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Theme

Mathematical analysis of epidemiological systems via fractional operators

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Dedications

I dedicate this humble work to :
⋆⋆ **My dear parents** , who took care of raising me, educated me and helped me in my life .⋆⋆

⋆⋆ **my dear brothers and my sisters** for they encourage me all the time. ⋆⋆

⋆⋆ **to my happiness** : my fiance **yassine**.⋆⋆

⋆⋆ All my relatives, friends, loved ones, and my colleagues. ⋆⋆
⋆⋆ Everyone who taught me letters in my life. ⋆⋆

⋆⋆ to all my dear teachers from the beginning of my study career. ⋆⋆

Thanks

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My big thanks to ⋆ **father and mother** ⋆,they were the best help to me,and they have the credit for reaching this moment.

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Introduction

Fractional calculus is known as a generalization of classical derivatives and integrals. In recent years, this type of calculus has been developed, which has given more accurate and realistic results. For example, if we talk about epidemics, the order of the fractional derivative represents individuals who are facing the outbreak of disease based on previous diseases, and this is what is known in epidemiology as memory .

The memory factor has an important role in studying the outbreak of epidemics as it can help stop the spread of the disease in the community. Due to this great role of fractional calculus, many researchers have used it in conducting research related to describing, predicting, and controlling the behavior of epidemics Among them ; Models to study control and treatment effects on the spread of HIV disease [6] , models to predict the behavior of the coronavirus epidemic in countries [7], models for anthrax [15], and a toxoplasmosis model[19] .

Some have also used this type of calculas in studying the dynamics of hepatitis B disease, such as ; modeling and analysis of the fractional HBV model with Atangana-Baleanu derivative in [5], a fractional order model for Hepatitis B virus with treatment in [17], analysis of a model of HBV infection with an antibody immune response using Caputo fractional derivative in [4], and in [11] Nadia Gul and others have studied the dynamics of fractional order Hepatitis B virus model with asymptomatic carriers.

It is worth noting that the non-locality represents the correlation between the real problem and the mathematical problem, so that the greater the nonlocality in the mathematical problem, the greater the accuracy in depicting the problem with mathematical equations. Non-local operators aimed to at-

tract more non-local natural problems, In pursuit of more realistic models , Abdon Atangana proposed new operators for fractiona calculus that allow linking between fractal calculus and fractional calculus , and aimed to attract more non-local natural problems that display at the same time fractal behaviors thus facilitating prediction of a complex systems (see [2]for more informations).This new operators will be called fractal-fractional differential and integral operators. With these new derivatives, researchers presented important works that studies the dynamics of epidemics, including : in [8] Sina Etemad and others have made some novel mathematical analyses on the fractal-fractional model of the AH1N1/09 virus and its generalized Caputo-type version, and in [19] we have a mathematical model of the transmission cycle of CC-Hemorrhagic fever via fractal-fractional operators and numerical simulations.

As we know, many differential problems have very complex analytical answers that we are unable to make use of ; therefore, it is necessary to use numerical approximations to better understand the results. Several researchers have proposed different methods of numerical approximation. The most widely used method in approximating solutions to nonlinear fractional differential equations that model epidemiological dynamics in the last few years has been the Adams-Bashforth numerical method, which is based on Lagrange polynomial and has given very satisfactory results, but recently, Abdon Atangana and Seda Igret Araz found that Newton polynomial is more accurate than Lagranges, so they proposed a new numerical scheme based on Newtons polynomial (see [16]for more information). Some researchers have used this new numerical method in their research, such as ; [14] studied the dynamics of CD4+ T-cells under the effect of HIV-1 infection based on a mathematical fractal-fractional model via the Adams-Bashforth scheme and Newton polynomials, a fractal-fractional model and numerical scheme based on Newton polynomials for Q fever disease under the Atangana-Baleanu derivative [3], and other limited papers such as ;[16].

The aim of this research is to analyze, study and explain the article[9] , while recalling the most important topics that are relied upon in this research In the first chapter, epidemiology was mentioned in general. The second chapter has been referred to some definitions of fractional derivatives The last chapter in which the main topic in the reference [9]was studied.

Notations

- ➤ $N(t)$ the total Population.
- ➤ $\mathbb{R}$ Set of real numbers.
- ➤ $\mathbb{N}$ the set of natural integers.
- ➤ $\mathbb{C}$ set of camplex numbers.
- ➤ Ω Omega set.
- ➤ R_0 the basic reproduction number.
- ➤ V^{-1} the invertible matrix of V.
- ➤ $J\mathcal{F}$ Jacobian matrix of F.
- ➤ FV^{-1} the next generation matrix.
- ➤$\rho(FV^{-1})$ the matrix spectral radius K
- ➤ DFE Disease-Free equilibrium point.
- ➤ EE point of endemic equilibrium .

Chapitre 1

MODELING IN EPIDEMIOLOGY

1.1 Introduction

A mathematical model is an abstract representation or interpretation of reality in different domains which is accessible to analysis and calculation based on a set of assumptions. Compartmental models are among the first mathematical models to have been used in epidemiology, which plays an important role in studying the evolution of infectious diseases and eradicating them and, at most, should make it possible to better understand epidemic phenomena and therefore better control them.

The idea has become to study demographic variations in societies. To model, it is first necessary to know the biology of the disease well [10] making a model of deterministic or stochastic compartments in discrete or continuous time depending on the disease to be studied, except the use of stochastic models is more complicated than the other. In this chapter, we are interested in this last type of model, which is based on two concepts : compartments and rules, compartments divide the population into various possible states by disease (susceptible, infected, etc.), rules specify the proportion of individuals moving from one compartment to another.

1.2 The classic models in compartments :

1.2.1 The SI, SIS model

The SI model is one of the classic models created by W. Hamar and developed in 1906 where individuals can be divided into two compartments :
The compartment or box of susceptible (healthy) individuals are receptive to the infectious agent who are not contaminated but can catch the disease and become contagious noted (S).

The compartment of infected individuals noted (I) are those affected and who are therefore infectious.
The infection is spread by direct contact between the susceptible and the infected. We see that in this model, there are no cures and is only relevant in incurable diseases or if the phenomenon of acquired immunity can be neglected [12]. An individual changes state (either infected or susceptible... etc.) he therefore changes his compartment with outgoing or incoming flows which indicate the rate of transfer between them. On the other hand, as the change in the number of infected people occurs over time, compartment I includes $I(t)$ and the same for $S(t)$. By the assumption of the constant of the size of the population the model is formed as follows :

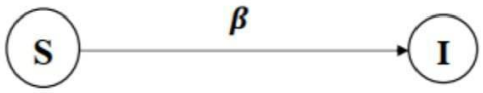

Figure 1.1 – SI model diagram.

The system of differential equations is written :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) \\ \dfrac{dI(t)}{dt} = \beta I(t)S(t) \end{cases} \tag{1.1}$$

With $N(t) = S(t) + I(t)$ is the total population and is constant through time t.
There are cases where susceptible becomes infected and the infected are cured at the rate γ but do not develop immunity and become susceptible like the case of tuberculosis, the following graph concisely summarizes the model :

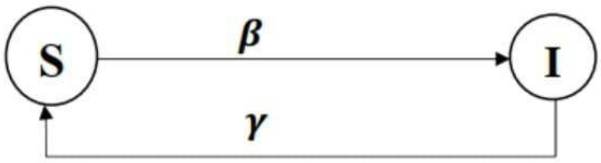

FIGURE 1.2 – The SIS model.

The associated differential equations are :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) + \gamma I(t) \\ \dfrac{dI(t)}{dt} = \beta I(t)S(t) - \gamma I(t) \end{cases} \tag{1.2}$$

With :

β : is the infection rate per unit time.

γ : the rate of each infected heals.

1.2.2 The SIR, SIRS model

The SIR model is the model proposed by kermack and MC Kendrick, consists of three categories of population : healthy people $S(t)$, infected people $I(t)$, recovered or cured people $R(t)$ who are conferred a immunization against reinfection or death.

The following figure schematizes the transfers of individuals between each group.

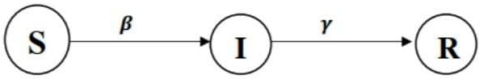

FIGURE 1.3 – SIR model.

Mathematically, the SIS model is given by the following system :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) \\ \dfrac{dI(t)}{dt} = \beta I(t)S(t) - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t) \end{cases} \tag{1.3}$$

Where :β is the transmission rate,γ is the recovery rate.

The term $\beta I(t)S(t)$ represents the number of contacts between healthy and infected people. On the other hand, we only encounter diseases, the individual has not acquired permanent immunization, he loses his immunity and returns to the S compartment at the rate η, this is the SIRS model schematizing as follows :

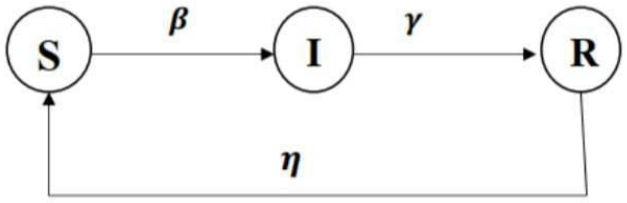

FIGURE 1.4 – The SIRS model.

This model is formulated as follows :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) + \eta R(t) \\ \dfrac{dI(t)}{dt} = \beta I(t)S(t) - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t) - \eta R(t) \end{cases} \tag{1.4}$$

With :

β : is the transmission rate. γ : the rate of each infected heals. η : the rate of loss of immunity (each removed becomes healthy again).

1.2.3 The SEI, SEIR model

The constitution of these models are based on a subpopulation is already infected but not yet contagious (non-infectious) i.e. susceptible subpopulations before go to class I, it requires spending a

period to make infectious s 'calls the latency period or incubation at an intermediate compartment denoted E(exposed), taking into account β the incubation rate of a disease. Schemes and ED are developed as follows :

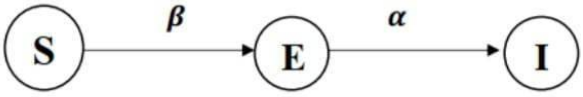

FIGURE 1.5 – SEI model.

The model is translated as follows :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) \\ \dfrac{dE(t)}{dt} = \beta I(t)S(t) - \alpha E(t) \\ \dfrac{dI(t)}{dt} = \alpha E(t) \end{cases} \tag{1.5}$$

As well as :

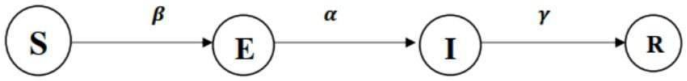

FIGURE 1.6 – SEIR model .

The EDO system is elaborated to the following :

$$\begin{cases} \dfrac{dS(t)}{dt} = -\beta I(t)S(t) \\ \dfrac{dE(t)}{dt} = \beta I(t)S(t) - \alpha E(t) \\ \dfrac{dI(t)}{dt} = \alpha E(t) - \gamma I(t) \\ \dfrac{dR(t)}{dt} = \gamma I(t) \end{cases} \tag{1.6}$$

1.3 The force of infection

The key parameter in all epidemiological models of infectious diseases is the force of infection. The latter accounts for the contamination process by expressing the probability that a susceptible individual will contract the disease so that each infected encounters at the rate C, each of these encounters with a type C individual causes contamination with the probability P, we score $\beta = CP$. It is this force of infection which moves individuals from compartment S to compartment I in the previous figures and which can be written in two different ways :

$$\Lambda = \beta I \tag{1.7}$$

If disease transmission increases with population density like (the influenza virus.

$$\Lambda = \frac{\beta I}{N} \tag{1.8}$$

If the transmission does not depend on it as (HIV).

1.4 The threshold theorem (basic reproduction number R_0) :

One of the first questions the epidemiologist asks is whether there is going to be an epidemic or not. The answer to this question in a very simple way by examining the system of differential equations. The first step is to translate our question into mathematical form. Then we calculate a quantity that describes the average number of secondary cases, generated by a typical infectious individual during his period of infectivity, when he is introduced into a population consisting entirely of susceptible, this quantity is called basic reproduction number, noted by R_0 , so the first idea of this number was by Théophile Lotz (1980) (Nichiura, Dietz, Eichner 2006). He finds that R_0 is a threshold and after Ross describes the first differential model and gives the threshold conditions as follows :
If $R_0 \leq 1$, then the disease-free point is globally stable, i.e. an individual infects on average less than one, which means that the disease disappears from the population. Conversely, if $R_0 > 1$, then the endemic point is globally stable, i.e. the disease can spread in the population. Note that is determined

according to the parameters of the model and later, it is used in the equilibrium stability theorems of the disease of the population.

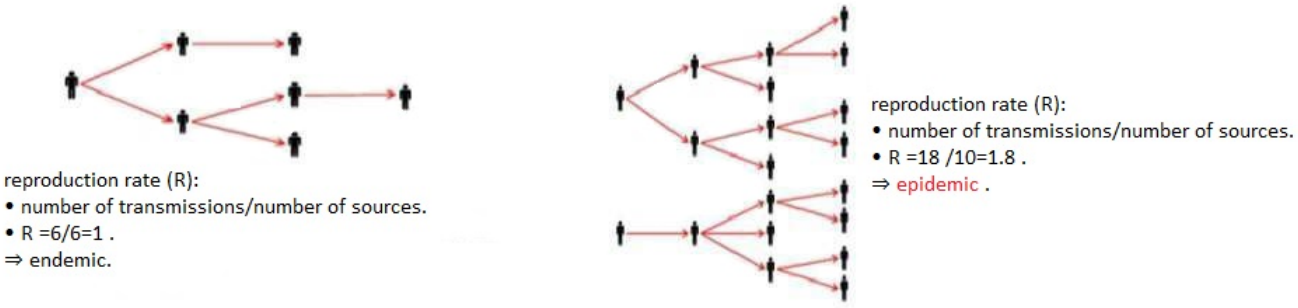

FIGURE 1.7 – transmission dynamics of an infectious disease between individuals to estimate secondary cases R_0

Chapitre 2

Some functions used in fractional calculus

2.1 The Euler gamma function

Definition 1 *One of the basic functions of fractional calculus is Euler's gamma function denoted* Γ *the function which is defined dy the following integral*

$$\Gamma(\alpha) = \int_0^{+\infty} t^{\alpha-1}\mathrm{e}^{-t}\mathrm{d}t, \quad \alpha > 0, \quad t \in \mathbb{R}$$

where $\Gamma(1) = 1$,$\Gamma(0_+) = +\infty$

Γ *is a strictly increasing function for* $0 < \alpha \leq 1$.

Example 2 *let calculate* $\Gamma(2)$ *:*

$$\begin{aligned}
\Gamma(2) &= \lim_{\varepsilon\to+\infty} \int_0^{\varepsilon} t^{2-1}\mathrm{e}^{-t}\mathrm{d}t \\
&= \lim_{\varepsilon\to+\infty} \int_0^{\varepsilon} t\mathrm{e}^{-t}\mathrm{d}t \\
&= \lim_{\varepsilon\to+\infty} \left[-t\mathrm{e}^{-t} - \mathrm{e}^{-t}\right]_0^{\varepsilon} \\
&= \lim_{\varepsilon\to+\infty} (-\frac{\varepsilon}{\mathrm{e}^{\varepsilon}} - \frac{1}{\mathrm{e}^{\varepsilon}} + 0 + \mathrm{e}^{0}) \\
&= 1.
\end{aligned}$$

2.1.1 Some useful properties of the Gamma function

Proposition 3

$$(1). \quad \Gamma(n+1) = n\Gamma(n), \quad \forall n \in \mathbb{N}^*$$

,

$$(2). \quad \Gamma(n+1) = n!, \quad \forall n \in \mathbb{N}^*,$$

$$(3). \qquad \Gamma\left(\frac{1}{2}\right) = \sqrt{\pi}.$$

Proof.

$$\begin{aligned}(1). \quad \Gamma(n+1) &= \int_0^{+\infty} t^{(n+1)-1}\mathrm{e}^{-t}\mathrm{d}t \\ &= \int_0^{+\infty} t^n\mathrm{e}^{-t}\mathrm{d}t \\ &= \left[-t^n\mathrm{e}^{-t}\right]_{t=0}^{t=+\infty} + n\int_0^{+\infty} t^{n-1}\mathrm{e}^{-t}\mathrm{d}t \\ &= n\Gamma(n).\end{aligned}$$

(2). Since $\Gamma(1) = 1$, hence by using (1) we get

$$\Gamma(2) = 1.\Gamma(1) = 1!$$

$$\Gamma(3) = 2.\Gamma(2) = 2.1! = 2!$$

$$\Gamma(4) = 3.\Gamma(2) = 3.2! = 3!$$

$$\Gamma(n+1) = n\Gamma(n) = n.(n-1)! = n!$$

which can easily be proved by induction.

(3).From Definition 2.1.1,we can write :

$$\Gamma\left(\frac{1}{2}\right) = \int_0^{+\infty} t^{-\frac{1}{2}}\mathrm{e}^{-t}\mathrm{d}t$$

If we take $t = y^2$ then, we obtain

$$\Gamma\left(\frac{1}{2}\right)) = 2\int_0^{+\infty} \mathrm{e}^{-y^2}\mathrm{d}y \tag{2.1}$$

In a similar way

$$\Gamma\left(\frac{1}{2}\right) = 2\int_0^{+\infty} \mathrm{e}^{-x^2}\mathrm{d}x \tag{2.2}$$

By multiplying(2.1)and(2.2)we get :

$$\left[\Gamma\left(\frac{1}{2}\right)\right]^2 = 4\int_0^{+\infty}\int_0^{+\infty} \mathrm{e}^{-(x^2+y^2)}\mathrm{d}x\mathrm{d}y \tag{2.3}$$

the last equation represents a double integral,which can be evaluated in polar coordinates to obtain :

$$\left[\Gamma\left(\frac{1}{2}\right)\right]^2 = 4\int_0^{\frac{\pi}{2}}\int_0^{+\infty} r\mathrm{e}^{-r^2}\mathrm{d}r\mathrm{d}\theta = \pi, \tag{2.4}$$

consequently,

$$\Gamma\left(\frac{1}{2}\right) = \sqrt{\pi}.$$

■

2.2 The Euler beta function

The beta function is defined by the Euler integral of the first Kind as

$$\beta(p,q) = \int_0^1 x^{p-1}(1-x)^{q-1}\mathrm{d}x, \quad p>0, \quad q>0. \tag{2.5}$$

Example 4 *Let calculate* $\beta(2,3)$

$$\begin{aligned}
\beta(2,3) &= \int_0^1 x(1-x)^2\mathrm{d}x \\
&= \int_0^1 x(1-2x+x^2)\mathrm{d}x \\
&= \int_0^1 x(1-2x^2+x^3)\mathrm{d}x \\
&= \left[\left(\frac{x^2}{2}-\frac{2x^3}{3}+\frac{x^4}{4}\right)\right]_0^1 \\
&= \frac{1}{2}-\frac{2}{3}+\frac{1}{4} \\
&= \frac{1}{12}
\end{aligned}$$

2.2.1 Relationship between the gamma and the beta function

The gamma and beta are connected by the following expression

$$\beta(p,q) = \frac{\Gamma(p)\Gamma(q)}{\Gamma(p+q)} \tag{2.6}$$

Proof. . Consider the set $D = [0,+\infty[\times[0,+\infty[$. We wave

$$\begin{aligned}\Gamma(p)\Gamma(q) &= \int_0^{+\infty} \mathrm{e}^{-x}x^{p-1}\mathrm{d}x \int_0^{+\infty} \mathrm{e}^{(-y)}x^{q-1}\mathrm{d}y \\ &= \int_0^{+\infty}\int_0^{+\infty} \mathrm{e}^{-(x+y)}x^{p-1}y^{q-1}\mathrm{d}x\mathrm{d}y\end{aligned}$$

performing the change of variables $y = u - x$, we find

$$\begin{aligned}\Gamma(p)\Gamma(q) &= \int_0^{+\infty}\int_0^{+\infty} \mathrm{e}^{-u}x^{p-1}(u-x)^{q-1}\mathrm{d}x\mathrm{d}y \\ &= \int_0^{+\infty} \mathrm{e}^{-u} \int_0^{+\infty} \mathrm{e}^{-u}x^{p-1}(u-x)^{q-1}\mathrm{d}x\mathrm{d}y\end{aligned}$$

let s use the change of variables $x = tu$, we obtain

$$\Gamma(p)\Gamma(q) = \int_0^{+\infty} \mathrm{e}^{-u} \int_0^{1} t^{p-1}u^{p-1}(1-t)^{q-1}u^q\mathrm{d}t\mathrm{d}u$$

$$= \int_0^{+\infty} \mathrm{e}^{-u}u^{p+q-1}\mathrm{d}u \int_0^{1} t^{p-1}(1-t)^{q-1}\mathrm{d}t$$

$\Gamma(p)\Gamma(q) = \Gamma(p+q)\beta(p,q)$

consequently,

$$\beta(p,q) = \frac{\Gamma(p)\Gamma(q)}{\Gamma(p+q)} \tag{2.7}$$

■

Example 5

$$\beta(2,3) = \frac{\Gamma(2)\Gamma(3)}{\Gamma(2+3)}\frac{1!2!}{4!} = \frac{1}{12}$$

2.2.2 Some properties of the beta function

$$\beta(p,q) = \beta(q,p)$$

We can also take the form of an integral

$$\beta(p,q) = 2\int_0^1 (\sin\theta)^{2p-1}(\cos\theta)^{2q-1}\mathrm{d}\theta$$

withe the change of variables $t = \sin^2\theta$

the Gamma function can be represented also by the limit

$$\Gamma(z) = \lim_{n\to+\infty} \frac{n!n^z}{z(z+1)...(z+n)}$$

where we assume that $Re(z) > 0$.

2.2.3 The Mittag-Leffler function

The Mittag-Leffler function plays a very important role in the theory of whole order differential equations, and it is found widely used in solving fractional differential equations. This function was presented by G. M. Mittag-Leffler, and studied by A. Wiman.

Definition 6 *The Mittag-Leffler function $E_\alpha(x)$ is defined by :*

$$E_\alpha(x) = \sum_{n=0}^{+\infty} \frac{x^n}{\Gamma(n\alpha+1)}, (x \in \mathbb{C}, \alpha > 0), \tag{2.8}$$

and the generalized Mittag-Leffler function $E_{\alpha,\beta}(z)$ is defined as follows :

$$E_{\alpha,\beta}(x) = \sum_{n=0}^{+\infty} \frac{x^n}{\Gamma(n\alpha+\beta)}, \quad (\alpha,\beta > 0), \tag{2.9}$$

Example 7 *For special values given to α and β we have :*

$$E_{1,1}(x) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(k+1)} = \sum_{k=0}^{\infty} \frac{x^k}{k!} = e^x.$$

$$E_{1,2}(x) = \sum_{k=0}^{\infty} \frac{x^k}{\Gamma(k+2)} = \sum_{k=0}^{\infty} \frac{x^k}{(k+1)!} = \frac{1}{x}\sum_{n=0}^{\infty} \frac{x^{k+1}}{(k+1)!} = \frac{e^x-1}{x}.$$

$$E_{1,3}(x) = \sum_{k=0}^{\infty} \frac{x^k}{\Gamma(k+3)} = \sum_{k=0}^{\infty} \frac{x^k}{(k+2)!} = \frac{1}{x^2} \sum_{k=0}^{\infty} \frac{x^{k+2}}{(k+2)!} = \frac{e^x - 1 - x}{x^2}.$$

2.3 Caputo fractional derivatives

Definition 8 *Let f a function such that $\frac{d^n}{dt^n} f \in L^1([a,b])$ and $\alpha \in]n-1, n[$ with $n \in \mathbb{N}^*$. The fractional derivative of order α of f in the Caputo sense on the **left** and on the **right** are defined by :*

$$^C\mathcal{D}^{\alpha}_{a^+} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_a^t (t-r)^{n-\alpha-1} f^{(n)}(r) d\tau, \tag{2.10}$$

and

$$^C\mathcal{D}^{\alpha}_{b^-} f(t) = \frac{(-1)^n}{\Gamma(n-\alpha)} \int_t^b (r-t)^{n-\alpha-1} f^{(n)}(r) d\tau, \tag{2.11}$$

respectively.

Remark 9

Taking into account the Definition 8, we have :

$$^C\mathcal{D}^{\alpha}_{a^+} f(t) = (I^{n-\alpha}_{a^+} D^n f)(t), \tag{2.12}$$

and

$$^C\mathcal{D}^{\alpha}_{b^-} f(t) = (-1)^n (\mathcal{I}^{n-\alpha}_{b^-} D^n f)(t). \tag{2.13}$$

Specifically, when $o < \alpha < 1$ we have :

$$^C\mathcal{D}^{\alpha}_{a^+} f(t) = (\mathcal{I}^{1-\alpha}_{a^+} D^1 f)(t), \tag{2.14}$$

and

$$^C\mathcal{D}^{\alpha}_{b^-} f(t) := (-1)(\mathcal{I}^{1-\alpha}_{b^-} D^1 f)(t). \tag{2.15}$$

If $\alpha = n \in \mathbb{N}$ and the usual derivative $f^{(n)}(t)$ of order n exists, then $^C\mathcal{D}^n_{a^+}$ and $^C\mathcal{D}^n_{b^-}$ are represented by :

$$^C\mathcal{D}^n_{a^+} = f^{(n)}(t) \quad and \quad ^C\mathcal{D}^n_{b^-} = (-1)^n f^{(n)(t)}, \tag{2.16}$$

where

$$\mathcal{D}^n = \frac{d^n}{dt^n}.$$

Example 10 — ***The derivative of a constant function in the Caputo sense.***

The derivative of a constant function in the Caputo sense is zero

$$^{C}\mathcal{D}^{\alpha}C = 0. \tag{2.17}$$

— ***The derivative of*** $f(t) = (t-a)^{\beta}$ ***in the Caputo sense.***

Let α *be an integer and* $0 \leq n-1 < \alpha < n$ *with* $\beta > n-1$*, then we have*

$$f^{(n)}(t) = \frac{\Gamma(\beta+1)}{\Gamma(\beta-n+1)}(t-a)^{\beta-n}, \tag{2.18}$$

hence

$$^{C}\mathcal{D}^{\alpha}(t-a)^{\beta} = \frac{\Gamma(\beta+1)}{\Gamma(n-\alpha)\Gamma(\beta-n+1)}\int_{a}^{t}(t-r)^{n-\alpha-1}(r-a)^{\beta-n}d\tau, \tag{2.19}$$

When changing the variable $r = a + r(t-a)$*, we get*

$$\begin{aligned}
^{C}\mathcal{D}^{\alpha}(t-a)^{\beta} =& \frac{\Gamma(\beta+1)}{\Gamma(n-\alpha)\Gamma(\beta-n+1)}\int_{a}^{t}(t-r)^{n-\alpha-1}(r-a)^{\beta-n}dr \\
=& \frac{\Gamma(\beta+1)}{\Gamma(n-\alpha)\Gamma(\beta-n+1)}(t-a)^{\beta-\alpha}\int_{a}^{1}(1-r)^{n-\alpha-1}r^{\beta-n}dr \\
=& \frac{\Gamma(\beta+1)B(n-\alpha,\beta-n+1)}{\Gamma(n-\alpha)\Gamma(\beta-n+1)}(t-a)^{\beta-\alpha} \\
=& \frac{\Gamma(\beta+1)\Gamma(n-\alpha)\Gamma(\beta-n+1)}{\Gamma(n-\alpha)\Gamma(\beta-n+1)\Gamma(\beta-\alpha+1)}(t-a)^{\beta-\alpha} \\
=& \frac{\Gamma(\beta+1)}{\Gamma(\beta-\alpha+1)}(t-a)^{\beta-\alpha}.
\end{aligned}$$

2.3.1 Properties of the fractional derivation

Theorem 11 *Let* $\alpha > 0$ *and* $n = [\alpha]+1$ *such that* $n \in \mathbb{N}^{*}$ *then the following equations :*

1/

$$^{C}\mathcal{D}^{\alpha}\mathcal{I}_{a}^{\alpha}f = f. \tag{2.20}$$

2/

$$\mathcal{I}_{a}^{\alpha}(^{C}\mathcal{D}^{\alpha}f(t)) = f(t) - \sum_{k=0}^{n-1}\frac{f^{(k)}(a)(t-a)^{k}}{k!}, \tag{2.21}$$

are true for almost for all $t \in [a,b]$*.*

Theorem 12 *Let f and g be two functions whose fractional derivatives of Caputo exist, for λ and $\mu \in \mathbb{R}$, then : ${}^{C}\mathcal{D}^{\alpha}(\lambda f + \mu g)$ exists, and we have :*

$$ {}^{C}\mathcal{D}^{\alpha}(\lambda f(t) + \mu g(t)) = \lambda {}^{C}\mathcal{D}^{\alpha} f(t) + \lambda {}^{C}\mathcal{D}^{\alpha} g(t). $$

Chapitre 3

Mathematical modeling and theoretical model analysis

3.1 Introduction

Since long time ago, scientists and researchers have sought to control the epidemics sweeping the world each in its specialty. With a mixture of mathematics and data, mathematicians have provided important work that allows us to better understand the way epidemics spread, and thus help in developing precautionary measures and health policies to confront them. In order to better understand how mathematics contributes to the analysis of epidemiological systems. We do a study HIV dynamics with fractional operator of Caputo type. First, we give brief mathematical formulation of HIV/AIDS population in integer order derivative. Then, we present some background results related to the model. The integer model is then generalized by using the Caputo derivative and present the mathematical results that associated to the model. We use novel technique for the solution of fractional mathematical model of HIV using Newton polynomial approach and obtain the numerical solution graphically.

3.2 HIV MODEL FORMULATION

The section determine the model formulation of the HIV transmission with awareness effect. The HIV model is divided into five human populations, susceptible unaware class (S_u), the susceptible aware class (S_a), the HIV infected class (I), the HIV infected under ART treatment class (C) and individuals with AIDS class (A).Thus the total population denoted by N is given as $N(t) = S_u + S_a + I + C + A$.

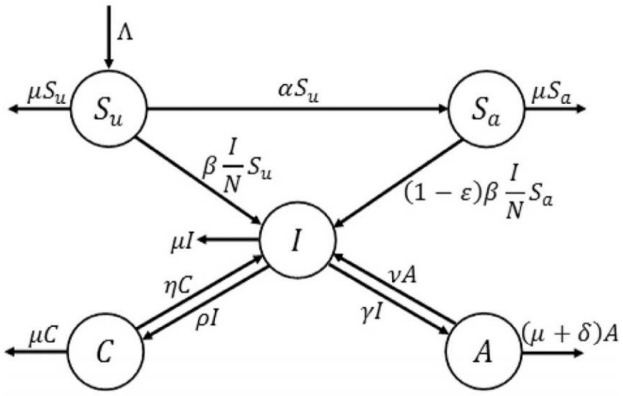

FIGURE 3.1 – The plot represents the HIV/AIDS transition diagram.

Table 1 : Descriptions of the parameters of the model

parameter	Description
Λ	Recruitment rate
β	Transmission rate by HIV
α	Change rate from S_u to S_a
μ	Natural death rate
ε	The proportion of S_a become infected
η	Rate of individuals in the class C leave to the class I
ν	Transition rate from A to I
ρ	Rate at which I leads to C
γ	Progression rate from I to A
δ	AIDS induced death rate

3.2.1 Interaction between HIV compartment individuals

The assumption used in the mathematical model of the spread of HIV/AIDS in the presence of an aware population are as follows :

1. S_u populations can move to S_a populations, but not vice versa .

2. Population C is assumed not to spread HIV because it is considered to be aware of the dangers of HIV by following routine ART treatment .

3. Population A is assumed not to spread HIV because the AIDS population is considered to be so ill (until isolated).

4. The population of HIV and AIDS sufferers is considered to be able to access ART treatment.

5. Population A who starts ART treatment will enter the HIV infected class (I) ,and if they continue to routinely follow treatment will enter population C.

6. The death rates due to HIV/AIDS only occur in populations affected by AIDS.

The completing information about the flow of the parametrs from one compartment to another is briefly schown in **figure** (3.1).

The system of differential equations describing the HIV model with awareness effect is described as :

$$\begin{cases} \frac{dS_u}{dt} = \Lambda - \frac{\beta S_u I}{N}(\alpha + \mu)S_u \\ \frac{dS_a}{dt} = \alpha S_u - (1-\varepsilon)\frac{\beta S_a I}{N} - \mu S_a \\ \frac{dI}{dt} = \frac{\beta I}{N}(S_u + (1-\varepsilon)S_a) + \eta C + \nu A - (\rho + \gamma + \mu)I \\ \frac{dC}{dt} = \rho I - (\eta + \mu)C, \\ \frac{dA}{dt} = \gamma I - (\nu + \delta + \mu)A. \end{cases} \tag{3.1}$$

subject to the initial conditions,

$$\begin{cases} S_u(0) = S_{u0} \geq 0 \\ S_a(0) = S_{a0} \geq 0 \\ I(0) = I_0 \geq 0 \\ C(0) = C_0 \geq 0 \\ A(0) = A_0 \geq 0 \end{cases} \tag{3.2}$$

the description of the parameters for the HIV model is displayed in table 1.

the model(3.1) has the biologically feasible region on Ω withe

$$\Omega = \left\{ (S_u, S_a, I, C, A) \in \mathbb{R}^5_+ : 0 \leq N \leq \frac{\Lambda}{\mu} \right\}$$

It should be noted that the region shown by Ω in positively invariant .The region shown for the model(3.1) is well-posed and the entire solutions for the initial values belonging to Ω, remains in Ω for every time $t \geq 0$.

3.3 A FRACTIONAL MODEL

The present section describes the dynamics of HIV with fraction derivative of Caputo type. We generalize the model(3.1) by applying the definition of Caputo and obtained the following system :

$$\begin{cases} {}_0^C D_t^p S_u = \Lambda - \frac{\beta S_u I}{N}(\alpha + \mu) S_u \\ {}_0^C D_t^p S_a = \alpha S_u - (1-\varepsilon)\frac{\beta S_a I}{N} - \mu S_a \\ {}_0^C D_t^p I = \frac{\beta I}{N}(S_u + (1-\varepsilon)S_a) + \eta C + \nu A - (\rho + \gamma + \mu) I \\ {}_0^C D_t^p C = \rho I - (\eta + \mu) C, \\ {}_0^C D_t^p A = \gamma I - (\nu + \delta + \mu) A. \end{cases} \tag{3.3}$$

$$\begin{cases} S_u(0) = S_{u0} \geq 0 \\ S_a(0) = S_{a0} \geq 0 \\ I(0) = I_0 \geq 0 \\ C(0) = C_0 \geq 0 \\ A(0) = A_0 \geq 0 \end{cases}$$

3.4 POSITIVE OF THE SOLUTION

Theorem 13 *The solution (S_u, S_a, I, C, A) of the given model (3.1)-(3.3) are positive and belongs to $\mathbb{R}^5_+$.*

Proof.

1) For the first equation we have :

$${}_0^c D_t^p S_u = \Lambda - \frac{\beta S_u I}{N} - (\alpha + \mu) S_u \tag{3.4}$$

First we have : $N > I$ then $\frac{I}{N} < 1$ the equation (3.4) becomes :

$${}_0^cD_t^pS_u = \Lambda - \beta S_u - (\alpha + \mu)S_u$$

$$\Longrightarrow {}_0^cD_t^pS_u(t) \geq -(\beta + \alpha + \mu)S_u(t)$$

we solve the inequality using the Laplac Transform (L.T)

$$L({}_0^cD_t^pS_u(t)) = -L(\beta + \alpha + \mu)S_u(t) \tag{3.5}$$

on the one hand we have the L.T of the caputo derivative

$$L\{{}_0^cD_t^pf(t)\} = S^pF(s) - \sum_{k=0}^{n-1} S^{\alpha-k-1}f^{(k)}(0)$$

since $0 < p < 1$ permission $n = [\alpha] + 1 = 1$
hence L.T derivative caputo be

$$L\{{}_0^cD_t^pf(t)\} = S^pF(s) - \sum_{k=0}^{n-1} S^{p-1}f^{(k)}(0)$$

we go back to (3.5)
$L^cD^pS_u(t) = -L(\beta + \alpha + \mu)S_u(t)$

$$\Longrightarrow Lt^pS_u(t) - t^{p-1}S_u^{(0)}(0) = -(\beta + \alpha + \mu)S_u(t)LS_u(0)$$

$$\Longrightarrow (t^p + \beta + \alpha + \mu)LS_u(t) = t^{p-1}S_u^{(0)}(0)$$

$$\Longrightarrow LS_u(t) = \frac{t^{p-1}}{t^p + (\beta + \alpha + \mu)}S_u(0)$$

we use Laplace transform inverse

$$\Longrightarrow LS_u(t) = L^{-1}\left(\frac{t^{p-1}}{t^p + (\beta + \alpha + \mu)}S_u(0)\right)$$

now from the L.T table we find that

$$\frac{S^{\alpha-\beta}}{S^{\alpha}-a} = t^{\beta-1}.E_{\alpha,\beta}(at^{\alpha}) \tag{3.6}$$

we use (3.6) to find

$S_u(t) = S(0), E_p(-(\beta+\alpha+\mu)t^p)$

since we have $S_u(0) > 0$ and $0 \leq E_p(-(\beta+\alpha+\mu)) \leq 1$

then $S_u(t) \geq S_u(0)E_p(-(\beta+\alpha+\mu)t^p) \geq 0$

2) ${}_0^cD_t^pS_a(t) = \alpha S_u(t) - (1-\varepsilon)\frac{\beta S_a I}{N} - \mu S_a(t) \geq -(((1-\varepsilon)\beta+\mu)S_a(t))$

It follows that $S_a(0) > 0$ and $0 \leq E_p(-((1-\varepsilon)\beta+\mu)) \leq 1$

$E_p(-((1-\varepsilon)\beta+\mu)t^p) \geq 0$

3) ${}_0^cD_t^pI(t) = \frac{\beta I}{N}(S_u + (1-\varepsilon)S_a) + \eta C + \nu A - (\rho+\gamma+\mu)I(t) \geq -(\rho+\gamma+\mu)I(t)$

Wich implies $I(0) > 0$ and $0 \leq E_p(-(\rho+\gamma+\mu)) \leq 1$

$E_p(-(\rho+\gamma+\mu)t^p) \geq 0$

4) ${}_0^cD_t^pC(t) = \rho I - (\eta+\mu)C(t) \geq -(\eta+\mu)C(t)$,

This gives $C(0) > 0$ and $0 \leq E_p(-(\eta+\mu)) \leq 1$

$E_p(-(\eta+\mu)t^p) \geq 0$

5) ${}_0^cD_t^pA(t) = \gamma I - (\nu+\delta+\mu)A(t) \geq -(\nu+\delta+\mu)A(t).$

This $A(0) > 0$ and $0 \leq E_p(-(\nu + \delta + \mu)) \leq 1$

$E_p(-(\nu + \delta + \mu)t^p) \geq 0$

The solution (S_u, S_a, I, C, A) of the given models (3.3)are positive and belongs to $\mathbb{R}^5_+$.

■

3.5 Analysis of model equilibria :

We have analyzed the existence and stability of all equilibria for this model which are composed of two states of equilibria :

- The point of equilibrium without disease DFE (Disease-Free Equilibrium).
- The endemic equilibrium point EE.

Definition 14 *The endemic equilibrium point is an equilibrium solution where the disease persists in the population.*

Definition 15 *The disease-free equilibrium point DFE is an equilibrium point where disease occurs in the population.*

3.5.1 Equilibrium point without DFE disease :

Theorem 16 *The system (3.3) admits a point of equilibrium called « point of equilibrium without disease »,given by :*

$$E_0 = \left(\frac{\Lambda}{\mu + \alpha}, \frac{\alpha\Lambda}{\mu(\mu + \alpha)}, 0, 0, 0\right)$$

Proof. In the absence of HIV the invectious compartment (exposed symptomatic and asymptomatic)are empty that :

$$I(t) = C(t) = A(t) = 0$$

$$\begin{cases} {}_0^c D_t^p S_u = \Lambda - \frac{\beta S_u I}{N} - (\alpha + \mu) S_u = 0 \\ \\ {}_0^c D_t^p S_a = \alpha S_u - (1-\varepsilon)\frac{\beta S_a I}{N} - \mu S_a = 0 \end{cases} \tag{3.7}$$

From(3.7), we get

$$S_u = \frac{\Lambda}{\mu + \alpha} \, ; \, S_a = \frac{\alpha \Lambda}{\mu(\mu + \alpha)}.$$

then

$$E_0 = \left(\frac{\Lambda}{\mu + \alpha}, \frac{\alpha \Lambda}{\mu(\mu + \alpha)}, 0, 0, 0 \right)$$

■

3.5.2 The estimated basic reproduction number R_0 :

Basic reproductive number control is the central concept in the study of the spread of communicable diseases. Biologically, it is the number of secondary infections caused by a single infectious in a population, this number is calculated at the DFE by the method of **Van den Drissche** and **Watmough** (the next generation method) [18]. We have in this method a matrix $F(X)$ for the rate of appearance of new cases of infections and $V(X)$ for the rate of transfer of individuals, for this, we assemble the compartments which are infected $(I(t), C(t), A(t))$ by the system and we compose the right part in $\dfrac{dX}{dt} = F(X) - V(X)$

$$\mathcal{F} = \begin{pmatrix} \beta I(S_u + (1-\varepsilon)S_a) \\ 0 \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} -\eta C - \nu A - (\rho + \gamma + \mu) I \\ -\rho I + (\eta + \mu) C \\ -\gamma I + (\nu + \delta + \mu) A \end{pmatrix}$$

Then : the Jacobian matrices F and V at the point $E_0 = \left(\dfrac{\Lambda}{\mu + \alpha}, \dfrac{\alpha \Lambda}{\mu(\mu + \alpha)}, 0, 0, 0 \right)$ (DFE) of $\mathcal{F}$ and $\mathcal{V}$ are respectively :

$$F = J(\mathcal{F}) = \frac{\partial \mathcal{F}}{\partial X} = \begin{pmatrix} \frac{\beta(\mu + (1-\varepsilon)\alpha)}{\mu + \alpha} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$V = J(\mathcal{V}) = \frac{\partial \mathcal{V}}{\partial X} = \begin{pmatrix} \rho + \gamma + \mu & -\eta & -\nu \\ -\rho & \eta + \mu & 0 \\ -\gamma & 0 & \mu + \delta + \nu \end{pmatrix},$$

The invertible matrix of V is :

$$V^{-1} = \begin{pmatrix} \frac{k_2 k_1}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & \frac{\eta k_1}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & \frac{k_2\gamma}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} \\ \frac{\rho\eta}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & \frac{(\rho+\gamma+\mu)k_1 - \gamma\nu}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & -\frac{\rho\nu}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} \\ \frac{\gamma k_2}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & \frac{\gamma\eta}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} & \frac{(\rho+\gamma+\mu)k_2 - \rho\eta}{(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2} \end{pmatrix}$$

Where $k_1 = \mu + \delta + \nu$ and $k_2 = \eta + \mu$.

The matrix of the new generation is defined by :

$$FV^{-1} = \begin{pmatrix} \frac{\beta(\mu + (1-\varepsilon)\alpha)k_2k_1}{(\mu+\alpha)[(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2]} & \frac{\beta(\mu + (1-\varepsilon)\alpha)\eta k_1}{(\mu+\alpha)[(\rho+\gamma+\mu)k_2k_1 - \eta\rho k_1 - \nu\gamma k_2]} & \frac{\beta(\mu +}{(\mu+\alpha)[(\rho+\gamma} \\ 0 & 0 & \\ 0 & 0 & \end{pmatrix}$$

Hence the value of R_0 is mathematically defined as the spectral radius of FV^{-1}, the largest eigenvalue of matrix FV^{-1}.

$R_0 = \rho(FV^{-1})$ so the

$$R_0 = \frac{\beta[\mu + (1-\varepsilon)\alpha]k_1k_2}{(\mu+\alpha)\left(\mu[k_2(k_1+\gamma) + \rho k_1 + \gamma\delta] + \eta\gamma\delta\right)}$$

3.5.3 Stability of equilibrium point :

DFE local stability :

Theorem 17 *The disease-free equilibrium (DFE)* $E_0 = \left(\frac{\Lambda}{\mu+\alpha}, \frac{\alpha\Lambda}{\mu(\mu+\alpha)}, 0, 0, 0\right)$ *of system (3.3) is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. We analyze the local stability of the DFE E_0. the Jacobian of the model (3.3) at the DFE is given by

$$J(E_0) = \begin{pmatrix} -(\mu+\alpha) & 0 & -\frac{\beta\mu}{\mu+\alpha} & 0 & 0 \\ \alpha & -\mu & -\frac{\beta\alpha(1-\varepsilon)}{\mu+\alpha} & 0 & 0 \\ 0 & 0 & k_3 - k_4 & \eta & \nu \\ 0 & 0 & \rho & -k_2 & 0 \\ 0 & 0 & \gamma & 0 & -k_1 \end{pmatrix}$$

Where $k_3 = \frac{\beta(\mu + (1-\varepsilon)\alpha)}{\mu+\alpha}$ and $k_4 = \rho + \gamma + \mu$.
The eigenvalues of $J(E_0)$ are $\lambda_1 = -\mu$,$\lambda_2 = -(\mu+\alpha)$ and the other three eigenvalues are solution of

$$\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0 \tag{3.8}$$

Where
$b_1 = k_1 + k_2 - k_3 + k_4 = k_1 + k_4 + \eta + \frac{(\mu k_1 k_2 - MR_0)}{k_1 k_2}$,
$b_2 = k_1k_2 + (k_1 + k_2)(k_4 - k_3) - (\rho\eta + \gamma\nu)$,
$b_3 = (1 - R_0)(\mu + \alpha)\,(\mu[k_2(\gamma + k_1) + \rho k_1 + \gamma\delta] + \eta\gamma\delta)$,
$M = (\mu[k_2(k_1 + \gamma) + \rho k_1 + \gamma\delta] + \eta\gamma\delta)$.

Using Routh-Hurwitz criteria,we will examine that all the solutions of Eq(3.8) have negative real parts if only if $b_1, b_3 > 0$ and $b_1, b_2 > b_3$. The coefficient $b_1 > 0$ if $(\mu k_1 k_2 - MR_0) > 0$ or equivalent to $R_0 < \frac{\mu k_1 k_2}{M} < 1$, while $b_3 > 0$ if $R_0 < 1$. Now,

$b_1b_2 - b_3 = (k_1 + k_2 - k_3 + k_4)\,(k_1k_2 + (k_1 + k_2) + (k_4 - k_3) - (\rho\eta + \gamma\nu))$
$-\,(1 - R_0)(\mu + \alpha)\,(\mu[k_2(\gamma + k_1) + \rho k_1 + \gamma\delta] + \eta\gamma\delta)$

$$= \frac{M}{k_1 k_2}\left(\frac{\mu k_1 k_1 k_2}{M} - R_0(k_1 + k_2 + 2k_4)\right)$$

$$= \frac{M(k_1 + k_2 + 2k_4)}{k_1 k_2}(R_1 - R_0)$$

With

$$\mathrm{R}_1 = \frac{\mu k_1^2 k_2}{M(k_1 + k_2 + 2k_4)} = \frac{\mu k_1^2 k_2}{(\mu[k_2(k_1 - \gamma) + \rho k_1 + \gamma\delta] + \eta\gamma\delta)(k_1 + k_2 + 2k_4)} < \frac{\mu k_1^2 k_2}{\mu k_1^2 k_2} = 1.$$

So, $b_1 b_2 - b_3 > 0$ in only if $R_0 < R_1 < 1$.

■

3.5.4 The endemic equilibrium point EE :

During the propagation of HIV , system (3.3) admits another equilibrium point which coexists with the disease-free equilibrium point given by :

$$E_1 = (S_u^*, S_a^*, I^*, C^*, A^*)$$

Obtaining the endemic equilibrium point tends to solve the following equations :

$$\begin{cases} \Lambda - \frac{\beta S_u I}{N}(\alpha + \mu)S_u = 0 \\ \alpha S_u - (1 - \varepsilon)\frac{\beta S_a I}{N} - \mu S_a = 0 \\ \frac{\beta I}{N}(S_u + (1 - \varepsilon)S_a) + \eta C + \nu A - (\rho + \gamma + \mu)I = 0 \\ \rho I - (\eta + \mu)C = 0 \\ \gamma I - (\nu + \delta + \mu)A = 0 \end{cases} \tag{3.9}$$

Solving the system above, we find the endemic equilibrium point :

$$E_1 = (S_u^*, S_a^*, I^*, C^*, A^*)$$

$$\begin{cases} S_u^* = \dfrac{\Lambda}{k^* + \mu + \alpha} \\ S_a^* = \dfrac{\varepsilon\Lambda}{((1-\varepsilon)k^* + \mu)(k^* + \mu + \alpha)} \\ I^* = \dfrac{K_1 K_2 k^* \Lambda((1-\varepsilon)(k^* + \alpha) + \mu)}{((1-\varepsilon)k^* + \mu)(k^* + \mu + \alpha)M} \\ C^* = \dfrac{K_1 \rho k^* \Lambda((1-\varepsilon)(k^* + \alpha)\mu)}{((1-\varepsilon)k^* + \mu)(k^* + \mu + \alpha)M} \\ A^* = \dfrac{K_2 \gamma k^* \Lambda((1-\varepsilon)(k^* + \alpha)\mu)}{((1-\varepsilon)k^* + \mu)(k^* + \mu + \alpha)M} \end{cases} \tag{3.10}$$

where
$K^* = \dfrac{\beta I^*}{N^*}$.
Using some algebraic calculation, we have
$a_1 k^{*2} + a_2 k^* + a_3 = 0,$
where
$a_1 = (1-\varepsilon),$
$a_2 = (1-\varepsilon)(\mu+\alpha) + \mu - \left(\dfrac{(1-\varepsilon)(\mu+\alpha)\mu R_0}{\mu + (1-\varepsilon)\alpha}\right)$
$a_3 = \mu(\mu+\alpha)(1-R_0).$
Here, coefficient a_1 is always positive, while the positivity of the cofficient a_3 depends on the sign of R_0. If $R_0 < 1$ then $a_3 > 0$ while $R_0 > 1$,then $a_3 < 0$.More discussion on these can be shown in the following statement :

Theorem 18 *For the HIV system (3.3) the following are exists :*
(i) if $a_3 < 0$ or $R_0 > 1$, then there exists a unique endemic equilibrium,
(ii) $a_2 < 0$ and eiter $a_3 = 0$ or $a_2^2 - 4a_1a_3 = 0$, then we have a unique endemic equilibrium,
(iii) if $a_3 > 0$, $a_2 < 0$ and $a_2^2 - 4a_1a_3 > 0$, then, the possibility of the two endemic equilibria
(iv) no possibility of the endemic equilibia otherwise.

Numerical Simulation

Table 2 : Parameter values of HIV model (3.3) for Indonesia.

parameter	Value	reference
Λ	$\frac{229.800.000}{6.7.39}$	Estimated
β	0.3465	Fitting
α	0.2351	Fitting
μ	$\frac{1}{67.39}$	Estimate
ε	0.3243	Fitting
η	0.2059	Fitting
ν	0.7661	Fitting
ρ	0.1882	Fitting
γ	3.6523e-04	Fitting
δ	0.7012	Fitting

A brief explanation of the numerical scheme is shown below for the fractional differential equation :

$$ {}_0^C D_t^P z(t) = f(t, z(t)) \tag{3.11} $$

where the derivative is Caputo and f represents a nonlinear function. In order to have a numerical algorithm for the solution of the above fractional differential equations, we reformulate the problem given by :

$$ z(t) - z(0) = \frac{1}{\Gamma(p)} \int_0^t (t-\tau)^{p-1} f(\tau, z(\tau)) d\tau, \qquad see[9] \tag{3.12} $$

Our purpose in this part is to analyze the dynamics of HIV transmission in the presence of aware individuals under the influence of the change in the fractional order. We carried out the numerical simulations for our fractional model using the numerical method based on the Newton polynomial approach [1]. We used the parameter values listed in [13] to obtain the graphical results.The numerical

simulation is performed for the classical order ($P = 1$), and for various values of the fractional order ($P = 0.98, P = 0.96$, and $P = 0.8$).

In **figure** (3.2), we see that when the number of susceptible aware people increases, the number of susceptible unaware,infected, and individuals with AIDS decreases. On the other hand, the number of people under ART treatment increases.The fractional order has a clear effect on the spread of HIV infection in the community, such that we observe in the five groups that the growth and decay rates are faster in the small value of the memory index P compared to the larger values.

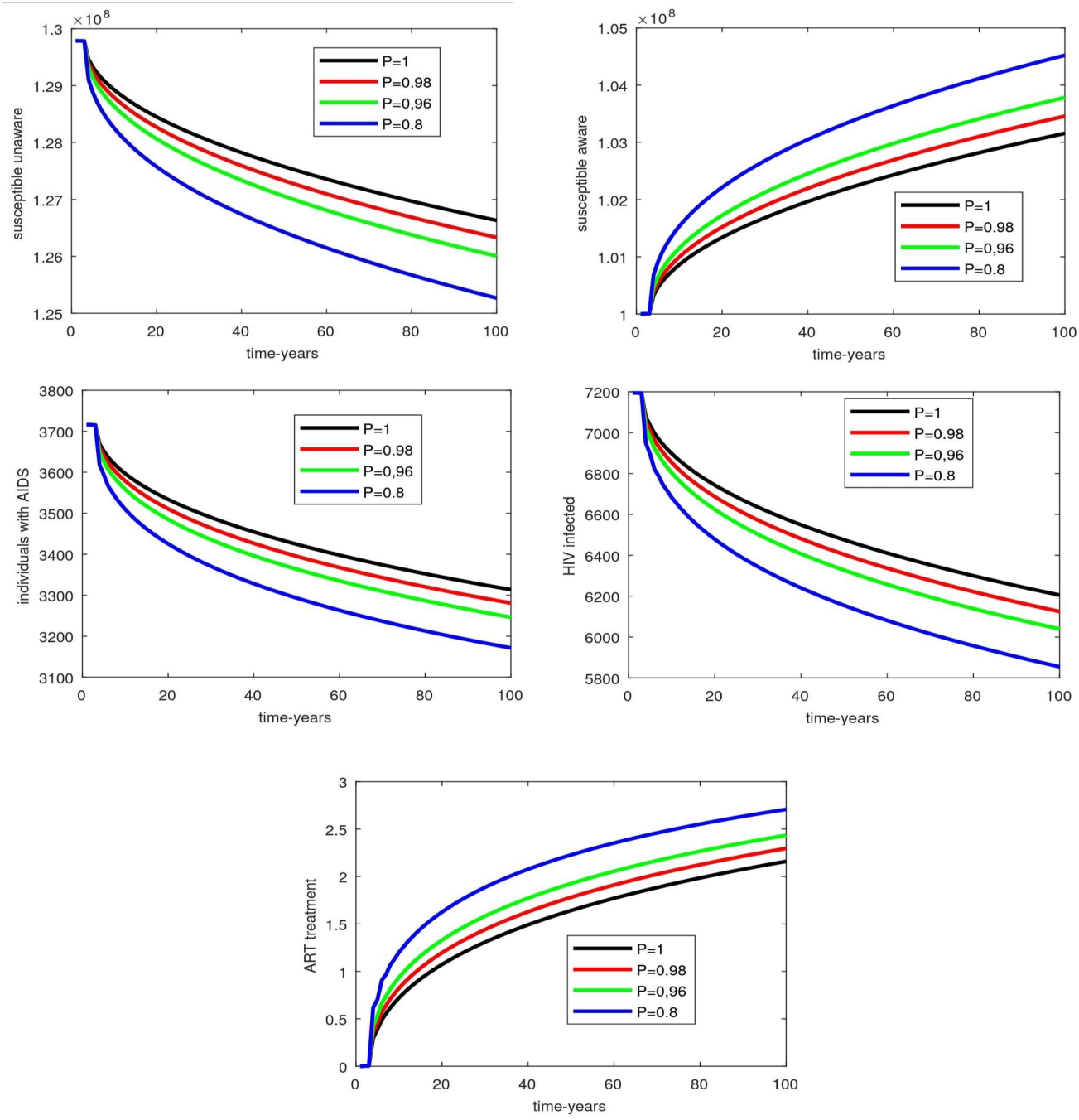

FIGURE 3.2 – dynamics of the fractional model for different values of fractional order P

To determine the effect of the transmission rate β on the spread of HIV among people, we performed a numerical simulation of infected classes for different values of transmission rate β, and the fractional order P. The graphical results are illustrated in **figure**(3.3).

We observed that the high value of β led to an increase in the number of people infected with HIV .

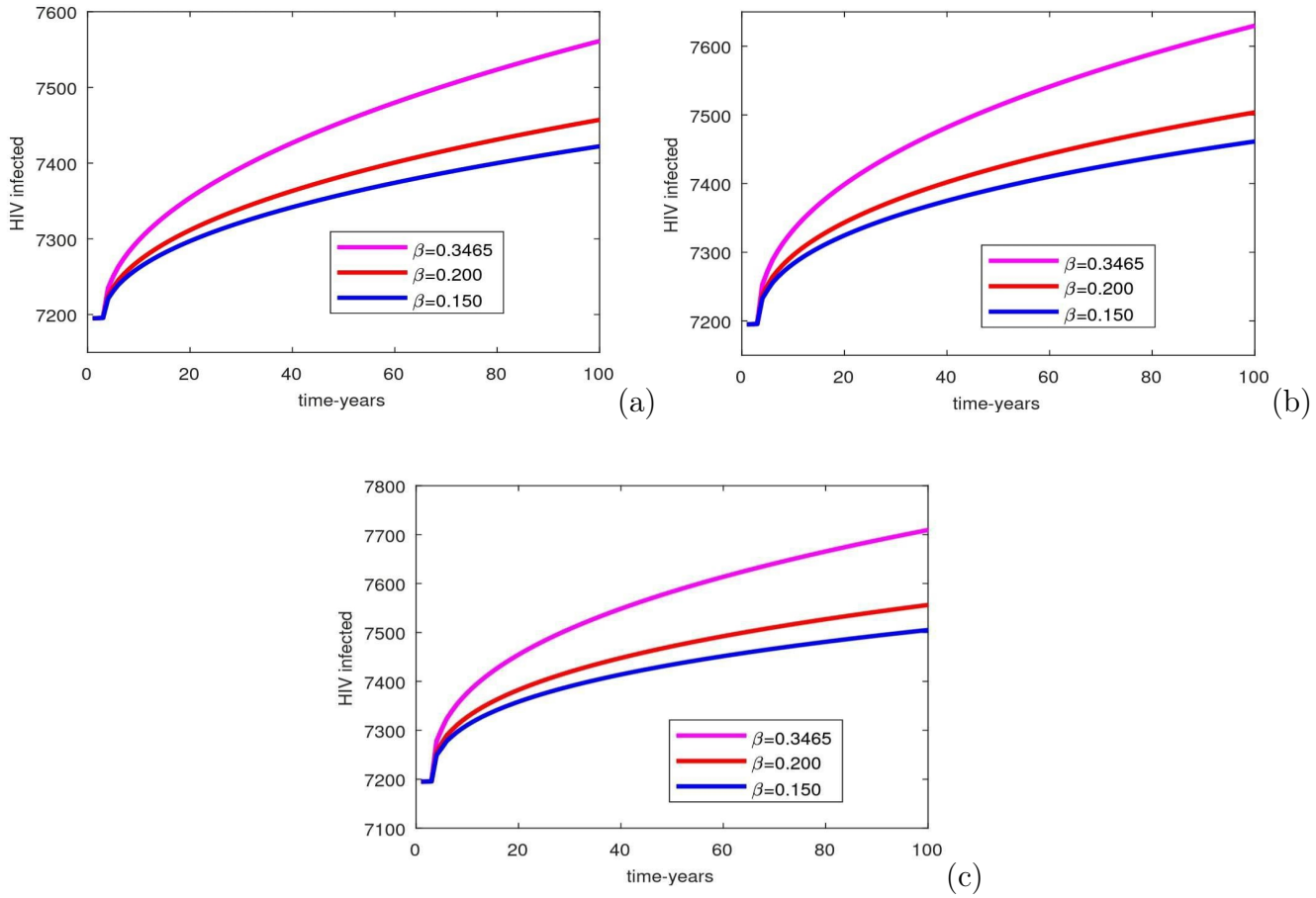

FIGURE 3.3 – The effect of the transmission rate beta on the dynamics of HIV-infected individuals with different values of P such that (a) : P =1,(b) : P =0.96,(c) : P =0.8.

Conclusion

We formulated and analyzed anew mathematical model for HIV/AIDS infected individuals in Indonesia for the years $2006-2018$. We computed the basic reproduction number R_0 for the model for the real data of the years $2006-2018$. Some mathematical results related to the model are shown. The local stability of the model for $R_0 < 1$ are provided. We presented a novel numerical approach for the solution of the HIV/AIDS model which is based on the Newton polynomial approach and presented the results by considering different orders of p. The local asymptotic stability of the fractional model for the case when $R_0 > 1$ and $R_0 < 1$ are shown. Some parameters effect on the HIV, infected with ART treatment and those infected with AIDS are shown. The analysis and the results provided are useful for the public health authorities of Indonesian government to make necessary steps in order to reduce the burden of HIV infection by making their citizen to aware about the HIV and their possible controls/prevention.

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الملخص

يهدف هذا العمل إلى دراسة النموذج الوبائي المتعلق بديناميكيات انتشار مرض فيروس نقص المناعة البشرية من خلال نمذجة المشكلة الوبائية في مسألة رياضية باستخدام نظام المعادلات التفاضلية بإستخدام مشتقات كابتو الجزئية. بادئ ذي بدء ، نناقش الخصائص الأساسية للنموذج ، ونقدم بعض النتائج الخلفية المتعلقة بالنموذج ، المنطقة المجدية بيولوجيًا ، ثم نحسب رقم التكاثر الأساسي R_0 وناقشنا استقرارنظامنا. أخيرًا نحسب الحلول العددية لنموذج الوباء الكسري ونحصل على الحل العددي بيانياً باستخدام كثير حدود نيوتن.

الكلمات المفتاحية: نمذجة – مشتقات كابتو – كثير حدود نيوتن .

Abstract

This work aims to study the epidemiological model related to the dynamics of the spread of HIV disease by modeling the epidemiological problem in a mathematical problem using the system of differential equations using partial Caputo derivatives. First of all we discuss the basic characteristics of the model, present some background results related to the model, the biologically feasible region, then we calculate the basic reproduction number R_0 , and discuss the stability of our system. Finally, we calculate the numerical solutions of the fractal epidemic model and obtain the numerical solution graphically using Newton polynomial .

key words: modeling - Caputo derivatives - Newton polynomial .

Résumé

Ce travail vise à étudier le modèle épidémiologique lié à la dynamique de propagation de la maladie à VIH en modélisant le problème épidémiologique en un problème mathématique utilisant le système d'équations différentielles utilisant les dérivées partielles de Caputo. Tout d'abord, nous discutons des caractéristiques de base du modèle, présentons quelques résultats de base liés au modèle, la région biologiquement réalisable, puis nous calculons le nombre de reproduction de base R_0 et discutons de la stabilité de notre système. Enfin, nous calculons les solutions numériques du modèle épidémique fractal et obtenons la solution numérique graphiquement à l'aide du polynôme de Newton.

Mots clé : modélisation - Dérivées de Caputo - Polynôme de Newton.