Vol.3.Issue.4., 2015
(Oct-Dec.)

Bulletin of Pharmaceutical and Medical Sciences (BOPAMS)
A Peer Reviewed International Journal http://www.bopams.com

RESEARCH ARTICLE

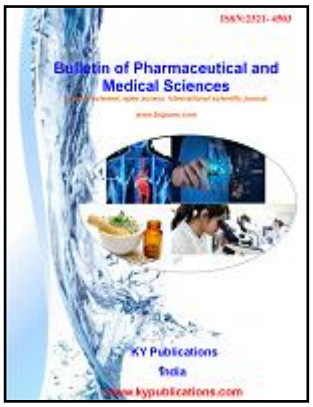
INTERNATIONAL STANDARD SERIAL NUMBER
E-ISSN 2341-4103

APPLICATION OF FUZZY DIFFERENTIAL EQUATION IN HIV INFECTION

Dr. EMAN A. HUSSIAN[1], MAZIN H. SUHHIEM[2]

[1]Department of Mathematics, College of Sciences, AL-Mustansiriyah University, Baghdad, Iraq
(dr_ emansultan@yahoo.com)

[2]Department of Statistics, College of Adm. and Econ. , University of sumar, Al-Rifaee , Iraq .
(mz80m@yahoo.com)



ABSTRACT

In this work, a dynamical system represent the infection and the propagation of HIV is considered . Since age , sex ,… are important parameters in treatment of HIV disease , it is natural to consider the variables as fuzzy variables . Thus we consider a fuzzy dynamical system to control the HIV disease .To solve such a fuzzy dynamical system, by using r-cuts , we can convert this system to a non- Fuzzy system of differential equations, then by using numerical methods we may attempts to solve these differential equations . Therefore , we will use a modified numerical method which is called modified fuzzy artificial neural network method to solve a non-linear system of fuzzy differential equations which describe the HIV infection .

Keywords : HIV , AIDS , CD4+ T-cells , Linear Fuzzy Model of HIV Infection , Non-Linear Fuzzy Model of HIV Infection

1. INTRODUCTION

Usage of fuzzy differential equation is a natural way to model dynamical systems under uncertainty. For example these equations are used to modeling the cell growth and dynamic of population, tumor growth and the phenomenon of nuclear disintegration under uncertainty .Moreover, transition from HIV to AIDS is described through a mathematical model with fuzzy transference rate correlated with the viral load and CD4+T-cells level by rule bases [6].

Various medicine therapies have been used for achieving an optimal solution for medicine therapy of HIV in people with HIV up to now. There is a question : What is the best medicine therapy for these patients knowing that therapeutic period is limited ? The answer may be an ordinary differential equation model which describes the interaction between HIV viruses and human body immune system and then applying a suitable optimal control on the system of equations using a suitable medicine program [5] .

In real world, there are various HIV-infected patients with different strengths of immune system causing uncertainty as to the immune cells level and the viral load during the different stages of the disease. A number of mathematical models has been formulated to describe various aspects of the interaction between HIV and the immune cells . The basic and the simple model of HIV infection that contains three state variables : healthy CD4+ T-cells , infected CD4+ T-cells , and viruses , is presented by Perelson, Neumann and et al. in [1] , and more complicated models containing other parts of the immune system such as the cytotoxic T-lymphocyte and the macrophages are presented in [3]. None of these models can mirror the mentioned uncertainties proposing a mathematical model with fuzzy parameters which could reflect such ambiguities would be desirable .

In this work a dynamical system represent the infection and the propagation of HIV has been considered , we solved the nonlinear system by using modified fuzzy artificial neural network method (For more details about this method see[9,10,11,12,13]) . Also , Najariyan , Farahi and Alavian [4] solved this problem by using generalized Euler method.

Based on the opinions of some specialists to this topic , also based on the results in [5 , 7 , 8] the results that we obtained by using modified fuzzy artificial neural network method are better than the results in [4] .

2. HIV and AIDS – An Important Distinction[2]

2.1. Human Immunodeficiency Virus (HIV)

HIV is a virus that aggressively attacks the immune system. Part of the reason HIV is such a serious disease is that it attacks and destroys cells of the immune system, called T-cells or CD4 cells that are designed to fight infections and diseases .

What CD4 Cells Do

CD4 cells , also known as T-cells or "helper " cells , are white blood cells essential to a healthy immune system which protects the body against bacterial,fungal,and viral infections. With a depleted immune system, the body will experience "opportunistic infections " which take advantage of the body's inability to fight infection, this characterizes the AIDS stage.

A normally functioning immune system has a " CD4 count " between 400 -1600 per cubic millimeter of blood in men, and between 500-1600 in women . A normal CD4 count can fluctuate slightly due to factors such as a good night's sleep ,nutrition ,menstruation among women, and the use of other medications or treatments . However , with HIV a person's CD4 count will drop severely usually stabilizing around 500-600 parts per cubic millimeter.

It is estimated that without treatment, CD4 cells will continue to deplete by approximately 45 cell every six months, with greater declines among those with higher CD4 counts before HIV infection. A CD4 count of 200-500 indicates that damage to the immune system has occurred.CD4 counts are typically used to help determine when antiretroviral treatment is needed, and it is recommended that once CD4 counts drop below 200-250 treatment is needed to prevent opportunistic infections and progression to the AIDS stage .

Evidence has also shown that if a person's CD4 count drops below 200,they are unlikely to respond well to treatment. This demonstrates the importance of well managed and accessible diagnostic treatment in the developing world, as people are being placed at higher rate of AIDS related illnesses without access and adherence to treatment in a timely fashion . Although a person infected with HIV can live a normal life for many years with no visible signs of illness, internally the HIV virus continues to damage the immune system until a time where the immune system will be too weak to fight off opportunistic infections and eventually will succumb to AIDS .

A person can be infected with HIV and not know it ,because symptoms or illnesses related to HIV may not occur for many years, or people may confuse the symptoms of the initial drop in CD4 cells as a flu or fatigue . But HIV infected persons are infectious for life even when asymptomatic for many years and can easily pass the infection on to others . Many people are not aware that they are HIV positive because they feel fine and transmit the disease unknowingly .This is why HIV testing is critical, and must be encouraged and readily accessible in order to stymie its progress among populations

2.2.Acquired Immune Deficiency Syndrome (AIDS)

AIDS is a condition caused by HIV . AIDS occurs when an individual is experiencing more than one chronic opportunistic infection, which results from the destruction of the body's defense by the Human Immunodeficiency Virus , HIV . It is important to remember that AIDS is very different from HIV and that it is possible, with access and adherence to treatment , to live a long , healthy life with HIV and never progress to the AIDS stage .

The timeline for someone to be infected by HIV and developed to AIDS varies from region to region. For instance, in US or Europe, the average time form HIV infection to AIDS is roughly 11 years without

treatment, while in developing countries the average time is much shorter. This is due to multiple factors related to poverty such as poor nutrition, lack of access to health care to treat preexisting infections, and greater likelihood for exposure to diseases such as malaria or tuberculosis .

3. Opportunistic Infections[1,3,5]

Various illnesses experienced by individuals who are HIV-positive are referred to as opportunistic infections . Some of these illnesses are caused by organisms that would not normally affect a healthy immune system. People living with advanced HIV infection, may experience opportunistic infections of the skin, lungs, brain, eyes, and other organs .Opportunistic illnesses common in persons diagnosed with AIDS include pneumonia ,parasitic and viral infections, and some types of cancers.

It is important to distinguish that no one actually dies from AIDS or HIV , rather a person with AIDS dies from an infection or a condition that his or her weakened immune system can no longer fight off because of AIDS .

3.1. How is HIV Transmitted ?

HIV is found in the body fluids of an infected person including :

- Blood
- Semen (produced by men before and during sex)
- Vaginal fluids (produced by women before and during sex)
- Breast milk

HIV is transmitted by body fluids in the following ways :

- Vaginal sex
- Oral sex
- Anal sex (among men and women)
- Mother-to-Child Transmission (during pregnancy ,delivery and breastfeeding)
- Injecting Drug Use
- Injections/needles (contaminated needles)
- Blood Transfusions
- Cutting tools (skin-piercing instruments , tattoo needles and circumcision instruments).

4. What Happens in The Body When HIV-Infection Occurs ?[2,4]

HIV infects cells that are part of the body's immune system. As more cells are infected by the virus, the immune system becomes less able to fight off disease.

To productively infect a cell , HIV must introduce its genetic material into the interior of the cell .This process begins with attachment and entry of the virus , uncoating of the virus membrane and integration of the virus genes into the human gene . The human cell is hijacked to manufacture viral building blocks for multiple copies that are subsequently assembled, eventually breaking out of the infected cell in search of other cells to infect . The virus kills the cells it infects and also kills uninfected bystander cells . The virus ensures that the human cell survives until its own multiplication is completed . Even more damaging , HIV establishes stable dormant forms that are reservoirs of infection that cannot be reached by currently available drugs . These reservoirs make complete eradication- and a cure for AIDS - a challenge .

Soon after HIV infection occurs , the body's immune system mounts an attack against the virus by means of specialized killer cells and soluble proteins called antibodies that usually succeed in temporarily lowering the amount of virus in the blood . HIV still remain active , though , continuing to infect and kill vital cells of the immune system . Over time ,viral activity significantly increases , eventually overwhelming the body's ability to fight off disease .

5. Linear Fuzzy Model of HIV Infection [6]

HIV infection can be characterized as a disease of the immune system , with progressive depletion of defensive cells, resulting in susceptibility to opportunistic infections .CD4+ T-cells,CTLs,and the virus particles play important roles in HIV infection . CD4+ T-cells are a fundamental component of the human immune

response system .These cells can be considered "messengers" or the command centers of the immune system , and they signal other immune cells that an invader is to be fought .

The immune response cells , or cytotoxic lymphocytes (CTLs),are the cells that respond to this message and set out to eliminate infection by killing infected cells . HIV can infect a number of cells in the body however, its main target is the CD4+ T-cells . HIV enters these cells by a complex process and begins to replicate , then the new virus particles are released by bursting the infected cells .

CD4+ T-cells are generated from sources within the body and are lost either by having finite life span or by bursting during the proliferation of HIV , which leads to a drop in the number of these cells, after infection and an accelerated decrease during the later stages of the disease that signals the onset AIDS. In accordance with experimental findings , too high a level of HIV impairs establishment of a lasting CTL response .

This is a delicate task, since CD4+ T-cells population, which plays an essential role in stimulation of immune response, depletes dramatically with raising the HIV load. The rate of CD4+ T-cells depletion varies greatly from patient to patient , depending on the strength or weakness of the immune system .

More precisely, a stronger immune system leads to a lower rate of CD4+ T-cells depletion and vice versa . We have a similar argument about the proliferation rate of HIV particles . Therefore ,the levels of the immune cells as well as the HIV viral load during the different stages of the disease can be considered as fuzzy quantities . According to these descriptions , the interaction of HIV with the immune system can be modeled by a system of linear differential equations with fuzzy parameters as follows :

$$\begin{aligned}\dot{\tilde{x}} &= \tilde{\lambda} - \tilde{\sigma}\tilde{x} - \tilde{c}\tilde{v}\\ \dot{\tilde{v}} &= \tilde{k}\tilde{v} - \tilde{a}\tilde{z}\\ \dot{\tilde{z}} &= \tilde{h}\tilde{x} - \tilde{\tau}\tilde{v}\end{aligned} \qquad (1)$$

Where the fuzzy functions $\tilde{x}(t)$, $\tilde{z}(t)$, and $\tilde{v}(t)$ indicate the level of CD4+ T-cells , CTLs , and the HIV viral load at time t,respectively .Most of the terms in the model have straightforward interpretations as follows . The first equation in (1) represents the dynamics of the concentration of CD4+ T-cells . The CD4+ T-cells are produced from a source , such as the thymus , at a constant rate $\tilde{\lambda}$. Here , we have assumed that CD4+ T-cells have a finite life span and die at a rate $\tilde{\sigma}$ per cell .Therefore ,the number of these cells, which are lost due to natural death , is represented through the loss term $\tilde{\sigma}\tilde{x}$ in the first equation .

Moreover , the CD4+ T-cell population is lost through infection by a virus particle at a rate of $\tilde{c}$, and so the term $\tilde{c}\tilde{v}$ models the rate that free viruses destroy CD4+ T-cells . The second equation in (1) depicts the rate of change in the virus population . An HIV particle uses a host cell to replicate itself and thus proliferates with a growth rate $\tilde{k}$. Thus , the total amount of produced viruses is given by the term $\tilde{k}\tilde{v}$. Infected cells are killed by CTLs , and hence viruses are lost through an immune response . Assuming that a CTL eliminates the virus particles at a rate $\tilde{a}$, the number of virus particles eliminated by the immune response is given by the term $\tilde{a}\tilde{z}$.

The third equation in (1) describes the dynamics of CTLs during HIV infection . A CD4+ T-cell stimulates CTLs to proliferate at a rate $\tilde{h}$. Therefore , CD4+ T-cells effect on proliferation of CTLs is expressed by the term $\tilde{h}\tilde{x}$. The term $\tilde{\tau}\tilde{v}$ takes into account loss of CTLs due to increasing the HIV viral load where $\tilde{\tau}$ is the rate at which the virus induced impairment of CD4+ T-cell function occurs .

6. Non-Linear Fuzzy Model of HIV Infection[4]

Consider the following equations for HIV infection :

$$\begin{aligned}\frac{dn}{dt} &= r - an - \beta nv\\ \frac{di}{dt} &= \beta nv - bi\\ \frac{dv}{dt} &= ki - sv\end{aligned} \qquad (2)$$

The parameters and the initial values are shown respectively in table (1) and table (2)

Table (1):Parameters of the microscopic HIV model .

$r = 7$	$a = 0.007$	$\beta = 42163 \times 10^{-11}$
$b = 0.0999$	$s = 0.2$	$k = 90.67$

Table (2): Initial conditions of the crisp model .

$n(0)$	1000
$i(0)$	5
$v(0)$	2000
t initial	0 time units
t final	500 time units

Where n denotes the uninfected cells , i denotes infected cells and V is the number free virus particles .

Uninfected cells are produced at a constant rate r , and die at a rate an , and uninfected cells with free virus produce the infected cells at rate βnv , in the second equation of (2) infected cells die at rate bi ,and in the third equation free virus is produced from infected cells at rate ki , and die at rate sv .

Since age , sex , feeding ,… are important parameters in treatment of HIV disease , it is natural to consider the variables as fuzzy variables , the fuzzy form of differential equation system (2) is :

$$\begin{aligned} \frac{d\tilde{n}}{dt} &= r - a\tilde{n} - \beta\tilde{n}\tilde{v} \\ \frac{d\tilde{i}}{dt} &= \beta\tilde{n}\tilde{v} - b\tilde{i} \\ \frac{d\tilde{v}}{dt} &= k\tilde{i} - s\tilde{v} \end{aligned} \qquad (3)$$

The initial conditions of the fuzzy model are shown in table (3)

Table (3) :Initial conditions of the fuzzy model .

$\tilde{n}(0)$	(850,1000,1150)
$\tilde{i}(0)$	(3,5,7)
$\tilde{v}(0)$	(1750,2000,2250)

7 Numerical Results

In this section we report on some numerical results and the solution of system (3) . In this solution we used a three-layer feed-forward fuzzy neural network having one input unit , one hidden layer with 10 hidden units (neurons) and one output unit.

Rewrite the system (3) with the constants in the table (1) to get

$$\begin{aligned} \frac{d\tilde{n}}{dt} &= 7 - 0.007\tilde{n} - 0.00000042163\tilde{n}\tilde{v} \\ \frac{d\tilde{i}}{dt} &= 0.00000042163\tilde{n}\tilde{v} - 0.0999\tilde{i} \\ \frac{d\tilde{v}}{dt} &= 90.67\tilde{i} - 0.2\tilde{v} \,. \qquad t \in [0,500] \end{aligned} \qquad (4)$$

with the fuzzy initial conditions :

$$\begin{aligned} [n(0)]_r &= [150r + 850, 1150 - 150r], \\ [i(0)]_r &= [2r + 3, 7 - 2r] \\ [v(0)]_r &= [250r + 1750, 2250 - 250r] \quad . \end{aligned} \qquad (5)$$

The fuzzy trial solutions for the system (4) are :

$$\begin{aligned} [n_T(t)]_r &= [150r + 850, 1150 - 150r] + t[N_1(Q(t), p_1]_r \\ [i_T(t)]_r &= [2r + 3, 7 - 2r] + t[N_2(Q(t), p_2]_r \\ [v_T(t)]_r &= [250r + 1750, 2250 - 250r] + t[N_3(Q(t), p_3]_r \,. \end{aligned} \qquad (6)$$

The error function for m = 10 units in the hidden layer and for g = 11 equally spaced points inside the interval $[0, 500]$ is trained, we consider one FFNN for each fuzzy trial solution.

For this problem , minimized error function has the form

$E = \sum_{i=1}^{11}(E_{ir}^{L} + E_{ir}^{U})$, where (7)

$E_{ir}^{L} =$

$[t_i \frac{\partial[N_1(Q(t_i),p_1)]_r^L}{\partial t} + (1 + 0.0077t_i + 0.0001rt_i)[N_1(Q(t_i),p_1)]_r^L + (0.0004t_i + 0.0001rt_i)[N_3(Q(t_i),p_3)]_r^L +$
0.00000042163t2[N1Qti,p1]rL[N3Qti,p3]rL−0.4238+1.25r+0.0158r2]2
+ $[t_i \frac{\partial[N_2(Q(t_i),p_2)]_r^L}{\partial t} - (0.0007t_i + 0.0001rt_i)[N_1(Q(t_i),p_1)]_r^L + (1 + 0.0999t_i)[N_2(Q(t_i),p_2)]_r^L +$
0.0004ti+0.0001rti[N3Qti,p3]rL−0.00000042163t2[N1Qti,p1]rL[N3Qti,p3]rL−0.3265−0.0002r−0.0158r2]
2
$+[t_i \frac{\partial[N_3(Q(t_i),p_3)]_r^L}{\partial t} - 90.67t_i[N_2(Q(t_i),p_2)]_r^L + (1 + 0.2t_i)[N_3(Q(t_i),p_3)]_r^L - 131.34r + 77.99]^2$ (8)

$E_{ir}^{U} =$

$[t_i \frac{\partial[N_1(Q(t_i),p_1)]_r^U}{\partial t} + (1 + 0.0079t_i - 0.0001rt_i)[N_1(Q(t_i),p_1)]_r^U + (0.0005t_i - 0.0001rt_i)[N_3(Q(t_i),p_3)]_r^U +$
0.00000042163t2N1Qti,p1rUN3Qti,p3rU+1.8762−1.3131r+0.0158r2]2+
$[t_i \frac{\partial[N_2(Q(t_i),p_2)]_r^U}{\partial t} + (-0.0009t_i + 0.0001rt_i)[N_1(Q(t_i),p_1)]_r^U + (1 + 0.0999t_i)[N_2(Q(t_i),p_2)]_r^U +$
−0.0005ti+0.0001rtiN3Qti,p3rU−0.00000042163t2N1Qti,p1rUN3Qti,p3rU−0.39+0.0633r−0.0158r2]2+[ti
∂N3Qti,p3rU∂t−90.67tiN2Qti,p2rU+1+0.2tiN3Qti,p3rU+131.34r−184.69]2 (9)

Where

$[N_1(Q(t),p_1)]_r^L = \sum_{j=1}^{10}[v_{j1}]_r^L\, s(Q(t)w_{j1} + b_{j1})$ (10)

$[N_2(Q(t),p_2)]_r^L = \sum_{j=1}^{10}[v_{j2}]_r^L\, s(Q(t)w_{j2} + b_{j2})$ (11)

$[N_3(Q(t),p_3)]_r^L = \sum_{j=1}^{10}[v_{j3}]_r^L\, s(Q(t)w_{j3} + b_{j3})$ (12)

$\frac{\partial[N_1(Q(t),p_1)]_r^L}{\partial t} = \sum_{j=1}^{10}[v_{j1}]_r^L\, w_{j1}\, \epsilon\, s'(Q(t)w_{j1} + b_{j1})$ (13)

$\frac{\partial[N_2(Q(t),p_2)]_r^L}{\partial t} = \sum_{j=1}^{10}[v_{j2}]_r^L\, w_{j2}\, \epsilon\, s'(Q(t)w_{j2} + b_{j2})$ (14)

$\frac{\partial[N_3(Q(t),p_3)]_r^L}{\partial t} = \sum_{j=1}^{10}[v_{j3}]_r^L\, w_{j3}\, \epsilon\, s'(Q(t)w_{j3} + b_{j3})$ (15)

$[N_1(Q(t),p_1)]_r^U = \sum_{j=1}^{10}[v_{j1}]_r^U\, s(Q(t)w_{j1} + b_{j1})$ (16)

$[N_2(Q(t),p_2)]_r^U = \sum_{j=1}^{10}[v_{j2}]_r^U\, s(Q(t)w_{j2} + b_{j2})$ (17)

$[N_3(Q(t),p_3)]_r^U = \sum_{j=1}^{10}[v_{j3}]_r^U\, s(Q(t)w_{j3} + b_{j3})$ (18)

$\frac{\partial[N_1(Q(t),p_1)]_r^U}{\partial t} = \sum_{j=1}^{10}[v_{j1}]_r^U\, w_{j1}\, \epsilon\, s'(Q(t)w_{j1} + b_{j1})$ (19)

$\frac{\partial[N_2(Q(t),p_2)]_r^U}{\partial t} = \sum_{j=1}^{10}[v_{j2}]_r^U\, w_{j2}\, \epsilon\, s'(Q(t)w_{j2} + b_{j2})$ (20)

$\frac{\partial[N_3(Q(t),p_3)]_r^U}{\partial t} = \sum_{j=1}^{10}[v_{j3}]_r^U\, w_{j3}\, \epsilon\, s'(Q(t)w_{j3} + b_{j3})$ (21)

For $\epsilon = 0.9$,approximate solutions for this problem can be found in tables (4)-(8) and figures (1)-(6) .

8. Discussion and Conclusion

After solution the fuzzy system (4) by using different values for r ,we noticed over time that number of non-infected cells begin downward , while the number of infected cells begin upward . As for the number of viruses , it also starts increase . Increase the number of infected cells and decrease the number of non-infected cells is normal . The reason for increasing the number of viruses , it returns to the infected cells will become a source for the production of these viruses, increase the number of infected cells increases the number of viruses that attack other cells .

Najariyan , Farahi and Alavian [4] solved problem (4) by using generalized Euler method. Based on the opinions of some specialists to this topic , also based on the results in [5 , 7 , 8] the results that we obtained by using MFANN method are better than the results in [4] .

This preference back to that our results close to reality (more logical and admissibility) in terms of the number of infected cells , the number of non-infected cells and the number of viruses generated during a specific period of time .

Table (4) : Numerical results for system (4), for r = 0.5 .

t	$[n_T(t)]_r^L$	$[i_T(t)]_r^L$	$[v_T(t)]_r^L$	$[n_T(t)]_r^U$	$[i_T(t)]_r^U$	$[v_T(t)]_r^U$
0	925	4	1875	1075	6	2125
50	915	20	2010	1000	25	8305
100	905	36	2145	924	43	14483
150	849	51	2280	894	62	20662
200	773	67	2414	884	80	26841
250	698	83	2549	874	99	33020
300	622	99	2684	864	117	39200
350	547	114	2819	853	136	45379
400	471	130	2954	843	154	51558
450	396	146	3089	833	173	57737
500	320	162	3223	823	191	63916

Table (5) : Numerical results for system (4) , for r = 0.6 .

t	$[n_T(t)]_r^L$	$[i_T(t)]_r^L$	$[v_T(t)]_r^L$	$[n_T(t)]_r^U$	$[i_T(t)]_r^U$	$[v_T(t)]_r^U$
0	940	4	1900	1060	6	2100
50	924	20	2630	991	24	7663
100	907	36	3360	922	42	13226
150	853	52	4091	891	60	16694
200	784	68	4821	874	78	24351
250	715	84	5551	858	96	29914
300	646	100	6281	841	114	35477
350	577	116	7011	825	132	41039
400	508	132	7741	808	150	46602
450	439	148	8472	792	168	52165
500	370	164	9202	775	186	57728

Table (6) : Numerical results for system (4) , for r = 0.7 .

t	$[n_T(t)]_r^L$	$[i_T(t)]_r^L$	$[v_T(t)]_r^L$	$[n_T(t)]_r^U$	$[i_T(t)]_r^U$	$[v_T(t)]_r^U$
0	955	4	1925	1045	6	2075
50	932	8	3250	983	20	7043
100	909	36	4576	921	42	12010
150	859	52	5901	886	60	16978
200	797	68	7226	863	78	21945
250	735	84	8552	840	96	26913
300	673	100	9877	817	114	31881
350	610	116	11202	794	132	36848
400	548	132	12528	771	150	41816
450	486	148	13853	748	168	46783
500	424	164	15178	725	186	51751

Table (7) : Numerical results for system (4) , for r = 0.8 .

t	$[n_T(t)]_r^L$	$[i_T(t)]_r^L$	$[v_T(t)]_r^L$	$[n_T(t)]_r^U$	$[i_T(t)]_r^U$	$[v_T(t)]_r^U$
0	970	5	1950	1030	5	2050
50	940	21	3892	974	23	6415
100	911	38	5833	919	40	10780
150	863	54	7775	881	58	15145
200	808	71	9717	852	75	19509
250	752	87	11658	822	93	23874
300	697	104	13600	793	110	28239
350	641	120	15542	763	128	32604
400	585	137	17483	733	145	36969
450	530	153	9425	704	163	41334
500	474	170	21367	674	180	45698

Table (8) : Numerical results for system (4) , for r = 0.9.

t	$[n_T(t)]_r^L$	$[i_T(t)]_r^L$	$[v_T(t)]_r^L$	$[n_T(t)]_r^U$	$[i_T(t)]_r^U$	$[v_T(t)]_r^U$
0	985	5	1975	1015	5	2025
50	949	21	4512	966	22	5835
100	913	38	7049	917	39	9646
150	868	55	9586	877	56	13456
200	819	71	12122	841	73	17266
250	770	88	14659	805	90	21077
300	722	104	17196	769	107	24887
350	673	121	19733	733	124	28697
400	624	137	22270	697	141	32508
450	575	154	24807	661	158	36318
500	526	171	27343	625	175	40128

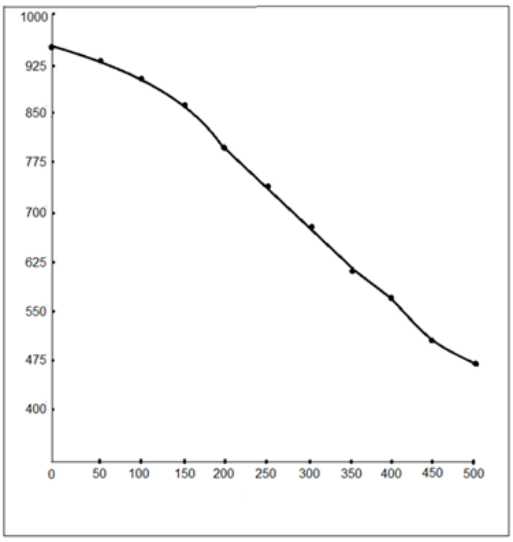


Fig. (1) Numerical results for system (4), $[n_T(t)]_r^L$, for $r = 0.75$.

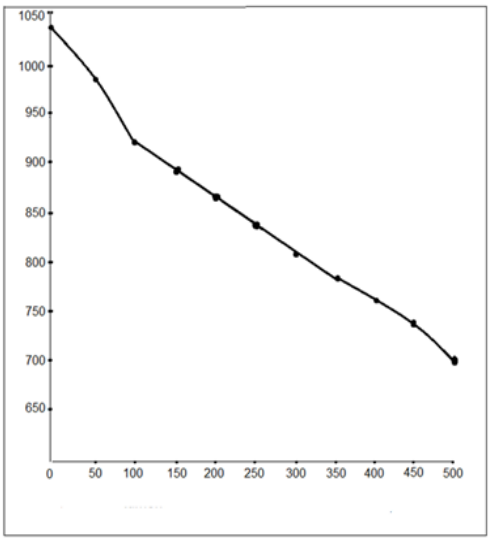


Fig. (2) Numerical results for system (4), $[n_T(t)]_r^U$, for $r = 0.75$.

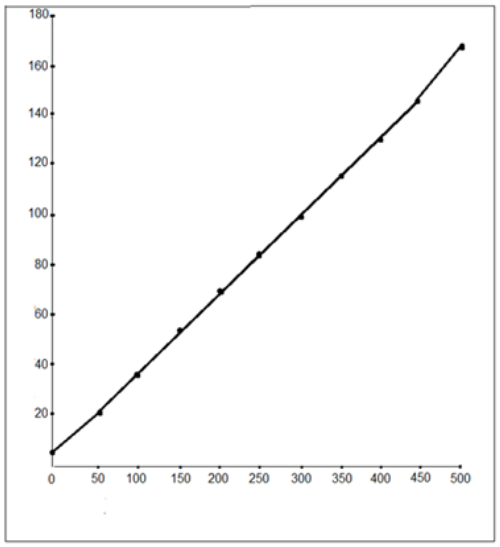


Fig. (3) Numerical results for system (4), $[i_T(t)]_r^L$, for $r = 0.75$.

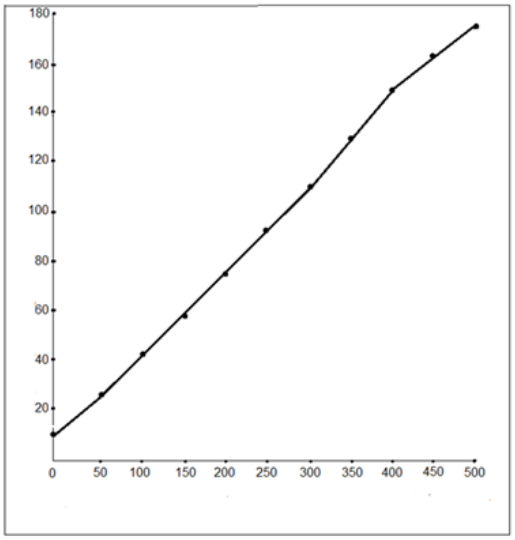


Fig. (4) Numerical results for system (4), $[i_T(t)]_r^U$, for $r = 0.75$.

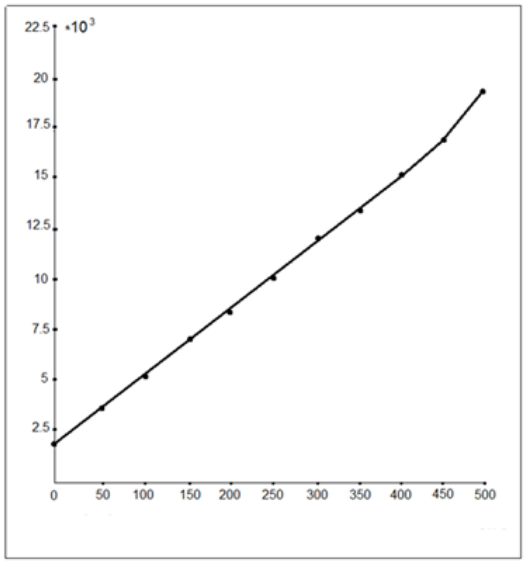


Fig. (5) Numerical results for system (4), $[v_T(t)]_r^L$, for $r = 0.75$.

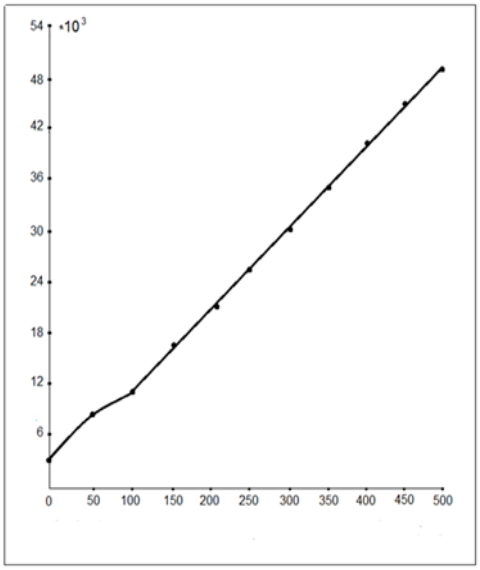


Fig. (6) Numerical results for system (4), $[v_T(t)]_r^U$, for $r = 0.75$.

REFERENCES

[1]. Perelson A. S., Neumann A. U. , et al. , "HIV-1 Dynamics in Vivo:Virion Clearance Rate , Infected Cell Life-Span , and Viral Generation Time",Journal of Science, Vol. 271 ,No. 5255 ,1582-1586 ,1996 .

[2]. Coogan M. M.,Greenspan J. ,et al. ,"Oral Lesions in Infection with Human Immunodeficiency Virus " , Bulletin of the World Health Organization , 83 , 700-706 , 2005 .

[3]. Hadjiandreou M. M. ,Conejeros R. ,et al. ,"Long-Term HIV Dynamics Subject to Continuous Therapy and Structured Treatment Interruptions", Chemical Engineering Science, Vol. 64 , No. 7 , 1600-1617,2009 .

[4]. Najariyan M., Farahi M. H. , et al. ,"Optimal Control of HIV Infection by Using Fuzzy Dynamical Systems", Journal of Mathematics and Computer Science,Vol. 2 , No. 4 , 639-649 , 2011 .

[5]. Zadeh H. G., Nejad H. C. ,"Presentation of a Fast Solution For Solving HIV-Infection Dynamics and Chemotherapy Optimization Based on Fuzzy : AVK Method " , Journal of AIDS and HIV Research , Vol.4,No.3 , 60-67 , 2012.

[6]. Zarei H., Kamyad A. V. , et al. ,"Fuzzy Modelling and Control of HIV Infection", Computational and Mathematical Methods in Medicine, Volume 2012 , Article ID 893474, 1-17 , 2012 .

[7]. Wcummins N., Badley A. D. ,"Making Sense of How HIV Kills Infected CD4T Cells : Implications For HIV Cure ", Molecular and Cellular Therapies , Vol. 2 , No. 20 , 1-7 , 2014 .

[8]. Brenchley J.M., Slides M. S. ,et al.," An HIV Epidemiological Shift and Changing Paradigms in HIV Care", ReCent Medical News , 1-8 ,March 2015 .

[9]. Hussian E. A., Suhhiem M. H. ,"Numerical Solution of Fuzzy Differential Equations Based on Taylor Series by Using Fuzzy Neural Networks", Journal of Advances in Mathematics ,Vol. 11 , No. 3 , 4080-4093 , 2015 .

[10]. Hussian E. A., Suhhiem M. H. ,"Numerical Solution of Fuzzy Differential Equations by Using Modified Fuzzy Neural Networks", International Journal of Mathematical Archive ,Vol. 6 , No. 6 , 84-94 , 2015 .

[11]. Hussian E. A., Suhhiem M. H. ," Modified Artificial Neural Networks For Solving Fuzzy Differential Equations ", Journal of Mathematical Theory And Modeling ,Vol. 5 , No. 6 , 176-192 , 2015 .

[12]. Hussian E. A., Suhhiem M. H. ," Numerical Solution of Partial Differential Equations by Using Modified Artificial Neural Networks ", Journal of Network and Complex Systems ,Vol. 5 , No. 6 , 11-21, 2015 .

[13]. Hussian E. A., Suhhiem M. H. ," Numerical Solution of Fuzzy Partial Differential Equations by Using Modified Fuzzy Neural Networks ", British Journal of Mathematics And Computer Science ,Vol. 12 , No. 2 , 1-20 , 2015 .