ANALYSIS OF A FRACTIONAL ORDER MODEL FOR INFLUENZA A

BY

NWACHUKWU, ANTHONIA UCHENNA

PG/M.Sc/16/83304

A PROJECT SUBMITTED TO THE DEPARTMENT OF MATHEMATICS, UNIVERSITY OF NIGERIA, NSUKKA, IN PARTIAL FUFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF A MASTER OF SCIENCE (M. Sc) DEGREE IN MATHEMATICS

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SUPERVISOR: PROF. M.O. OYESANYA

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APPROVAL PAGE

THIS THESIS BY NWACHUKWU, ANTHONIA UCHENNA HAS BEEN APPROVED FOR THE AWARD OF MASTERS OF SCIENCE (M.SC) DEGREE IN DEPARTMENT OF MATHEMATICS OF THE UNIVERSITY OF NIGERIA, NSUKKA.

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CERTIFICATION

NWACHUKWU, ANTHONIA UCHENNA a postgraduate student of the Department of Mathematics, in the Faculty of Physical Sciences, University of Nigeria, Nsukka with Registration Number: PG/M.Sc./16/83304 specializing in Applied Mathematics has satisfactorily completed the requirements of course work and thesis for the award of degree of masters of Science (M.Sc.) in Mathematics.

Declaration is also made that the work embodied in this thesis is original and has not been submitted in part or in full for any other diploma or degrees of this university or any other University.

DEDICATION

This work is dedicated in its entirety to my parents Chief and Lolo A. J. Nwachukwu and also to my late brother, Master Chijioke Emmanuel Nwachukwu.

ACKNOWLEDGEMENT

I wish to express my profound gratitude to the Almighty God who in its own wisdom made my pursuit a worthwhile and saw me through the periods of my adventure.

At this juncture, I want to acknowledge my supervisor Prof. M.O. Oyesanya who has been very supportive throughout the periods of my study. I have really tapped from his immeasurable wealth of knowledge. God will bless you sir.

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I also appreciate my friends you all contributed to my academic success. I will always cherish you and I wish that we had never had to part Stanley (Course Rep), Chibuike, Oge, Humphery, Nkem, Agu, Austine and others I am indeed very grateful.

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ABSTRACT

In this project work, a fractional order mathematical model that describe the transmission dynamics of Influenza A Virus was given and control mechanism for the spread of the Virus was given.

Qualitative dynamics of the model is determined by the basic reproduction number,R_0 using the next generation matrix. Through the analysis of the model, the stability conditions of the equilibria were derived using Jacobian transformation method. To illustrate our theoretical analysis, some numerical simulations were carried out using Adams-type predictor-corrector method to show the behavior of the solutions of the proposed fractional order system.

The simulation results provide a theoretical basis to control the spread of influenza A and also show that the fractional model is better than the classical model.

CHAPTER ONE
GENERAL INTRODUCTION

1.1 INFLUENZA VIRUS

Influenza, commonly known as "the flu", is an infectious disease caused by an influenza virus. Symptoms can be mild or severe. The most common symptoms include: a high fever, runny nose, sore throat, muscle pains, headache, coughing, and feeling tired. These symptoms typically begin two days after exposure to the virus and most last less than a week. The cough, however, may last for more than two weeks. In children, there may be nausea and vomiting, but these are not common in adults. Nausea and vomiting occur more commonly in the unrelated infection gastroenteritis, which is sometimes inaccurately referred to as "stomach flu" or the "24-hour flu" .Complications of influenza may include viral pneumonia, secondary bacterial pneumonia, sinus infections, and worsening of previous health problems such as asthma or heart failure [1].

Three types of influenza viruses affect people, namely Type A, Type B, and Type C. Usually, the virus is spread through the air from coughs or sneezes. This is believed to occur mostly over relatively short distances. It can also be spread by touching surfaces contaminated by the virus and then touching the mouth or eyes. A person may be infectious to others both before and during the time they are showing symptoms. The infection may be confirmed by testing the throat, sputum, or nose for the virus.. A type of polymerase chain reaction that detects the virus's ribonucleic acid (RNA) is more accurate [2].

1.2 TYPE A INFLUENZA

Influenza A is caused by infection with a virus. It is often called ‘the flu’. The influenza A virus is always changing and evolving. As well as infecting people, influenza A virus can infect animals,

including birds (causing avian flu) and pigs (causing swine flu, H1N1). In some cases, these types of influenza can be passed on to humans through air. Influenza A viruses are divided into subtypes on the basis of two proteins on the surface of the virus: hemagglutinin (HA) and neuraminidase (NA). There are 18 known HA subtypes and 11 known NA subtypes. Many different combinations of HA and NA proteins are possible. For example, an "H7N2 virus" designates an influenza A virus subtype that has an HA 7 protein and an NA 2 protein. Similarly, an "H5N1" virus has an HA 5 protein and an NA 1 protein.

All known subtypes of influenza A viruses can infect birds, except subtypes H17N10 and H18N11, which have only been found in bats. Only two influenza A virus subtypes (i.e., H1N1, and H3N2) are currently in general circulation among people. Some subtypes are found in other infected animal species. For example, H7N7 and H3N8 virus infections can cause illness in horses, and H3N8 virus infection cause illness in horses and dogs [2].

1.2.1 LINEAGES OF INFLUENZA A VIRUSES

Avian influenza (AI) viruses – influenza viruses which infect birds –have evolved into distinct genetic lineages in different geographic locations. These different lineages can be distinguished by studying the genetic make-up of these viruses [3]. For example, AI viruses circulating in birds in Asia, called Asian lineage AI viruses, can be recognized as genetically different from AI viruses that circulate among birds in North America (called North American lineage AI viruses). These broad lineage classifications can be further narrowed by genetic comparisons that allow researchers to group the most closely related viruses together. Thus, the North American lineage of H7N9 viruses could be further broken down into the North American 'wild bird' lineage versus

the North American ‘poultry’ lineage. The host, time period and geographical location are often used in the lineage name to help further delineate one lineage from another.

1.2.2 HIGHLY PATHOGENIC AND LOW PATHOGENIC AVIAN INFLUENZA A VIRUSES

Avian influenza A viruses are designated as highly pathogenic avian influenza (HPAI) or low pathogenicity avian influenza (LPAI) based on molecular characteristics of the virus and the ability of the virus to cause disease and mortality in chickens in a laboratory setting. HPAI and LPAI designations do not refer to the severity of illness in cases of human infection with these viruses; both LPAI and HPAI viruses have caused severe illness in humans. Poultry infected with LPAI viruses may show no signs of disease or only exhibit mild illness (such as ruffled feathers and a drop in egg production) which may not be detected. Infection of poultry with HPAI viruses can cause severe disease with high mortality. Both HPAI and LPAI viruses can spread rapidly through poultry flocks. HPAI virus infection can cause disease that affects multiple internal organs with mortality up to 90% to100% in chickens, often within 48 hours. However, ducks can be infected without any signs of illness. There are genetic and antigenic differences between the influenza A virus subtypes that typically infect only birds and those that can infect birds and people.

Avian influenza viruses rarely infect people because it is not common among humans. The most frequently identified subtypes of avian influenza that have caused human infections are H5, H7 and H9 viruses. Other viruses, such as H10N8, H10N7, and H6N8, have been detected in people also, but to a lesser extent.

1.2.3 INFLUENZA A H5

There are nine known subtypes of H5 viruses (H5N1, H5N2, H5N3, H5N4, H5N5, H5N6, H5N7, H5N8, and H5N9). Most H5 viruses identified worldwide in wild birds and poultry are LPAI, but occasionally HPAI viruses have been detected. Sporadic H5 virus infection of humans has occurred, such as with Asian lineage HPAI H5N1 viruses currently circulating among poultry in Asia and the Middle East. Human infection of H5N1 virus infections have been reported in 16 countries, often resulting in severe pneumonia and greater than 50% mortality.

1.2.4 INFLUENZA A H7

There are nine known subtypes of H7 viruses (H7N1, H7N2, H7N3, H7N4, H7N5, H7N6, H7N7, H7N8, and H7N9). Most H7 viruses identified worldwide in wild birds and poultry are LPAI viruses. H7 virus infection in humans is uncommon. The most frequently identified H7 viruses associated with human infection are Asian lineage avian influenza A (H7N9) viruses, which were first detected in China in 2013. While human infections are rare, these have commonly resulted in severe respiratory illness and death. In addition to Asian lineage H7N9 viruses, H7N2, H7N3, H7N7 virus infections have been reported. These viruses have primarily caused mild to moderate illness in people, with symptoms that include conjunctivitis and/or upper respiratory tract symptoms.

1.2.5 INFLUENZA A H9

There are nine known subtypes of H9 viruses (H9N1, H9N2, H9N3, H9N4, H9N5, H9N6, H9N7, H9N8, and H9N9); all H9 viruses identified worldwide in wild birds and poultry are LPAI viruses. H9N2 virus has been detected in bird populations in Asia, Europe, the Middle East and Africa.

Rare, sporadic H9N2 virus infections in people have been reported to generally cause mild upper respiratory tract illness; one infection has results in death.

1.3 SYMPTOMS OF INFLUENZA A

If you have influenza, you will have some or all of these symptoms:

- fever and chills
- headache and muscle aches
- feeling tired and weak
- sneezing, and stuffy or runny nose
- sore throat and cough

Children may also have abdominal pain, nausea and vomiting. It's a bit like a very bad cold, but a cold doesn't usually give you aches and pains.

1.4 TREATMENT OF INFLUENZA A

If you have influenza A, you are likely to get better within a week or so by:

- resting in bed
- taking mild pain killers to relieve your pain
- drinking plenty of liquids
- eating light foods like fruits, when you're hungry

In some people, the flu can be severe and lead to serious complications like pneumonia. This is mostly likely to affect the very young, the elderly, pregnant women, Indigenous people, and people with chronic health problems.

1.5 SIGNIFICANCE OF THE STUDY

This study of Influenza A is significant because of its outbreak. Therefore, it is important we mitigate against it so that we would avoid it invading the human population.

In this paper, we consider the fractional order SIRC model associated with the evolution of influenza A disease in human population. Qualitative dynamics of the model is determined by the basic reproduction number, R_0. We give a detailed analysis for the asymptotic stability of disease-free and positive fixed points. Numerical simulations are presented to verify the obtained results

1.6 AIMS AND OBJECTIVE OF THE STUDY

Our aim of the study is to construct a fractional order model that will provide a lasting solution to humanity so that influenza A virus does not invade the human population. Therefore, our specific objectives are as follows:

1. Construct a fractional order mathematical model that can describe the transmission dynamics of Influenza A virus.
2. To see the factors that influences the spread of Influenza A virus and hence, how to control them.
3. Carry out a detailed analysis of the model fractional differential equation.
4. Interpret the result of the analysis obtained from the modeled equation.

1.7 SCOPE OF THE STUDY

In the study of Influenza, A virus, we try to look at the dynamics of influenza A virus transmission dynamics. We also try to construct a fractional model using a SIRC approach, where S is the susceptible compartment, I is the infected compartment, R is the removed/recovered compartment and C is the cross-immune compartment. The stability analysis of the model were

studied. The locally asymptotic stability (LAS) was undertaken and sensitivity analysis of the models were also considered after which numerical simulation of the models was presented.

1.8 DEFINITIONS AND THEOREMS

1.8.1 FRACTIONAL CALCULUS

The idea of generalizing the notion of differentiation $\frac{d^p f(x)}{dx^p}$ to non-integer orders of p appeared at the birth of the differential calculus itself. The first attempt to discuss such an idea recorded in history was contained in the correspondence of Leibniz [4, 5]. In one of his letters to Leibniz concerning the theorem on the differentiation of a product of functions, Bernoulli asked about the meaning of theorem in the case of non-integer order of differentiation. Leibniz in his letters to L'Hopital (1695) and to Wallis (1697), made some remarks on the possibility of considering differentials and derivatives of order 1/2.

Recently, many mathematicians and applied researchers have tried to model real processes using the fractional calculus [6, 7]. Fractional calculus and fractional-order differential equations date back to near the foundations of calculus, and they have been used in engineering fields for several decades [8, 9, 10]. Many applications of fractional calculus amount to replacing the time derivative in a given evolution equation by a derivative of fractional order [11, 12]. The concept of fractional calculus has tremendous potential to change the way we see the model, and control the nature around us. The major reason of using fractional differential equations are naturally related to systems with memory which exists in most biological and systems [13, 14]. Also, they are closely related to fractals, which are abundant in biological systems [15, 12]. Moreover, fractional order differential equations are, at least, as stable as their integer order counterpart [16, 17]. In other words, the fractional order derivative can capture the history of the variable that is. It

is difficult to be done by means of the integer order derivatives. The physical meaning of the fractional order is considered in [7] to be the index of memory.

We use Caputo derivative as it is attractive when physical models are presented because of clarity in the physical interpretation of the prescribed data [18]. Also, Caputo derivative is useful because the initial conditions for the fractional-order models with the Caputo derivatives can be the same as for the integer-order differential equations [19, 20].

1.8.2 Definition 1.1

The Caputo Fractional derivative of order α of a function $f: \mathbb{R}^+ \to \mathbb{R}$ is given by

$$ {}_0^C D_t^\alpha f(t) = \frac{1}{\Gamma(\alpha - n)} \int_a^t \frac{f^{(n)}(\tau) d\tau}{(t-\tau)^{\alpha+1-n}} \qquad (n-1 < \alpha < n). \qquad (1.1) $$

1.8.3 Definition 1.2

The formula for the Laplace transform of the Caputo derivative is given by

$$ \int_0^\infty e^{-st} \{ {}_0^C D_t^\alpha f(t) \} dt = s^\alpha F(p) - \sum_{k=0}^{n-1} s^{\alpha-k-1} f^{(k)}(0), \qquad (n-1 < \alpha \leq n). \qquad (1.2) $$

1.8.4 Definition 1.3

The fractional integral of order α of a function $f: \mathbb{R}^+ \to \mathbb{R}$ is given by

$$ J^\alpha f(x) = \frac{1}{\Gamma(\alpha)} \int_0^x (x-t)^{\alpha-1} f(t) dt\,, \qquad \alpha > 0, x > 0. \qquad (1.3) $$

1.8.5 Definition 1.4

The fractional integral of the Caputo derivative of order α of a function $f: \mathbb{R}^+ \to \mathbb{R}$ is given by

$$ J^\alpha \{ D^\alpha f(t) \} = f(t) - \sum_{k=0}^{n-1} f^{(k)}(0) \frac{t^k}{k!}\,, \qquad t > 0. \qquad (1.4) $$

1.8.6 Definition 1.5

A two-term parameter function of the Mittag-Leffler type is defined by the series expansion

$$E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}, \qquad (\alpha > 0,). \tag{1.5}$$

1.8.7 Definition 1.6

The Taylor's series expansion of two variables is given as: If f(x, y) and all its partial derivatives up to the nth order are finite and continuous for all points (x, y) where

$$a \le x \le a + h, b \le y \le b + k$$

Then $f(a+h, b+k) = f(a,b) + \left(h\frac{\partial}{\partial x} + k\frac{\partial}{\partial y}\right) f + \frac{1}{2!}\left(h\frac{\partial}{\partial x} + k\frac{\partial}{\partial y}\right)^2 f + \frac{1}{3!}\left(h\frac{\partial}{\partial x} + k\frac{\partial}{\partial y}\right)^3 f +$

$\cdots$ (1.6)

CHAPTER TWO

LITERATURE REVIEW OF INFLUENZA A MODELS

Kermack and McKendrick [21]were the first to introduce an epidemic model with partial immunity and have recently been studied in the context of influenza. In order to simplify the problem certain definite assumptions were made, namely, that all individuals were equally susceptible, and that death resulted, or complete immunity was conferred, as the result of an attack. The infectivity of the individual and his chances of death or recovery were represented by arbitrary functions, and the chance of a new infection occurring was assumed to be proportional to the product of the infected and susceptible members of the population. In spite of the introduction of the arbitrary functions, it was shown that in general a critical density of population existed, such that if the actual density was less than this, no epidemic could occur, but if it exceeded this by n an epidemic would appear on the introduction of a focus of infection, and further that if n was small relative to the population density, the size of the epidemic would be 2n per unit area. It was shown that these conclusions could be readily extended to the case of a metaxenous disease, that is, one in which transmission takes place through an intermediate host.

Casagrandi et al. [22] introduced SIRC model by adding a new compartment *C*, which can be called cross-immune compartment, to the SIR model. This cross-immune compartment (C) describes an intermediate state between the fully susceptible (*S*) and the fully protected one (R). They developed a simple ordinary differential equation model to study the epidemiological consequences of the drift mechanism for influenza A viruses. Improving over the classical SIR approach, they introduced a fourth class (C) for the cross-immune individuals in the population, i.e., those that recovered after being infected by different strains of the same viral subtype in the past years. The SIRC model predicted that the prevalence of a virus is maximum for an

intermediate value of R_0, the basic reproduction number. Via a bifurcation analysis of the model, they discussed the effect of seasonality on the epidemiological regimes. For realistic parameter values, the model exhibited a rich variety of behaviors, including chaos and multistable periodic outbreaks. Comparison with empirical evidence shows that the simulated regimes are qualitatively and quantitatively consistent with reality, both for tropical and temperate countries. They found that the basins of attraction of coexisting cycles can be fractal sets, thus predictability can in some cases become problematic even theoretically.

Jodar et al. [23] developed two nonstandard finite difference schemes to obtain numerical solutions of an influenza A disease model presented by Casagrandi et al [24]. Qualitative dynamics of the model is determined by the basic reproduction number, R_0. Numerical schemes developed here are based on nonstandard finite difference methods. Our aim is to transfer essential properties of the continuous model to the discrete schemes and to obtain unconditional stable schemes. The proposed numerical schemes have two fixed points which are identical to the critical points of the continuous model and it is shown that they have the same stability properties. Numerical simulations with different initial conditions, parameters values and time step sizes are developed for different values of parameter $R0$, convergence to the disease free equilibrium point when $R0 < 1$ and to the endemic equilibrium point when $R0 > 1$ are obtained independent of the time step size. These numerical integration schemes are useful since can reproduce the dynamics of original differential equations.

Lin et al. [25]estimated the drift speed of the travelling wave solutions arising in a model where the virus mutation is described as a diffusion process in a linear, continuous strain space. Stochastic, individual based models aimed to understanding the dynamics of evolving diseases over discrete strain spaces and for finite populations are proposed. The major drawback of the

multi-strain models is that their analysis can become analytically difficult or numerically cumbersome if the number of strains is large enough to mimic the real variety in nature. An alternative path to viewing the emergence of new viral strains is that of introducing into the model a loss of immunity by the host, as originally suggested by Pease [26] for the case of influenza A. Pease based his evolutionary on R. Casagrandi et al. Mathematical Biosciences 200 (2006) 152–169 153 model on some data by Gill and Murphy [27] and Potter et al. [28], who experimentally showed that the probability of recovered individuals being reinfected by new circulating strains linearly increases with the time since last infection. Both the genetic and the antigenic evolution of influenza A viruses occurs indeed linearly through time, as emphasized in a detailed study by and discussed below. These results indicate that the classical single-strain SIRS model cannot be used as is to study influenza.

Gomes and colleagues [29] recently proposed to consider a 'temporary partial immunity' for the individuals in the R compartment to solve the issue. Here we introduce a new compartment C, which can be called cross-immune, to design an intermediate state between the fully susceptible state (S) and the fully protected one (R). Only by extending the SIR model with a new class one can indeed account for the fact that, when the cross-immune hosts are exposed to antigenically similar –but different – strains, their pre-existing immune responses are boosted as in the case of pertussis.

Epidemic models of temporary partial immunity – or variable susceptibility – were first introduced by Kermack and McKendrick [21] and have recently been studied in the context of influenza.

THE PRESENT WORK

The present model captures a fractional order SIRC model that describes the transmission dynamics of Influenza A virus with its stability for both Disease free equilibrium and Endemic free equilibrium and the discussion of the numerical results gotten from the developed model. It also captures why the fractional models are better than classical models.

CHAPTER THREE

METHODOLOGY

3.1 MODEL FORMULATION

1. This model assumes that the natural death rates μ in the classes is equal to the birthrate, so that population size N is constant. In this model, the population N(t) is divided into four subpopulations: The Susceptible, Infectious, Recovered, and the cross-immune class with sizes denoted by S(t), I(t), R(t), and C(t) respectively.
2. All recruitment is done by birth into the susceptible population and occurs at a constant birth rate.
3. It is assumed that as the population size (the density of individuals) increases, so does the contact rate (density dependent transmission).
4. The infective rate of an infected individual is proportional to the number of Susceptible, the coefficient of the proportion is a constant β, so that the total number of new infected at time t is βS(t)I(t).
5. An individual who is infected through direct contact with an infectious individual gets infected and enters into the infectious class at the rate β.
6. It is assumed that the cross-immune class interacts with the infected class at the rate βCI.
7. Recovered class, R(t), consists of individual who have recovered from the illness and also some a fraction of those who are cross immune (1- δ).
8. δ is the fraction of the exposed cross-immune individuals who are recruited in a unit time into the infective population
9. θ is the rate at which the cross-immune population becomes susceptible.
10. People can die naturally

3.2 DESCRIPTION OF VARIABLES AND PARAMETERS OF THE MODEL

VARIABLES:

S(t) = Population representing susceptible individuals at time t

I(t) = Population representing Infectious individuals at time t

R(t) = Population representing recovered individuals at time t

C(t) = Population representing cross-immune individuals at time t

N(t) = Total population size at time t

PARAMETERS:

μ = Per capita birth rate

δ = Fraction of the exposed cross-immune individuals who are recruited in a unit time into the infective population

(1- δ) = Fraction of the exposed cross-immune individuals who are recruited in a unit time into the recovered population

β = Transmission rate of the disease when a contact occurs

γ = The rate at which the infectious individuals progressed to be recovered

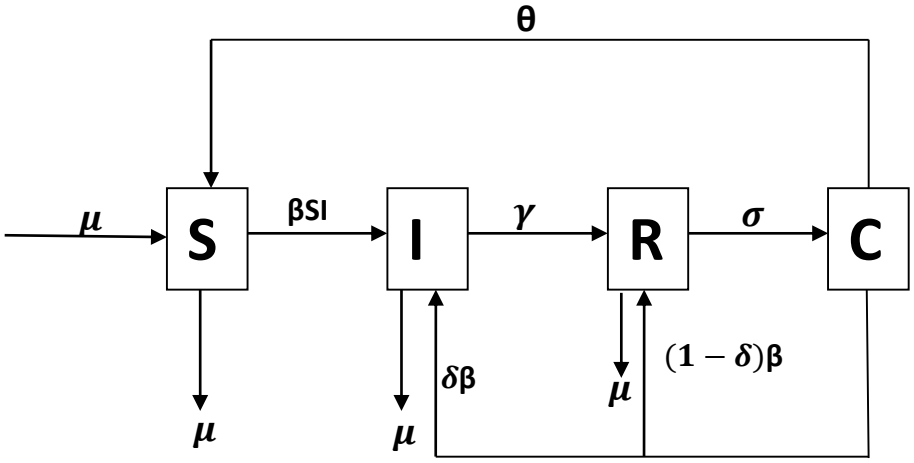
$\theta =$ is the rate at which the cross-immune population becomes susceptible.

σ = The rate at which the recovered individual gets cross-immune.

μ = Natural death rate

Now, putting these assumptions together, we have a model flow diagram and thus model equations for the transmission of Influenza A virus

3.3 MODEL FLOW DIAGRAM FOR TRANSMISSION DYANMICS FOR INFLUENZA A VIRUS



3.4 MATHEMATICAL MODEL FOR TRANSMISSION DYNAMICS OF INFLUENZA A VIRUS (CLASSICAL CASE)

$$\frac{dS}{dt} = \mu(1 - S) - \beta SI + \theta C$$

$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I + \delta\beta CI \qquad (3.1)$$

$$\frac{dR}{dt} = (1 - \delta)\beta CI - \mu R + \gamma I - \sigma R$$

$$\frac{dC}{dt} = \sigma R - \theta C - \beta CI - \mu C$$

Now if replace the integer derivatives in system (3.1) by Caputo derivatives of orderα, then the modified system can be written as:

3.5 MATHEMATICAL MODEL FOR TRANSMISSION DYNAMICS OF INFLUENZA A VIRUS (FRACTIONAL CASE)

$$D^{\alpha}S(t) = \mu(1 - S) - \beta SI + \theta C$$

$$D^{\alpha}I(t) = \beta SI - \gamma I - \mu I + \delta\beta\mathrm{CI} \tag{3.2}$$

$$D^{\alpha}R(t) = (1-\delta)\beta\mathrm{CI} - \mu R + \gamma I - \sigma R$$

$$D^{\alpha}C(t) = \sigma R - \theta C - \beta CI - \mu C$$

Following the method by Diethelm [30], the fractionalized system (3.2) becomes:

$$D^{\alpha}S(t) = \mu^{\alpha}(1-S) - \beta^{\alpha}SI + \theta^{\alpha}C$$

$$D^{\alpha}I(t) = \beta^{\alpha}SI - \gamma^{\alpha}I - \mu^{\alpha}I + \delta^{\alpha}\beta^{\alpha}\mathrm{CI} \tag{3.3}$$

$$D^{\alpha}R(t) = (1-\delta^{\alpha})\beta^{\alpha}\mathrm{CI} - \mu^{\alpha}R + \gamma^{\alpha}I - \sigma^{\alpha}R$$

$$D^{\alpha}C(t) = \sigma^{\alpha}R - \theta^{\alpha}C - \beta^{\alpha}CI - \mu^{\alpha}C$$

CHAPTER FOUR

4.1 ANALYSIS OF THE MODEL

4.1.1 INVARIANT REGION AND POSITIVITY OF THE MODEL SOLUTIONS

Let N = S + I + R + A; then $D^{\alpha}N(t) = \mu^{\alpha} - \mu^{\alpha}N,$ (4.1)

Let $\Omega = \{(S, I, R, C); S, I, R, C \leq 0, S + I + R + C \leq 1\},$ (4.2)

Lemma 3.1. The closed set $\Omega = \{(S, I, R, C) \in R_{+}^{4}: S + I + R + C = 1\}$ is positively invariant with respect to model (3.3).

Proof. The fractional derivative of the total population, obtained by adding all the equations of model (3.3), is given by

$$D^{\alpha}N(t) = \mu^{\alpha} - \mu^{\alpha}N(t) \quad (4.3)$$

Taking the Laplace transform of (3.3), we have

$$S^{\alpha}N(s) - S^{\alpha-1}N(0) = \frac{\mu^{\alpha}}{S} - \mu^{\alpha}N(s) \quad (4.4)$$

$$\Rightarrow N(s) = \frac{\mu^{\alpha}}{S(S^{\alpha}+\mu^{\alpha})} + \frac{S^{\alpha-1}}{S^{\alpha}+\mu^{\alpha}}N(0) \quad (4.5)$$

and taking the inverse Laplace, we obtain

$N(t) = N(0)E_{\alpha,1}(-\mu^{\alpha}) + \mu^{\alpha}t^{\alpha}E_{\alpha,\alpha+1}(-\mu^{\alpha}t^{\alpha})$, where $E_{\alpha,\beta}$ is the Mittag-Leffler function. Therefore, all solutions of the model with initial conditions in Ω remain in Ω for all $t \geq 0$. Thus, region Ω is positively invariant with respect to the model (3.2).

4.1.2 THE BASIC REPRODUCTION NUMBER (R_0)

The basic reproduction number denoted by R_0 is a parameter used to determine how long a disease will prevail in a particular population and this is determined by the value of R_0. If the basic reproduction number $R_0 < 1$, it implies that an infected individual produces an average less than one infected person, and by that calculation it means that with time the disease will die out from the population. But if $R_0 > 1$, it means that an infected person produces more than one infected person in the population. So, the disease will surely remain in the population. So for the disease to die out of the population with time, the basic reproduction number must be less than one.

Diekmann et al [31] defined the basic reproduction number R_0, as the number of secondary infections that one infectious individual will create over the duration of the infectious period, provided that everyone is susceptible.

Now considering the infected classes, we have

$$D^{\alpha}I(t) = \beta^{\alpha}SI - \gamma^{\alpha}I - \mu^{\alpha}I + \delta^{\alpha}\beta^{\alpha}\mathrm{CI}$$

$$= \beta^{\alpha}SI - (\gamma^{\alpha} + \mu^{\alpha} + \delta^{\alpha}\beta^{\alpha}\mathrm{C})I$$

$$= \left\{ \left[\frac{\beta^{\alpha}S}{(\gamma^{\alpha}+\mu^{\alpha}+\delta^{\alpha}\beta^{\alpha}\mathrm{C})} - 1 \right] I(\gamma^{\alpha} + \mu^{\alpha} + \delta^{\alpha}\beta^{\alpha}\mathrm{C}) \right\} \Big|_{DFE,(1,0,0,0)}$$

$$= \left[\frac{\beta^{\alpha}}{(\gamma^{\alpha}+\mu^{\alpha})} - 1 \right] I(\gamma^{\alpha} + \mu^{\alpha})$$

$$\Rightarrow R_0 = \frac{\beta^{\alpha}}{(\gamma^{\alpha}+\mu^{\alpha})}. \qquad (4.7)$$

4.1.4 EQUILIBRIUM POINTS AND THEIR ASYMPTOTIC STABILITY

4.1.4.1 LOCAL STABILITY OF THE DFE OF THE MODEL*:*

To determine model equilibria of the fractional order model (3.3), let

$$\begin{cases} D^{\alpha}S(t) = 0 \\ D^{\alpha}I(t) = 0 \\ D^{\alpha}R(t) = 0 \\ D^{\alpha}C(t) = 0 \end{cases}$$

Then system (3.3) becomes

$$D^{\alpha}S(t) = \mu^{\alpha}(1 - S) - \beta^{\alpha}SI + \theta^{\alpha}C$$

$$D^{\alpha}I(t) = \beta^{\alpha}SI - \gamma^{\alpha}I - \mu^{\alpha}I + \delta^{\alpha}\beta^{\alpha}\mathrm{CI} \qquad (4.1)$$

$$D^{\alpha}R(t) = (1 - \delta^{\alpha})\beta^{\alpha}\mathrm{CI} - \mu^{\alpha}R + \gamma^{\alpha}I - \sigma^{\alpha}R$$

$$D^{\alpha}C(t) = \sigma^{\alpha}R - \theta^{\alpha}C - \beta^{\alpha}CI - \mu^{\alpha}C$$

On solving the right hand side of equation (4.1) gives the disease free equilibrium state of the model.

Thus, the DFE is given by $E^0(S^0, I^0, R^0, C^0) = (1,0,0,0)$

4.2 LOCAL STABILITY OF THE DISEASE-FREE EQUILIBRIUM (DFE) OF THE MODEL:

It is important to remark that, the disease free equilibrium point is where the infectives in the model equals zero (I=R=C=0).

The Jacobian matrix J (E^0) for the fractional order model (3.3) computed at E^0 is given as:

$$J(E^0) = \begin{pmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial R} & \frac{\partial S}{\partial C} \\ \frac{\partial V}{\partial S} & \frac{\partial V}{\partial I} & \frac{\partial V}{\partial R} & \frac{\partial V}{\partial C} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial R} & \frac{\partial I}{\partial C} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial R} & \frac{\partial R}{\partial C} \end{pmatrix}$$

Which then gives

$$J(E^0) = \begin{pmatrix} -\mu^\alpha & -\beta^\alpha & 0 & \theta^\alpha \\ 0 & \beta^\alpha - (\gamma^\alpha + \mu^\alpha) & 0 & 0 \\ 0 & \gamma^\alpha & -(\sigma^\alpha + \mu^\alpha) & 0 \\ 0 & 0 & \sigma^\alpha & -(\theta^\alpha + \mu^\alpha) \end{pmatrix}$$

Hence, we have that

$$|J(E^0) - \lambda I| = \begin{vmatrix} -\mu^\alpha - \lambda & -\beta^\alpha & 0 & \theta^\alpha \\ 0 & \beta^\alpha - (\gamma^\alpha + \mu^\alpha) - \lambda & 0 & 0 \\ 0 & \gamma & -(\sigma^\alpha + \mu^\alpha) - \lambda & 0 \\ 0 & 0 & \sigma^\alpha & -(\theta^\alpha + \mu^\alpha) - \lambda \end{vmatrix} = 0$$

So we have that

$$[(-\mu^\alpha - \lambda_1)(-(\theta^\alpha + \mu^\alpha) - \lambda_2)(-(\sigma^\alpha + \mu^\alpha) - \lambda_3)(\beta^\alpha - (\gamma^\alpha + \mu^\alpha) - \lambda_4)] = 0,$$

$$\Rightarrow \lambda_1 = -\mu^\alpha, \lambda_2 = -(\theta^\alpha + \mu^\alpha), \lambda_3 = -(\sigma^\alpha + \mu^\alpha), \lambda_4 = \beta^\alpha - (\gamma^\alpha + \mu^\alpha) = (R_0 - 1)(\gamma^\alpha + \mu^\alpha)$$

This implies that $E^0 is\ locally\ asymptotically\ stable\ if\ R_0 < 1\ otherwise, is\ is\ unstable.$

4.3 ENDEMIC EQUILIBRIUM POINTS AND THEIR STABILITIES

At the point in time where the compartments of the population coexist is called the endemic equilibrium point. When there is infection in the system, then $S(t) \neq 0, V(t) \neq 0, I(t) \neq 0, R(t) \neq 0$ the model system of equation (3.3) has a non-trivial point and the endemic equilibrium point (EEP) is given as

$$E^* = (S^*, V^*, I^*, R^*)$$

The positive equilibrium $E^* = (S^*, I^*, R^*, C^*)$ satisfies

$$S^* = \frac{\mu + \gamma}{\beta} - \delta\left(\frac{\sigma\gamma I^*}{(\mu + \delta\sigma)\beta I^* + (\mu + \theta)(\mu + \sigma)}\right)$$

$$R^* = \frac{\gamma I^*(\beta I^* + \mu + \theta)}{(\mu + \delta\sigma)\beta I^* + (\mu + \theta)(\mu + \sigma)}$$

$$C^* = \frac{\sigma\gamma I^*}{(\mu + \delta\sigma)\beta I^* + (\mu + \theta)(\mu + \sigma)}$$

And I^* is the positive root of $g(I) = A_1 I^2 + A_2 I + A_3(1 - R_0), where\ R_0 = \frac{\beta}{(\gamma+\mu)}$ and

$$A_1 = \beta\mu(\gamma + \mu + \sigma\delta)$$

$$A_2 = \beta\mu(-\beta\mu + \theta(\sigma + \gamma + \mu) + (\gamma + \theta)(\sigma + 2\mu) + \sigma(-\beta + \mu)\delta)$$

$$A_3 = \mu(\mu + \theta)(\mu + \sigma)(\mu + \gamma).$$

4.4 STABILITY ANALYSIS OF THE ENDEMIC EQUILIBRIUM POINT

We now discuss the asymptotic stability of the endemic (positive) equilibrium of the system given (3.3). The Jacobian matrix J(E*) is given as:

$$J(E^*) = \begin{pmatrix} -\beta I^* - \mu & -\beta S^* & 0 & \theta \\ \beta I^* & \beta S^* + \delta\beta C - (\mu + \gamma) & 0 & \delta\beta I^* \\ 0 & (1 - \delta)\beta C^* + \gamma & -(\mu + \sigma) & (1 - \delta)\beta I^* \\ 0 & -\beta C^* & \sigma & -\beta I^* - (\mu + \theta) \end{pmatrix}$$

The characteristic equation of J(E*) is given as

$$(\lambda + \mu)(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0$$

where

$$a_1 = \theta + \sigma + 2\mu + 2\beta I^*,$$

$$a_2 = (\theta + \mu)(\sigma + \mu) + \beta I^*(\theta + \sigma + \gamma + 3\mu + \sigma\delta + \beta I^*),$$

$$a_3 = \frac{\beta I^*(\omega(\sigma + \mu)(\gamma + \mu) + \theta(\sigma + \gamma + \mu) - \sigma\gamma\delta) + \beta(\gamma + \mu + \sigma\delta)I^*(2\omega + \beta(\mu + \sigma\delta)I^*))}{\omega + \beta(\mu + \sigma\delta)I^*},$$

where $\omega = (\mu + \theta)(\theta + \mu)$.

Proposition 1. [6] The endemic equilibrium point E^* is asymptotically stable if all the eigenvalues λ of $J(E^*)$ satisfy $|\arg(\lambda)| > \frac{\alpha\pi}{2}$.

Let D (ϕ) denote the discriminant of a polynomial f. if $\phi(x) = x^3 + a_1x^2 + a_2x + a_3$ then, denote

$$D(\phi) = -\begin{vmatrix} 1 & a_1 & a_2 & a_3 & 0 \\ 0 & 1 & a_1 & a_2 & a_3 \\ 3 & 2a_1 & a_2 & 0 & 0 \\ 0 & 3 & a_1 & a_2 & 0 \\ 0 & 0 & 3 & 2a_1 & a_2 \end{vmatrix} = 18a_1a_2a_3 + (a_1a_2)^2 - 4a_3{a_1}^2 - 4a_2^3 - 27a_3^2.$$

Using the results of [32], we have

Proposition 2.

I. If the discriminant of $\phi(x)$, $D(\phi)$ is positive, then the endemic equilibrium point E^* is asymptotically stable iff Routh-Hurwitz conditions are satisfied, i.e,

$$a_1 > 0, a_3 > 0, a_1a_2 > a_3 \; if \; D(\phi) > 0.$$

II. If $D(\phi) < 0, a_1 \geq 0, a_2 \geq 0, a_3 > 0, 0.5 < \alpha < 2/3$, then then the endemic equilibrium point E^* is asymptotically stable.

III. If $D(\phi) < 0, a_1 > 0, a_2 > 0, a_1 a_2 = a_3, \alpha \epsilon (0.5,1)$, then then the endemic equilibrium point E^* is asymptotically stable.

IV. If $D(\phi) < 0, a_1 < 0, a_2 < 0, \alpha < 2/3$, then then the endemic equilibrium point E^* is unstable.

CHAPTER FIVE

DISCUSSION, SUMMARY AND CONCLUSION

5.1 NUMERICAL SIMULATIONS AND RESULTS

5.1.1 A BRIEF DESCRIPTION OF THE PREDICTOR-CORRECTOR APPROACH FOR THE NUMERICAL SOLUTION OF FRACTIONAL DIFFERENTIAL EQUATIONS

In this section, we described briefly the derivation of the fundamental algorithm that was developed for the solution of initial value problems with Caputo derivatives. The algorithm is a generalization of the classical Adams–Bashforth–Moulton integrator that is well known for the numerical solution of first-order problems [9, 10]

Our approach is based on the analytical property that every fractional initial value problem is equivalent to the Volterra integral equation

$$y(x) = \sum_{k=0}^{[\alpha]-1} {y_0}^{(k)} \frac{x^k}{k!} + \frac{1}{\Gamma(\alpha)} \int_0^x (x-t)^{\alpha-1} f\big(t, y(t)\big) dt \qquad (5.1)$$

in the sense that a continuous function is a solution of the initial value problem if and only if it is a solution of (5.1). For a brief derivation of this equivalence we refer to [11]. Note that the sum outside the integral on the right-hand side is completely determined by the initial values and hence is known.

In order to motivate the construction of our numerical method, we shall first briefly recall the idea behind the classical one-step Adams–Bashforth–Moulton algorithm for first-order equations. So, for a start, we focus our attention on the well-known initial-value problem for the first-order differential equation.

$$Dy(x) = f\big(x, y(x)\big), \qquad (5.2a)$$

$$y(0) = y_0. \qquad (5.2b)$$

We assume the function *f* to be such that a unique solution exists on some interval [0, *T*], say. Following [13], we suggest the use of the predictor-corrector technique of Adams where, for the sake of simplicity, we assume that we are working on a uniform grid $\{t_n = nh: n = 0,1, \dots, N\}$ with some integer *N* and *h* := *T/N*. The basic idea is, assuming that we have already calculated approximations$y_h(t_j) \approx y(t_j)(j = 1,2, \dots, n)$, that we try to obtain the approximation $y_h(t_{n+1})$ by means of equation

$$y(t_{n+1}) = y(t_n) + \int_{t_n}^{t_{n+1}} f\big(z, y(z)\big)dz. \qquad (5.3)$$

This equation follows upon integration of (5.2a) on the interval$[t_n, t_{n+1}]$. Of course, we know neither of the expressions on the right-hand side of Equation (5.3) exactly, but we do have an approximation for $y(t_n)$, namely $y_h(t_n)$, that we can use instead. The integral is then replaced by the two-point trapezoidal quadrature formula

$$\int_a^b g(z)dz \approx \frac{b-a}{2}\big(g(a) + g(b)\big), \qquad (5.4)$$

Thus giving an equation for the unknown approximation$y_h(t_{n+1})$, it being

$$y_h(t_{n+1}) = y_h(t_n) + \frac{h}{2}\big[f\big(t_n, y(t_n)\big) + f\big(t_{n+1}, y(t_{n+1})\big)\big], \qquad (5.5)$$

where again we have to replace $y(t_n)$ and $y(t_{n+1})$ by their approximations $y_h(t_n)$ and$y_h(t_{n+1})$, respectively. This yields the equation for the implicit one-step Adams–Moulton method, which is

$$y_h(t_{n+1}) = y_h(t_n) + \frac{h}{2}\big[f\big(t_n, y_h(t_n)\big) + f\big(t_{n+1}, y_h(t_{n+1})\big)\big]. \qquad (5.6)$$

The problem with this equation is that the unknown quantity $y_h(t_{n+1})$ appears on both sides, and due to the nonlinear nature of the function *f*, we cannot solve for $y_h(t_{n+1})$ directly in general.

Therefore, we may use Equation (5.6) in an iterative process, inserting a preliminary approximation for $y_h(t_{n+1})$ in the right-hand side in order to determine a better approximation that we can then use.

The required preliminary approximation$y_h^p(t_{n+1})$, the so-called predictor, is obtained in a very similar way, only replacing the trapezoidal quadrature formula by the rectangle rule

$$\int_a^b g(z)dz \approx (b-a)g(a), \qquad (5.7)$$

giving the explicit method

$$y_h^p(t_{n+1}) = y_h(t_n) + hf\big(t_n, y_h(t_n)\big), \qquad (5.8)$$

known as the forward Euler or one-step Adams–Bashforth method. It is well known [33] that the process defined by Equation (5.8) and

$$y_h(t_{n+1}) = y_h(t_n) + \frac{h}{2}\Big[f\big(t_n, y_h(t_n)\big) + f\left(t_{n+1}, y_h^p(t_{n+1})\right)\Big], \qquad (5.9)$$

known as the one-step Adams–Bashforth–Moulton technique, is convergent of order 2. Moreover, this method behaves satisfactorily from the point of view of its numerical stability [33]. It is said to be of the PECE (Predict, Evaluate, Correct, Evaluate) type because, in a concrete implementation, we would start by calculating the predictor in Equation (5.8), then evaluate $f\left(t_{n+1}, y_h^p(t_{n+1})\right)$, use this to calculate the corrector in Equation (5.9), and finally evaluate $f\big(t_{n+1}, y_h(t_{n+1})\big)$. This result is stored for future use in the next integration step. Having introduced this concept, we now try to carry over the essential ideas to the fractional order problem with some unavoidable modifications. The key is to derive an equation similar to (5.3). Fortunately, such an equation is available, namely Equation (5.1). This equation looks somewhat different from Equation (5.3), because the range of integration now starts at 0 instead of t_n. This

is a consequence of the non-local structure of the fractional-order differential operators. This however does not cause major problems in our attempts to generalize the Adams method. We simply use the product trapezoidal quadrature formula to replace the integral, where nodes $t_j (j = 0,1, \dots, n+1)$, are taken with respect to the weight function$(t_{n+1} - .)^{\alpha-1}$. In other words, we apply the approximation

$$\int_0^{t^{n+1}} (t_{n+1} - z)^{\alpha-1} g(z) dz \approx \int_0^{t^{n+1}} (t_{n+1} - z)^{\alpha-1} \tilde{g}_{n+1}(z) dz, \qquad (5.10)$$

where $\tilde{g}_{n+1}$ is the piecewise linear interpolant for g with nodes and knots chosen at the $t_j j = 0,1,2, \dots, n+1$.. Using standard techniques from quadrature theory (cf. [29]), we find that we can write the integral on the right-hand side of (11) as

$$\int_0^{t^{n+1}} (t_{n+1} - z)^{\alpha-1} g(z) dz = \frac{h^\alpha}{\alpha(\alpha+1)} \sum_{j=0}^{n+1} a_{j,n+1} g(t_j), \qquad (5.11)$$

where

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

This then gives us our corrector formula (i.e., the fractional variant of the one-step Adams–Moulton method), which is

$$y_h(t_n + 1) = \sum_{k=0}^{[\alpha]-1} \frac{t_{n+1}^k}{k!} y_0^{(k)} + \frac{h^\alpha}{\Gamma(\alpha+2)} f\left(t_{n+1}, y_h^p(t_{n+1})\right) + \frac{h^\alpha}{\Gamma(\alpha+2)} \sum_{j=0}^{n} a_{j,n+1} f\left(t_j, y_h(t_j)\right), \qquad (5.12)$$

where we have used the functional equation of the Gamma function twice (which implies the identity $\Gamma(\alpha)\alpha(\alpha+1)=\Gamma(\alpha+2)$) and the fact that $a_{n+1,n+1}=1$.

The remaining problem is the determination of the predictor formula that we require to calculate the value $y_h^p(t_{n+1})$. The idea we use to generalize the one-step Adams–Bashforth method is the same as the one described above for the Adams–Moulton technique: we replace the integral on the right-hand side of Equation (5.1) by the product rectangle rule

$$\int_0^{t^{n+1}} (t_{n+1}-z)^{\alpha-1} g(z)dz \approx \sum_{j=0}^{n} b_{j,n+1} g(t_j), \qquad (5.13)$$

where now

$$b_{j,n+1}=\frac{h^\alpha}{\alpha}((n+1-j)^\alpha-(n-j)^\alpha) \qquad (5.14)$$

Thus, the predicted value $y_h^p(t_{n+1})$ is determined by the fractional Adams– Bashforth method

$$y_h^p(t_n+1)=\sum_{k=0}^{[\alpha]-1}\frac{t_{n+1}^k}{k!}y_0^{(k)}+\frac{1}{\Gamma(\alpha)}\sum_{j=0}^{n} b_{j,n+1} f\left(t_j, y_h(t_j)\right). \qquad (5.15)$$

Our basic algorithm, the fractional Adams–Bashforth–Moulton method, is fully described now by Equations (5.15) and (5.13) with the weights $a_{j,n+1}$ and $b_{j,n+1}$ being defined according to (5.12) and (5.14), respectively.

We have thus completed the description of our numerical algorithm.

5.1.2 THE PREDICTOR-CORRECTOR MATLAB CODES FDE23.M FOR SOLVING FRACTIONAL DIFFERENTIAL EQUATIONS USING MATLAB

We used MATLAB to get the numerical solution of our model by Adams-type predictor-corrector method. Refer to http://www.mathworks.com/matlabcentral/fileexchange/32918 or to Appendix to get the code used.

We will propose two cases for the model (3.3) with various values of parameters. The table below shows the parameter values used for $R_0 = 0.096 < 1$.

Parameter	Value	Source
μ	0.02	Estimated
β	0.05	Estimated
σ	1	Estimated
γ	0.5	Estimated
δ	0.05	Estimated
θ	0.5	Estimated

The table below shows the parameter values used for $R_0 = 8.0 > 1$.

Parameter	Value	Source
μ	0.02	Estimated
β	2	Estimated
σ	1	Estimated
γ	0.5	Estimated
δ	0.05	Estimated
θ	0.5	Estimated

with initial values of the variables given as $S(0) = 50$, $I(0) = 5$,$R(0) = 3, C = 2$.

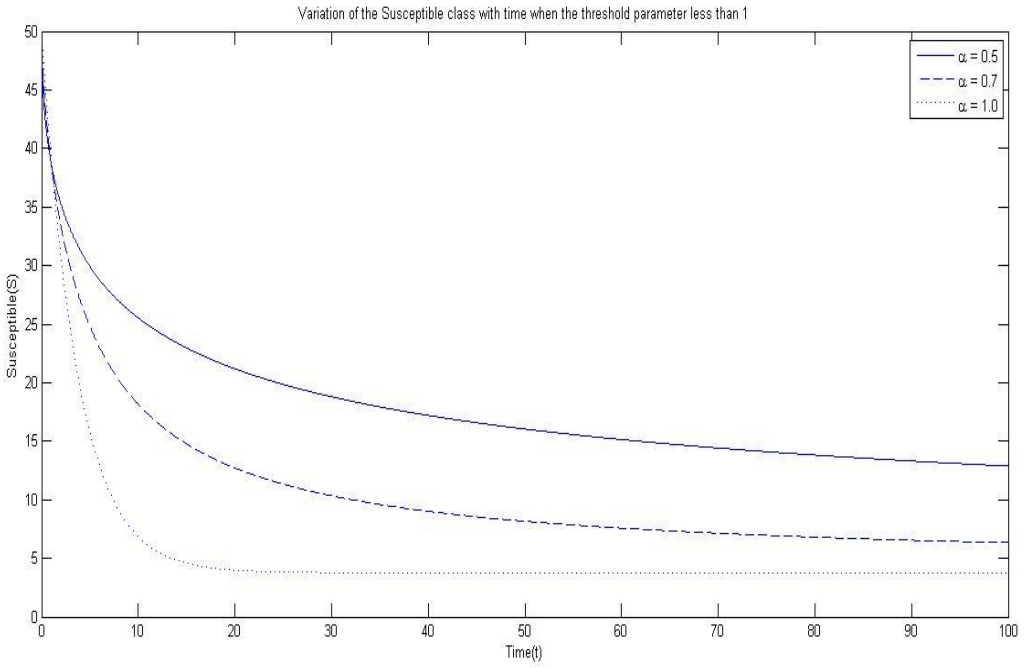


Figure 1: Variation of the Susceptible population with time when $R_0 < 1$.

The above graph shows how the susceptible class vary with time when $R_0<1$. Clearly, it can be seen that the decreasing did not intercept on the horizontal axis hence, tells us that the disease will not invade the population rather will vanish with time. Also, at every time, the curve with $\alpha = 0.5$ gave a higher value than $\alpha = 1.0$ hence the fractional case gives a better result than the classical case.

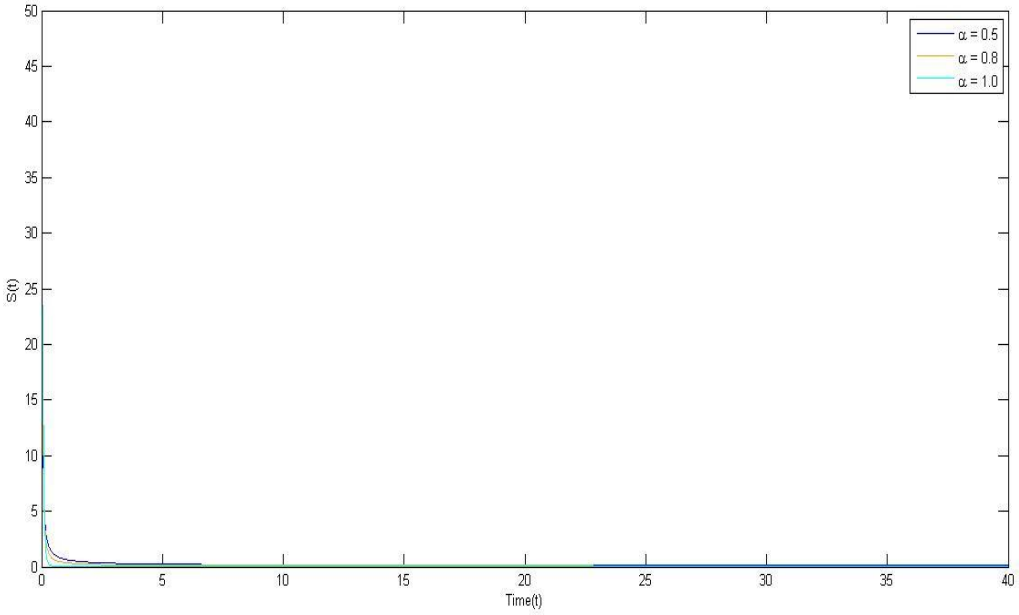


Figure 2: Variation of the Susceptible class with time when $R_0 > 1$.

Here, we considered what happens to the susceptible class alone. At endemic equilibrium ($R_0 > 1$), we see that as we increase the contact rate,β,that is, the rate at which susceptible humans interact with the infected humans and decrease the rate at which infected humans become recovered, it gave a decreasing asymptotic curve as shown above. This implies that the susceptible humans would keep getting infected and then recovered as an infected person would infect more than an average susceptible person in the population, hence all the susceptible humans would become infected with time as seen above.

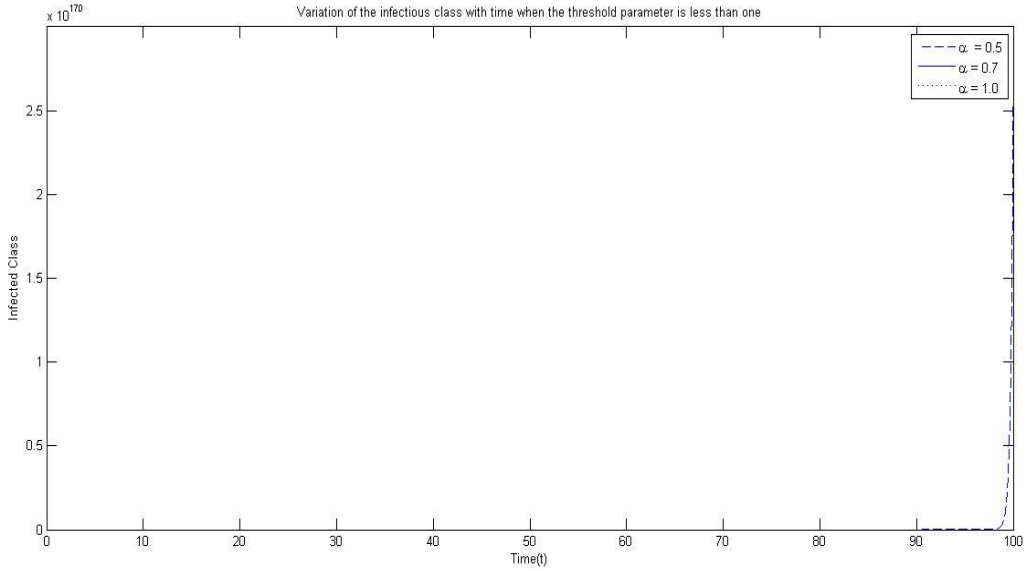


Figure 3: Variation of the Infectious class with time when $R_0 < 1$.

Here, we see that the infected humans are decreasing asymptotically with time. This implies that when $R_0<1$, an infected human would not infect up to one average human in the population therefore implies that the infected humans will recover continuously until no human would be infected in the population and hence, shows the stability of the disease-free equilibrium point.

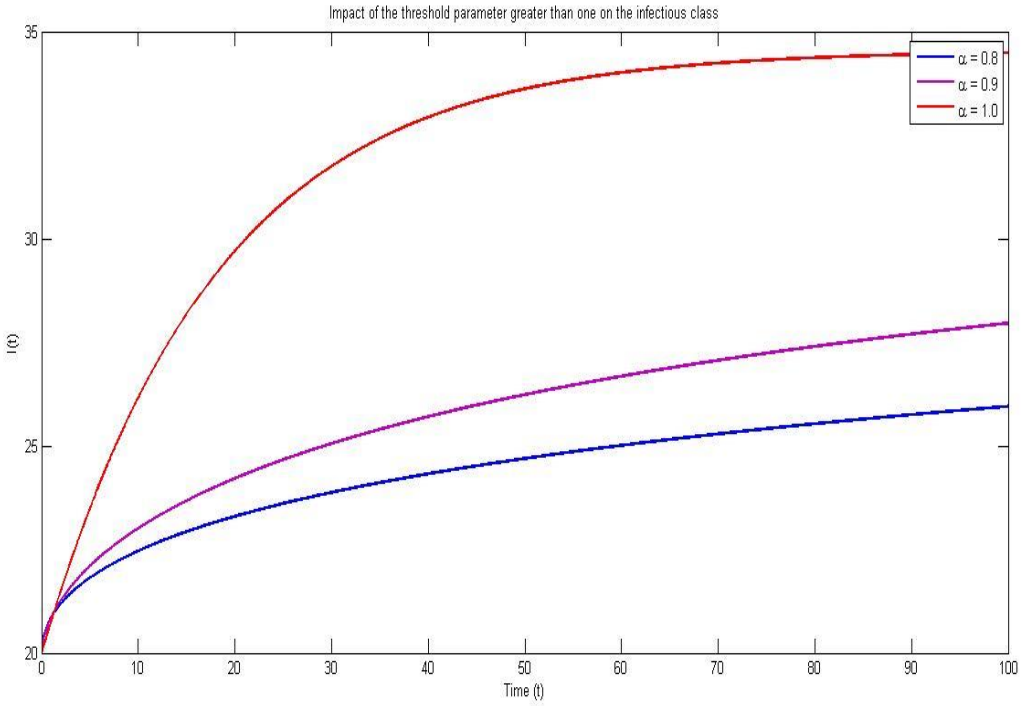


Figure 4: Variation of the Infectious class with time when $R_0 > 1$

The graph above shows the dynamics of the infected humans in an endemic equilibrium state. The graph shows that the infected humans keep increasing as the contact rate,β, was increased This implies that, an infected human would infect more than one average susceptible human hence, would keep increasing until all human become infected therefore making the disease to persist with time in the population.

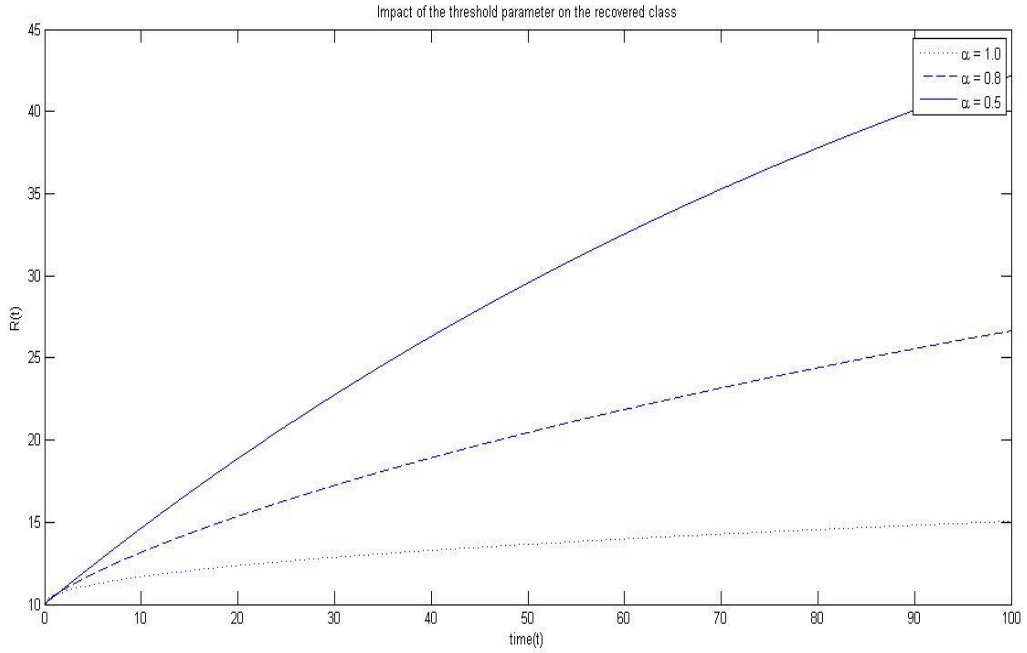


Figure 5: Variation of the recovered class with time when $R_0 < 1$.

Here, we see an increasing graph for the recovered class when $R_0<1$**.**

This shows that recovered class will increase over time as an infected humans does not infect up to an average susceptible human when the threshold parameter is less than 1,hence the infected humans keep decreasing as most of them get recovered until no human is infected when $R_0 < 1$.

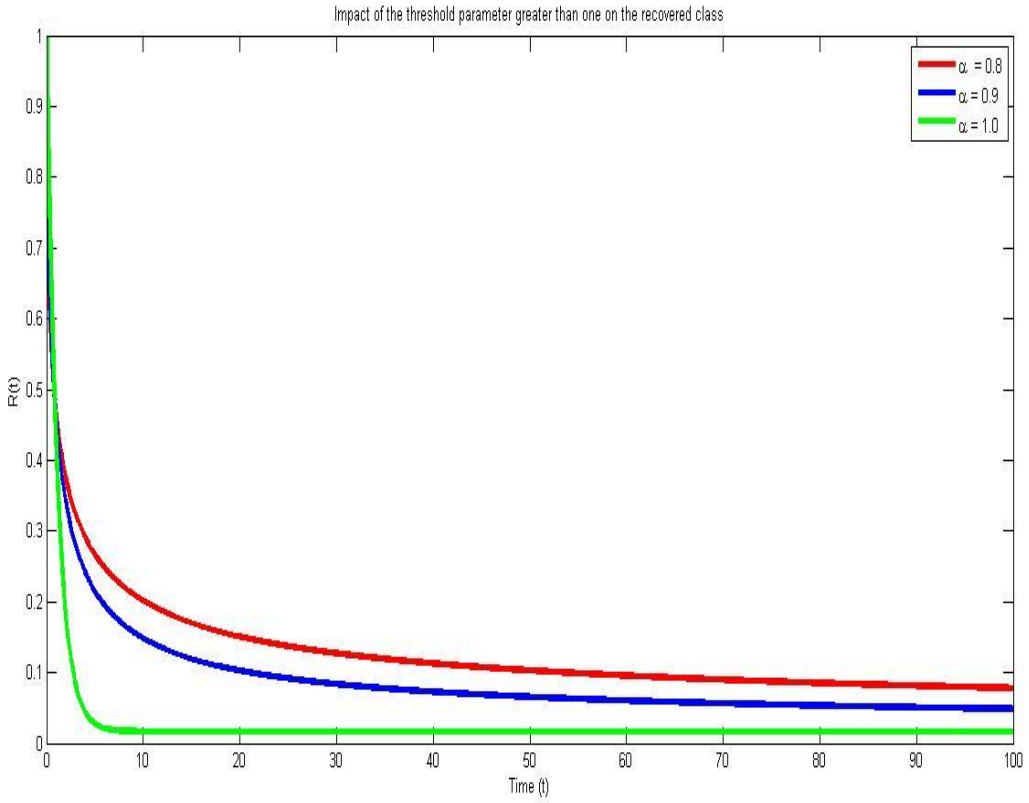


Figure 6: Variation of the recovered class with time with $R_0 > 1$.

The graph shows the variation of the recovered computers with time at endemic equilibrium point. At endemic equilibrium point, we see that we reduce the rate at which infected computers become recovered,γ, and also increase the contact rate,β, the recovered computers decreases asymptotically as seen above. This implies that, at EEP the virus would invade the population so the recovered class would be diminishing with time.

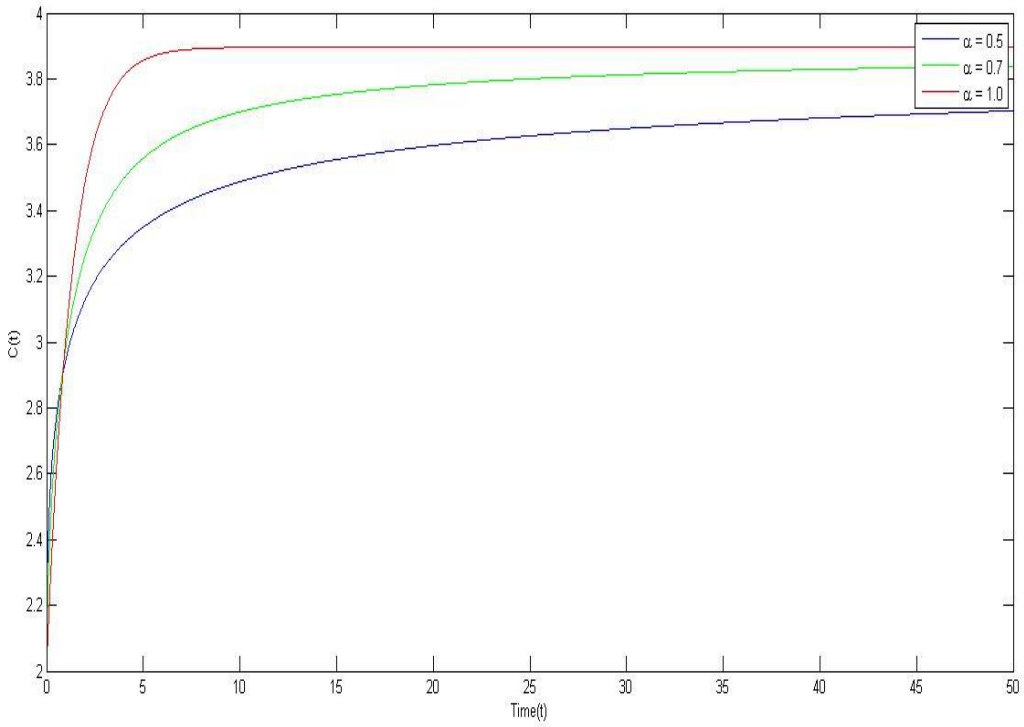


Figure 7: Variation of the cross-immune class with time when $R_0 < 1$.

This shows that the Cross-immue class increases over time when $R_0 < 1$ since the contact rate between the susceptible computers and the infected computers is very minimal thereby making the model stable. Clearly, it can also be seen that as the value of α increases, the model becomes unstable telling us that the fractional case gives a better result than the classical case

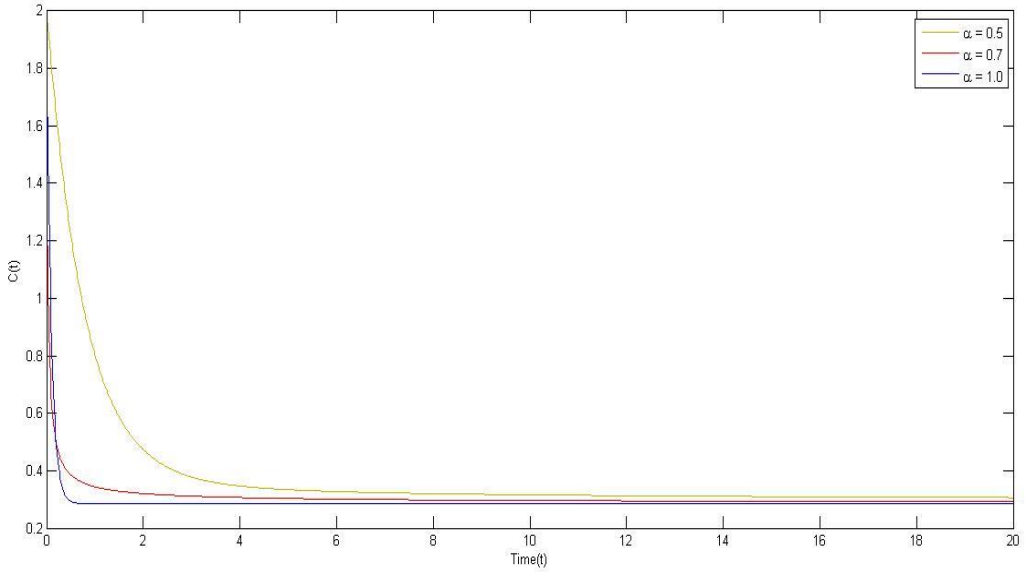


Figure 8: **Variation of the cross-immune class with time when $R_0 > 1$.**

This shows a decreasing asymptotic curve for the cross-immune class when $R_0 > 1$ hence it is line with the fact that when the threshold parameter is greater than one, more than an average of one susceptible individual would be infected, hence the disease invades the population.

5.1.3 DISCUSSION OF RESULTS

Figure 1 shows how the susceptible class vary with time when $R_0<1$. Clearly, it can be seen that the decreasing did not intercept on the horizontal axis hence, tells us that the disease will not invade the population rather will vanish with time. Also, at every time, the curve with $\alpha = 0.5$ gave a higher value than $\alpha = 1.0$ hence the fractional case gives a better result than the classical case. Figure 2 shows that when $R_0 > 1$ that the susceptible population would eventually go into extinction as the curve intercepts with the horizontal axis with time. Figure 3 depicts that the infectious class decreases with time and that the Influenza A vanishes with time when R0 < 1. Figure 4 has the same result as that of figure 3 as the variation didn't have so much impact on the infected class. Figure 5 shows an increasing curve for the recovered population when $R_0 < 1$ hence

signifies that the virus will not invade the population. Figure 6 shows a decreasing graph for the recovered class when $R_0 > 1$ hence it is line with the fact that when the threshold parameter is greater than one, the disease invades the population. Figure 7 shows an increasing curve for the cross-immune population when $R_0 < 1$ hence signifies that the virus will not invade the population. Figure 8 shows a decreasing graph for the cross-immune class when $R_0 > 1$ hence it is line with the fact that when the threshold parameter is greater than one, the disease invades the population

5.1.4 CONCLUSION

In this work, we considered the fractional order model for Influenza A. We have obtained a stability condition for equilibrium points. We have also given a numerical example and verified our results. One should note that although the equilibrium points are the same for both integer order and fractional order models, the solution of the fractional order model tends to the fixed point over a longer period of time. One also needs to mention that when dealing with real life problems, the order of the system can be determined by using the collected data. The transformation of a classical model into a fractional one makes it very sensitive to the order of differentiation.

5.1.5 RECOMMENDATIONS

Having conducted this research, we therefore recommend that the following are the best ways to prevent Influenza A virus:

1. Avoid close contact
2. Stay home when you are sick
3. Cover your nose or mouth when sneezing or coughing
4. Cleans your hands regularly

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APPENDIX

```
function [t, y] = fde12(alpha,fdefun,t0,tfinal,y0,h,param,mu,mu_tol)

%FDE12  Solves an initial value problem for a non-linear differential
%       equation of fractional order (FDE). The code implements the
%       predictor-corrector PECE method of Adams-Bashforth-Moulton type
%       described in [1].
%
%   [T,Y] = FDE12(ALPHA,FDEFUN,T0,TFINAL,Y0,h) integrates the initial value
%   problem for the FDE, or the system of FDEs, of order ALPHA > 0
%     D^ALPHA Y(t) = FDEFUN(T,Y(T))
%     Y^(k)(T0) = Y0(:,k+1), k=0,...,m-1
%   where m is the smallest integer grater than ALPHA and D^ALPHA is the
%   fractional derivative according to the Caputo's definition. FDEFUN is a
%   function handle corresponding to the vector field of the FDE and for a
%   scalar T and a vector Y, FDEFUN(T,Y) must return a column vector. The
%   set of initial conditions Y0 is a matrix with a number of rows equal to
%   the size of the problem (hence equal to the number of rows of the
%   output of FDEFUN) and a number of columns depending on ALPHA and given
%   by m. The step-size H>0 is assumed constant throughout the integration.
%
%   [T,Y] = FDE12(ALPHA,FDEFUN,T0,TFINAL,Y0,H,PARAM) solves as above with
%   the additional set of parameters for the FDEFUN as FDEFUN(T,Y,PARAM).
%
%   [T,Y] = FDE12(ALPHA,FDEFUN,T0,TFINAL,Y0,H,PARAM,MU) solves the FDE with
%   the selected number MU of multiple corrector iterations. The following
%   values for MU are admissible:
%   MU = 0 : the corrector is not evaluated and the solution is provided
%   just by the predictor method (the first order rectangular rule);
%   MU > 0 : the corrector is evaluated by the selected number MU of times;
%   the classical PECE method is obtained for MU=1;
%   MU = Inf : the corrector is evaluated for a certain number of times
%   until convergence of the iterations is reached (for convergence the
%   difference between two consecutive iterates is tested).
%   The defalut value for MU is 1
%
%   [T,Y] = FDE12(ALPHA,FDEFUN,T0,TFINAL,Y0,H,PARAM,MU,MU_TOL) allows to
%   specify the tolerance for testing convergence when MU = Inf. If not
%   specified, the default value MU_TOL = 1.E-6 is used.
%
%   FDE12 is an implementation of the predictor-corrector method of
%   Adams-Bashforth-Moulton studied in [1]. Convergence and accuracy of
%   the method are studied in [2]. The implementation with multiple
%   corrector iterations has been proposed and discussed for multiterm FDEs
%   in [3]. In this implementation the discrete convolutions are evaluated
%   by means of the FFT algorithm described in [4] allowing to keep the
```

```
%   computational cost proportional to N*log(N)^2 instead of N^2 as in the
%   classical implementation; N is the number of time-point in which the
%   solution is evaluated, i.e. N = (TFINAL-T)/H. The stability properties
%   of the method implemented by FDE12 have been studied in [5].
%
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%
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%   for fractional differential equations, Internat. J. Comput. Math. 87
%   (10) (2010) 2281-2290.
%
%   Copyright (c) 2011-2012, Roberto Garrappa, University of Bari, Italy
%   garrappa at dm dot uniba dot it
%   Revision: 1.2 - Date: July, 6 2012

% Check inputs
if nargin < 9
   mu_tol = 1.0e-6 ;
   if nargin < 8
      mu = 1 ;
      if nargin < 7
         param = [] ;
      end
   end
end

% Check order of the FDE
if alpha <= 0
   error('MATLAB:fde12:NegativeOrder',...
      ['The order ALPHA of the FDE must be positive. The value ' ...
       'ALPHA = %f can not be accepted. See FDE12.'], alpha);
```

```
end

% Check the step--size of the method
if h <= 0
   error('MATLAB:fde12:NegativeStepSize',...
      ['The step-size H for the method must be positive. The value ' ...
       'H = %e can not be accepted. See FDE12.'], h);
end

% Structure for storing initial conditions
ic.t0 = t0 ;
ic.y0 = y0 ;
ic.m_alpha = ceil(alpha) ;
ic.m_alpha_factorial = factorial(0:ic.m_alpha-1) ;

% Structure for storing information on the problem
Probl.ic = ic ;
Probl.fdefun = fdefun ;
Probl.problem_size = size(y0,1) ;
Probl.param = param ;

% Check number of initial conditions
if size(y0,2) < ic.m_alpha
   error('MATLAB:fde12:NotEnoughInputs', ...
      ['A not sufficient number of assigned initial conditions. ' ...
      'Order ALPHA = %f requires %d initial conditions. See FDE12.'], ...
      alpha,ic.m_alpha);
end

% Check compatibility size of the problem with size of the vector field
f_temp = f_vectorfield(t0,y0(:,1),Probl) ;
if Probl.problem_size ~= size(f_temp,1)
   error('MATLAB:fde12:SizeNotCompatible', ...
      ['Size %d of the problem as obtained from initial conditions ' ...
       '(i.e. the number of rows of Y0) not compatible with the ' ...
       'size %d of the output of the vector field FDEFUN. ' ...
       'See FDE12.'], Probl.problem_size,size(f_temp,1));
end

% Number of points in which to evaluate weights and solution
r = 16 ;
N = ceil((tfinal-t0)/h) ;
Nr = ceil((N+1)/r)*r ;
Q = ceil(log2(Nr/r)) - 1 ;
NNr = 2^(Q+1)*r ;
```

```
% Preallocation of some variables
y = zeros(Probl.problem_size,N+1) ;
fy = zeros(Probl.problem_size,N+1) ;
zn_pred = zeros(Probl.problem_size,NNr+1) ;
if mu > 0
   zn_corr = zeros(Probl.problem_size,NNr+1) ;
else
   zn_corr = 0 ;
end

% Evaluation of coefficients of the PECE method
nvett = 0 : NNr+1 ;
nalpha = nvett.^alpha ;
nalpha1 = nalpha.*nvett ;
PC.bn = nalpha(2:end) - nalpha(1:end-1) ;
PC.an = [ 1 , (nalpha1(1:end-2) - 2*nalpha1(2:end-1) + nalpha1(3:end)) ] ;
PC.a0 = [ 0 , nalpha1(1:end-2)-nalpha(2:end-1).*(nvett(2:end-1)-alpha-1)] ;
PC.halpha1 = h^alpha/gamma(alpha+1) ;
PC.halpha2 = h^alpha/gamma(alpha+2) ;
PC.mu = mu ; PC.mu_tol = mu_tol ;

% Initializing solution and proces of computation
t = t0 + (0 : N)*h ;
y(:,1) = y0(:,1) ;
fy(:,1) = f_temp ;
[y, fy] = Triangolo(1, r-1, t, y, fy, zn_pred, zn_corr, N, PC, Probl ) ;

% Main process of computation by means of the FFT algorithm
ff = [0 2 ] ; nx0 = 0 ; ny0 = 0 ;
for q = 0 : Q
   L = 2^q ;
   [y, fy] = DisegnaBlocchi(L, ff, r, Nr, nx0+L*r, ny0, t, y, fy, ...
                        zn_pred, zn_corr, N, PC, Probl ) ;
   ff = [ff ff]  ; ff(end) = 4*L ;
end

% Evaluation solution in TFINAL when TFINAL is not in the mesh
if tfinal < t(N+1)
   c = (tfinal - t(N))/h ;
   t(N+1) = tfinal ;
   y(:,N+1) = (1-c)*y(:,N) + c*y(:,N+1) ;
end
t = t(1:N+1) ; y = y(:,1:N+1) ;

end
```

```
%
=========================================================================
====
%
=========================================================================
====
% r : dimension of the basic square or triangle
% L : factor of resizing of the squares
function [y, fy] = DisegnaBlocchi(L, ff, r, Nr, nx0, ny0, t, y, fy, ...
                                  zn_pred, zn_corr, N , PC, Probl)

nxi = nx0 ; nxf = nx0 + L*r - 1 ;
nyi = ny0 ; nyf = ny0 + L*r - 1 ;
is = 1 ;
s_nxi(is) = nxi ; s_nxf(is) = nxf ; s_nyi(is) = nyi ; s_nyf(is) = nyf ;

i_triangolo = 0 ; stop = 0 ;
while ~stop

   stop = nxi+r-1 == nx0+L*r-1 | (nxi+r-1>=Nr-1) ; % Ci si ferma quando il triangolo attuale
finisce alla fine del quadrato

   [zn_pred, zn_corr] = Quadrato(nxi, nxf, nyi, nyf, fy, zn_pred, zn_corr, PC, Probl) ;

   [y, fy] = Triangolo(nxi, nxi+r-1, t, y, fy, zn_pred, zn_corr, N, PC, Probl) ;
   i_triangolo = i_triangolo + 1 ;

   if ~stop
      if nxi+r-1 == nxf   % Il triangolo finisce dove finisce il quadrato -> si scende di livello
         i_Delta = ff(i_triangolo) ;
         Delta = i_Delta*r ;
         nxi = s_nxf(is)+1 ; nxf = s_nxf(is)  + Delta ;
         nyi = s_nxf(is) - Delta +1; nyf = s_nxf(is)  ;
         s_nxi(is) = nxi ; s_nxf(is) = nxf ; s_nyi(is) = nyi ; s_nyf(is) = nyf ;
      else % Il triangolo finisce prima del quadrato -> si fa un quadrato accanto
         nxi = nxi + r ; nxf = nxi + r - 1 ; nyi = nyf + 1 ; nyf = nyf + r  ;
         is = is + 1 ;
         s_nxi(is) = nxi ; s_nxf(is) = nxf ; s_nyi(is) = nyi ; s_nyf(is) = nyf ;
      end
   end

end

end
```

```
%
================================================================================
%
================================================================================
function [zn_pred, zn_corr] = Quadrato(nxi, nxf, nyi, nyf, fy, zn_pred, zn_corr, PC, Probl)

coef_beg = nxi-nyf ; coef_end = nxf-nyi+1 ;
funz_beg = nyi+1 ; funz_end = nyf+1 ;

% Evaluation convolution segment for the predictor
vett_coef = PC.bn(coef_beg:coef_end) ;
vett_funz = [fy(:,funz_beg:funz_end) , zeros(Probl.problem_size,funz_end-funz_beg+1) ] ;
zzn_pred = real(FastConv(vett_coef,vett_funz)) ;
zn_pred(:,nxi+1:nxf+1) = zn_pred(:,nxi+1:nxf+1) + zzn_pred(:,nxf-nyf+1-1:end-1) ;

% Evaluation convolution segment for the corrector
if PC.mu > 0
   vett_coef = PC.an(coef_beg:coef_end) ;
   if nyi == 0 % Evaluation of the lowest square
      vett_funz = [zeros(Probl.problem_size,1) , fy(:,funz_beg+1:funz_end) ,
zeros(Probl.problem_size,funz_end-funz_beg+1) ] ;
   else % Evaluation of any square but not the lowest
      vett_funz = [ fy(:,funz_beg:funz_end) , zeros(Probl.problem_size,funz_end-funz_beg+1) ] ;
   end
   zzn_corr = real(FastConv(vett_coef,vett_funz)) ;
   zn_corr(:,nxi+1:nxf+1) = zn_corr(:,nxi+1:nxf+1) + zzn_corr(:,nxf-nyf+1:end) ;
else
   zn_corr = 0 ;
end

end

%
================================================================================
%
================================================================================
function [y, fy] = Triangolo(nxi, nxf, t, y, fy, zn_pred, zn_corr, N, PC, Probl)

for n = nxi : min(N,nxf)

   % Evaluation of the predictor
   Phi = zeros(Probl.problem_size,1) ;
```

```
    if nxi == 1 % Case of the first triangle
       j_beg = 0 ;
    else % Case of any triangle but not the first
       j_beg = nxi ;
    end
    for j = j_beg : n-1
       Phi = Phi + PC.bn(n-j)*fy(:,j+1) ;
    end

    St = StartingTerm(t(n+1),Probl.ic) ;
    y_pred = St + PC.halpha1*(zn_pred(:,n+1) + Phi) ;
    f_pred = f_vectorfield(t(n+1),y_pred,Probl) ;

    if PC.mu == 0
       y(:,n+1) = y_pred ;
       fy(:,n+1) = f_pred ;
    else
       j_beg = nxi ;
       Phi = zeros(Probl.problem_size,1) ;
       for j = j_beg : n-1
          Phi = Phi + PC.an(n-j+1)*fy(:,j+1) ;
       end
       Phi_n = St + PC.halpha2*(PC.a0(n+1)*fy(:,1) + zn_corr(:,n+1) + Phi) ;
       yn0 = y_pred ; fn0 = f_pred ;
       stop = 0 ; mu_it = 0 ;
       while ~stop
          yn1 = Phi_n + PC.halpha2*fn0 ;
          mu_it = mu_it + 1 ;
          if PC.mu == Inf
             stop = norm(yn1-yn0,inf) < PC.mu_tol ;
             if mu_it > 100 && ~stop
                warning('MATLAB:fde12:NonConvegence',...
                   strcat('It has been requested to run corrector iterations until convergence but ', ...
                   'the process does not converge to the tolerance %e in 100 iteration'),PC.mu_tol) ;
                stop = 1 ;
             end
          else
             stop = mu_it == PC.mu ;
          end
          fn1 = f_vectorfield(t(n+1),yn1,Probl) ;
          yn0 = yn1 ; fn0 = fn1 ;
       end
       y(:,n+1) = yn1 ;
       fy(:,n+1) = fn1 ;
    end
end
```

```
end


%
=========================================================================
====
%
=========================================================================
====
function z = FastConv(x, y)

r = length(x) ; problem_size = size(y,1) ;

z = zeros(problem_size,r) ;
X = fft(x,r) ;
for i = 1 : problem_size
   Y = fft(y(i,:),r) ;
   Z = X.*Y ;
   z(i,:) = ifft(Z,r) ;
end

end


%
=========================================================================
====
%
=========================================================================
====
function f = f_vectorfield(t,y,Probl)

if isempty(Probl.param)
   f = feval(Probl.fdefun,t,y) ;
else
   f = feval(Probl.fdefun,t,y,Probl.param) ;
end

end


%
=========================================================================
====
```

```
%
=========================================================================
====
function ys = StartingTerm(t,ic)

ys = zeros(size(ic.y0,1),1) ;
for k = 1 : ic.m_alpha
   ys = ys + (t-ic.t0)^(k-1)/ic.m_alpha_factorial(k)*ic.y0(:,k) ;
end

end
```