: The U.S. Government & Global Emerging Infectious Disease Preparedness and Response, (2014). http://kff.org/global-health-policy/fact-sheet/the-u-s-government-global-emerging-infectious-disease-preparedness-and-response/.
[62] London school of Hygine & Tropical medicine: Introduction to Infectious Disease Modelling and Its Applications, 2015. https://www.lshtm.ac.uk/study/cpd/siidma.html.
[63] Virginia Department of Health: Vector-borne Disease Control (2015): http://www.vdh.state.va.us/epidemiology/dee/vectorborne/.
[64] World Health Organization, 2011. Dengue Net database and geographic information system. [Online; accessed 1-October-2013]. http://apps.who.int/globalatlas/DataQuery/default.asp.
[65] World Health Organization: Vector-borne diseases (2014). http://www.who.int/mediacentre/factsheets/fs387/en/.
[66] World Health Organization: Ebola Situation Reports, 2015. http://apps.who.int/ebola/ebola-situation-reports.
[67] World Health Organization: HIV (2015). http://www.who.int/hiv/en/
[68] World Health Organization: What is TB? How is it treated? (2015). http://www.who.int/features/qa/08/en/.
[69] World Health Organization: Tuberculosis (2016). http://www.who.int/mediacentre/factsheets/fs104/en/.
[70] Who organization, Zika virus (2016). http://www.who.int/mediacentre/factsheets/zika/en/.
[71] World Health Organization: Malaria (2017). http://www.who.int/mediacentre/factsheets/fs094/en/.