Analysis of model parameters in equations on the HIV/AIDS virus

Nabendra Parumasur and Robert Willie

***Abstract*—** A simple HIV/AIDs finite dimensional mathematical model on interactions of the blood cells, the HIV/AIDs virus and the immune system is studied for consistence of the equations to the real biomedical situation that they model. Definitions to model parameters indicate either that the system of equations is derived from one in infinite dimensions or can be naturally extended to cover this situation. A better understanding to the illness modelled by the finite dimensional equations is furnished. Various case studies, extracted from the current literature, are considered and numerical results show that mathematical analysis is very powerful for understanding such systems. In particular, by examining the effect of parameters in the model leads one to infer important properties on the variables of the system, such as blow up of solutions.

***Keywords*—** Dynamical properties of solutions, HIV/AIDS mathematical model equations.

I. Introduction

In this paper, we study a simple system of equations modelling interactions between blood cells, the immune system and the HIV/AIDs virus. More precisely, we study the following nonlinear system of ordinary differential equations

$$\begin{cases} \dfrac{dV}{dt} = aY - bV \\ \dfrac{dX}{dt} = c - dX - \beta XV \\ \dfrac{dY}{dt} = \beta XV - fY - \gamma YZ \\ \dfrac{dZ}{dt} = g - hZ, \end{cases} \tag{1}$$

wherein the density variables and constant parameters hold the following biological significances

$V = Number\ of\ viruses\ in\ an\ organism,$

$X = Number\ of\ uninfected\ target\ cells,$

$Y = Number\ of\ infected\ cells,$

$Z = Immune\ system\ response\ cells,$

$a = Rate\ of\ viruses\ reproduction,$

$b = Rate\ of\ death\ of\ viruses,$

$c = Reproduction\ of\ uninfected\ cells,$

$d = Death\ rate\ of\ uninfected\ cells,$

$\beta = Multiplying\ constant\ to\ being\ infected,$

$f = Death\ rate\ of\ infected\ cells,$

$g = Reproduction\ rate\ of\ cells\ Z,$

$h = Death\ rate\ of\ cells\ Z,$

$\gamma = Multiplying\ constant\ to\ the\ elimination\ of\ infected\ cells.$

A virus is a small amount of genetic material surrounded by one or more protective shells. If it gains entry to a host cell, it hijacks the cell's machinery for its own replication. It then leaves the cell, and the process is repeated. Different viruses target different host cell types for this purpose. The evolution process described by the equations (1) is as follows. The virus is replicated by the infected cells, so its rate of production is taken proportional to Y and die at a specific rate b. The uninfected cells are constantly being produced by the organism at a rate c, they die at a rate d, and become infected by the virus at rate βV, thereby entering the Y class. Infected cells die at rate $f = e + d$ where d is the natural death rate and e the additional death rate owing to the infection. The relationship between the virus and uninfected cells is analogous to that of predator and prey models [1, 2, 3, 8, 14, 15, 16]. Thus βX is a functional response of the virus to the uninfected cells. We refer the reader to other reference sources for more detailed models [25-28].

Our above finite dimensional system of equations (1) modelling interactions between blood cells, the HIV/AIDs virus and the immune system agrees with other systems of equations formulated elsewhere, for examples in the following references [2, 6, 18, 22]. However, it is the simplest formulation. Other situations still in the case of ordinary differential equations, can include a saturating functional response, and some cells entering a latent class on their infection in which they do not produce new virions, but may do so at a later stage. More complicated models in this direction, are infinite dimensional systems of equations. These take into account as well spatial effects in the biological motions. Of concern in this paper, is to give relevance even in

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the simplest form of the well posedness of the finite dimensional evolution processes. This will include a study of the immediate dynamical properties, and consistence of the model equations with the biomedical phenomena considered. Thus, although infinite time considerations of the evolutionary processes studied may seem irrealistic, since by nature the life span of any living organism is temporarily limited. These will make sense here on assumption that the times considered are measured in extremely small units.

In the elementary text book [2] by Nicholas Britton, it is mentioned that one of the problems with the HIV/AIDs virus is that it targets the killer cells themselves. Thus the number of Z cells in the blood decreases from 1000 per microlitre in the early stages of the disease to about 200 per microlitre in its full blown stage. This is a steady decrease that may take approximately ten years, even in the absence of a drug treatment and is equivalent to a five fold decrease in the ratio $\frac{f}{g}$. The reason in the delay to surfacing up of the disease is that the virus can hide for longer periods in latent cells. These are Y cells that are not recognized as infected by the immune system. The virus load increases very gradually over the course of the infection until full blown AIDs occurs, when it breaks free of the immune system control. A process measurable numerical as a ratio below which the immune system becomes below. In other words, there is a coexistence period, to which medical scientists should develop a solution that will upgrade the potential of the immune system to recognize and combat effectively the virus. This problem in equivalent terms is therefore of finding a booster to the immune system before or on the blowing up time of the virus population in the organism.

This paper is organized as follows. In Section II, we study the local well posedness of the system of ordinary differential equations (1), and we provide an analysis the stability properties of the steady state solutions. More precisely, we will identify the stationary solutions to the system of the equations. Then via a linearization of the vector field about these equilibria, we will computate the eigenvalues and analyze their signs so as to classify the nature of the steady state solutions. In Section III, we study directly from the system of equations, the global long time asymptotic dynamics. This will yield hidden information on the behaviour of the density variables. Two typical situtions found are the following. Either the HIV/AIDs virus population tends to a null state or it blows up at infinite time. In either situation, we have characterized the effects on other density variables of the system of equations. In Section IV we present numerical validation of the theoretical results.

II. Local well-posedness and analysis of the steady states

In this section, we first prove that the system of equations (1) are locally well poseded. Then, we will study the stability properties of the stationary states. This quick approach to the dynamics generated by the equations (1) is correct. Since in the theory of evolution equations when a system of equations is given without initial conditions, then its the long time asymptotic limit behaviour of that system of equations that is modelled. To initiate, we have the following theorem.

Theorem 2.1 *Let* $E = \{W \in IR^4 : \| W \|_2 \leq \rho\}$ *where* $\| \cdot \|_2$ *denotes the euclidean norm of* IR^4, $\rho > 0$ *is an adequately chosen real number, and in the finite dimensional evolution equations (1) denote the vector field by*

$$F(U) = \begin{bmatrix} aY \\ c - \beta XV \\ \beta XV - \gamma YZ \\ g \end{bmatrix}$$

$$where\ U = (V, X, Y, Z)^{\mathrm{T}}. \tag{2}$$

Also assume that given are initial conditions $U_0 \in E$. *Then, the system of nonlinear ordinary differential equations (1) has unique continuous in time solution with values in* E, *which we write as follows*

$$U(t, U_0) = e^{\mathsf{A}t} U_0 + \int_0^t e^{\mathsf{A}(t-s)} F(U(s)) ds \tag{3}$$

where

$$\mathsf{A} = \begin{bmatrix} -b & 0 & 0 & 0 \\ 0 & -d & 0 & 0 \\ 0 & 0 & -f & 0 \\ 0 & 0 & 0 & -h \end{bmatrix},$$

$$e^{\mathsf{A}t} = diag[e^{-bt}, e^{-dt}, e^{-ft}, e^{-ht}] \tag{4}$$

and is such that it is continuously differentiable on $(0, T)$. *Moreover, for all* $t \in (0, T)$, $U(t) \in E$, *the system of ordinary differential equations (1) is verified, and* $U(0) = U_0$.

Proof. We only need to prove that the nonlinearity (2) is locally Lipschitz continuous. To do this, let $U_1 = (V_1, X_1, Y_1, Z_1)^{\mathrm{T}}, U_2 = (V_2, X_2, Y_2, Z_2)^{\mathrm{T}} \in E$ Then computing the difference

$$F(U_1) - F(U_2) = \begin{bmatrix} aY_1 - aY_2 \\ -\beta X_1 V_1 + \beta X_2 V_2 \\ \beta X_1 V_1 - \beta X_2 V_2 - \gamma Y_1 Z_1 + \gamma Y_2 Z_2 \\ 0 \end{bmatrix} \tag{5}$$

It follows on taking the euclidean norm on IR^4 that in estimating from above we obtain that

$$\begin{aligned}
&\| F(U_1) - F(U_2) \|_2^2 = a^2 \mid Y_1 - Y_2 \mid^2 + \beta^2 \mid X_1 V_1 - \\
&X_2 V_2 \mid^2 + \mid \beta X_1 V_1 - \beta X_2 V_2 - \gamma Y_1 Z_1 + \gamma Y_2 Z_2 \mid^2 \\
&\le a^2 \mid Y_1 - Y_2 \mid^2 + 3\beta^2 \mid X_1 V_1 - X_2 V_1 \mid^2 + 3\beta^2 \mid X_2 V_1 - \\
&X_2 V_2 \mid^2 + 2\gamma^2 \mid Y_1 Z_1 - Y_2 Z_1 \mid^2 + 2\gamma^2 \mid Y_2 Z_1 - Y_2 Z_2 \mid^2 \\
&\le a^2 \mid Y_1 - Y_2 \mid^2 + 3\beta^2 \mid V_1 \mid^2 \mid X_1 - X_2 \mid^2 + 3\beta^2 \mid X_2 \mid^2 \\
&\mid V_1 - V_2 \mid^2 + 2\gamma^2 \mid Z_1 \mid^2 \mid Y_1 - Y_2 \mid^2 + 2\gamma^2 \mid Y_2 \mid^2 \\
&\mid Z_1 - Z_2 \mid^2 \\
&\le a^2 \mid Y_1 - Y_2 \mid^2 + 3\beta^2 \rho^2 \mid X_1 - X_2 \mid^2 + 3\beta^2 \rho^2 \\
&\mid V_1 - V_2 \mid^2 + 2\gamma^2 \rho^2 \mid Y_1 - Y_2 \mid^2 + 2\gamma^2 \rho^2 \mid Z_1 - Z_2 \mid^2 \\
&\le \max\{a, 3\beta^2 \rho^2, 2\gamma^2 \rho^2\} \\
&\left(\mid V_1 - V_2 \mid^2 + \mid X_1 - X_2 \mid^2 + \mid Y_1 - Y_2 \mid^2 + \mid Z_1 - Z_2 \mid^2 \right) \\
&= \max\{a, 3\beta^2 \rho^2, 2\gamma^2 \rho^2\} \| U_1 - U_2 \|_2^2 .
\end{aligned}$$

Therefore, the nonlinear term (2) of the system of equations is locally Lipschitz continuous. Now general results on existence and uniqueness of solutions to nonlinear ordinary differential equations in [1, 3, 8, 16] conclude our theorem. In particular, the representation (3) follows in [16] Theorem 1. pp.60. Our proof of the theorem is complete.

In what follows, we study the dynamical properties of the steady state solutions to the system of equations (1). These equilibria are the following,

$$\begin{cases} (V^*, X^*, Y^*, Z^*) = (0, \frac{c}{d}, 0, \frac{g}{h}), \\ or \quad (V^*, X^*, Y^*, Z^*) = \left(\frac{c - dX^*}{\beta X^*}, X^*, \frac{b}{a} V^*, \frac{g}{h} \right) \\ where \quad X^* = \frac{b(fh + g\gamma)}{\beta a h} \end{cases} \tag{6}$$

The first of these stationary states is an uninfected equilibrium. It is important, for this equilibrium to notice the proportional rates yielded by the biological relevant constants. In particular, there are no virions and no infected cells production. The production of the uninfected cells is given as a ratio to their death, and so is for the production of the immune system response cells. To explain the spread of the virus inside the organism. Let Θ_0 denote the basic reproductive ratio of the virus. This is an average number of new virions given rise to by a single uninfected cell. Since the functional response of the virus to uninfected cells is βX, at a rate to infected cells of a unit time over b, and each infected cell gives birth to new virions at rate a for a time $\frac{1}{f}$. Thus from the first equilibrium state we find that $\Theta_0 = \frac{\beta c a}{dbf}$ and if $\Theta_0 > 1$ we have spread of the virus. This spread is explicitly modelled in the equations, see Section 3. Relating to the second equilibrium point of the system of equations, it is important, to note the type of constants yielding X^* which are from the death processes.

To discuss the nature of the above equilibria, we use a traditional approach [1, 3, 2, 14, 16, 15, 8] based on analysis of the signs of the eigenvalues to the linearized vector field of the system of equations (1). This allows us to conclude the following lemma.

Lemma 2.2 *Consider the evolution process of interactions modelled by the equations (1), and assume all given constants are positive. Then if it holds that*

$$\frac{a\beta hc}{2b} > 2d(fh + \gamma g), \tag{7}$$

the first steady solution in (6) has dynamical properties of a saddle limit equilibrium point, otherwise it is a sink. The second steady state solution of (6) has dynamical properties of a saddle limit equilibrium point.

Proof. Let $F(V, X, Y, Z)$ denote the vector field of the equations (1). Then computing the Jacobian matrix associated with $F(V, X, Y, Z)$, we find that

$$DF(V, X, Y, Z) = \begin{bmatrix} -b & 0 & a & 0 \\ -\beta X & -d - \beta V & 0 & 0 \\ \beta X & \beta V & -f - \gamma Z & -\gamma Y \\ 0 & 0 & 0 & -h \end{bmatrix}. \tag{8}$$

Therefore evaluating this at the first steady state solution and computing the determinant

$$\mid DF(0, \frac{c}{d}, 0, \frac{g}{h}) - \lambda I \mid = 0$$

we get that

$$(-h - \lambda)(-d - \lambda)\left((-b - \lambda)\left(-\frac{fh + \gamma g}{h} - \lambda \right) - \frac{a\beta c}{d} \right) = 0$$

which yields directly that

$$\lambda_{1,2} = -d, -h \in \sigma(DF(0, \frac{c}{d}, 0, \frac{g}{h}))$$

are eigenvalues and the other eigenvalues are determined from

$$\lambda^2 + \left(\frac{h(b + f) + \gamma g}{h} \right) \lambda + \frac{bd(fh + \gamma g) - a\beta hc}{dh} = 0.$$

More precisely,

$$\lambda_{3,4} = -\frac{h(b+f)+\gamma g}{2h} \pm \frac{1}{2}\sqrt{\left(\frac{h(b+f)+\gamma g}{h}\right)^2 - 4\frac{bd(fh+\gamma g)-a\beta hc}{dh}}. \quad (9)$$

Thus, if in this equation (9) we assume (7) holds. Then one of the eigenvalues is real and strictly positive. Thus the nature of the first equilibrium in (6) is that it is a saddle point. In otherwise, all the real parts of the eigenvalues are negative. Thus the steady state $(0,\frac{c}{d},0,\frac{g}{h})$ is a sink.

In case of the second steady state solution in (6) of (1). As above we find $DF(V^*,X^*,Y^*,Z^*)$ and compute the determinant $|DF(V^*,X^*,Y^*,Z^*)-\lambda I|=0$. This yields,

$$(-d-\beta V^*-\lambda)(-h-\lambda)\left((-b-\lambda)(-f-\frac{\gamma g}{h}-\lambda)-a\beta X^*\right)=0.$$

Consequently, we find that

$$\lambda_{1,2} = -d-\beta V^*, -h \in \sigma(DF(V^*,X^*,Y^*,Z^*))$$

and that the other two eigenvalues are

$$\lambda_{3,4} = \frac{(fh+\gamma g+bh)}{2h} \pm \frac{\sqrt{(fh+\gamma g+bh)^2-4h\left(b(fh+\gamma g)-a\beta X^* h\right)}}{2h}$$

$$= \frac{(fh+\gamma g+bh)}{2h}, \quad \textit{with multiplicity } 2. \quad (10)$$

It follows from the signs of all the above real eigenvalues that we deduce the equilibrium point in question is a saddle.

□

From the Lemma 2.2 , we deduce the geometrical properties of the dynamical system generated by the equations (1) are the following. The linearized positive semiflow has a set of its trajectories approaching $(0,\frac{c}{d},0,\frac{g}{h})$ as a stable state limit point at infinite time, while other trajectories eminate and diverge away from it. Thanks to the assumption (7). There are some instabilities in the evolution processes of the interactions. In the other case, all the real parts of the eigenvalues are negative. Thus the steady state $(0,\frac{c}{d},0,\frac{g}{h})$ is a stable in positive time equilibruim. To comment on the second steady state solution in (6) besides being a saddle equilibruim. We have already noted that the type of constants yielded in X^* are of the death processes. In (10) the eigenvalue is of multiplicity two. This implies a stronger contribution to the instability of the system of equations possibly a movement from the saddle state equlibrium $(0,\frac{c}{d},0,\frac{g}{h})$ after effects of the condition (7) in the dynamical system.

Finally for this section, we recall from [1, 3, 2, 14, 16, 15, 8], that if $U=(V,X,Y,Z)$, $A=DF(V^*,X^*,Y^*,Z^*)$ where F is the vector field in (1) and if $\dot{U}=AU$ corresponds to the linearized process of the equations. Then the long time asymptotic dynamics of the linearized equations are equivalent to those of the orignal system of ordinary equations (1).

III. GLOBAL WELL POSEDNESS OF THE MODEL EQUATIONS

In view of understanding the model equations given in the above section, we initiate a primary study of the equations (1) in the case described by the first three of the system equations with $\gamma=0$, this situation describes the evolution process of the interactions between immune system and the HIV/and AIDS virus in the absence of a immune response function. Slightly generalizing on the system of equations, we shall drop the assumption that all constants are positive. We conclude the following theorem.

Theorem 3.1 *Consider the system of ordinary evolution equations (1) in the absence of an immune system response variable. Assume that initial conditions at* $t_0 \in IR^+$ *are given. Then,* (i), *if all constants are positive we have the system of equations (1) is globally well posed, moreover the virus population tends to a null solution.* (ii). *If* $b, f \in IR^- \setminus \{0\}$ *are strictly negative,* $a,d,\beta \in IR^+ \setminus \{0\}$ *strictly positive, or* $a,b \in IR^- \setminus \{0\}$, $\beta \in IR^+ \setminus \{0\}$. *Then the virus population blows up at infinite time with other variables assuming null solutions.*

Proof. It is clear that the system of equations (1) is reducible to a system of two equations. Namely, modeling the speed of infection by the HIV/AIDS virus against changes in the uninfected cells population. This coupled system of equations is the following

$$\begin{cases} \dfrac{dX}{dt} = c - dX - \beta XV \\ \dfrac{d^2V}{dt^2} + (b+f)\dfrac{dV}{dt} + fbV = a\beta XV. \end{cases} \quad (11)$$

It is easy to see by comparison that there exists families $\{(X_n(t), V_n(t)) : n \in IN\}$ of globally defined upper and lower solutions which converge to the solution,

$$X(t) = e^{-dt}X(0) + \int_0^t e^{-d(t-s)}(c - \beta V(s)X(s))ds$$

$$V(t) = C_1 e^{-bt} + C_2 e^{-ft} + \frac{a\beta}{f-b}\int_0^t e^{-b(t-s)}X(s)V(s)ds + \frac{a\beta}{b-f}\int_0^t e^{-f(t-s)}X(s)V(s)ds \tag{12}$$

if $b \neq f$, of (11). This solution is by direct integration. Similarly, for the case $b = f$ see (14) below. More precisely, for existence of sub and supersolutions, we notice in the system of equations (11) the following. If $\beta = 0$ then the system of equations is uncoupled. Thus by integration we get its $X(t)$ component is a supersolution to the same solution component in (12). Moreover, $0 \leq \liminf_{t\to-\infty} X(t) < \infty$. This boundedness estimate is trivial in the positive time. Since all variables in (11) are non negative. Thus null solutions are subsolutions.

It is clear that the $V(t)$ solution component for $\beta = 0$ in (11) is a subsolution. Also there exists supersolutions to the coupled equation in (11), by virtue, of the boundedness of the $X(t)$ solution component. Furthermore, this supersolution will always decay to its null solution at infinite time. In this way (i) is proved.

Now to prove (ii), we note that if $b, f \in IR^- \setminus \{0\}$ are strictly negative, $a, d, \beta \in IR^+ \setminus \{0\}$ strictly positive. Then, considering one case of either $f - b > 0$ or exclusively $b - f > 0$, we get that $V(t) \to \infty$ as $t \to \infty$. On the other hand, it follows from the solution variable $X(t)$ that

$$\begin{aligned} X(t) &\leq \tilde{X}(0) - \beta\int_0^t V(s))X(s)ds \\ &\leq \tilde{X}(0) + \beta\int_t^0 V(s)X(s)ds \\ &\leq \tilde{X}(0)exp\left(-\beta\int_0^t V(s)ds\right) \to 0 \end{aligned} \tag{13}$$

where $\tilde{X}(0) = X(0) + \frac{c}{d}$, as $t \to \infty$ since $V(t) \to \infty$, and (13) is due to the standard Gronwall's inequality [1, 7, 10, 20]. Thus at infinity time $X(t) = 0$.

Next consider the case $f = b$. Then, by integration we get solution components

$$X(t) = e^{-dt}X(0) + \int_0^t e^{-d(t-s)}(c - \beta V(s)X(s))ds,$$

$$V(t) = C_1 e^{-bt} + C_2 te^{-bt} + ab\beta\int_0^t e^{-b(t-s)}sX(s)V(s)ds + ab\beta\int_0^t e^{-b(t-s)}tX(s)V(s)ds. \tag{14}$$

It follows if $a, b \in IR^- \setminus \{0\}$, $\beta \in IR^+ \setminus \{0\}$ then $V(t) \to \infty$ as $t \to \infty$. Thus again arguing as in (13) we get that $X(t) \to 0$ as $t \to \infty$. This yields the theorem, so either the coupled system of equations (1) is well posed globally and has at most one solution, or the density variable $V(t)$ blows up at infinite of positive time, and the $X(t)$ density variable tends to its null solution. Hence this and the convention $0 \cdot \infty = 0$ yields the conclusion of the last alternatives for the system of equations (1), with which our proof is complete. □

The system of equations as given in (1) taking into account the immune functional response is very complicated and detailed and will appear as a sequel to this paper. In this case one can infer properties on the blow up of the solutions similar to the case above. Various properties on the long time asymptotic behaviour of the solutions will be proved by considering the effects of model parameters and initial conditions.

IV. NUMERICAL EXPERIMENTS

We performed various numerical experiments to demonstrate concordance of the numerical results with the theoretical results derived above. Table I-VII lists parameters used in our simulations. The data taken from [9] and [24] . Some values have been slightly perturbed to fit the description of our model. The data in the third column corresponds to modified values which we have made in order to illustrate the global well posedness of the problem.

The solution profiles in Figs 1&2 correspond to the case in Sec. III (Thm . 3.1) with $b = -2.4, f = -0.4$. Fig. 1 shows the blow up of the virus population $V(t)$ and Fig. 2 demonstrates that $X(t)$ tends to a null solution. Fig. 3 shows a plot of $V(t)$ & $X(t)$ in the case $a = -224.06, b = -2.4$. Once again $V(t)$ blows up and $X(t)$ tends to the null solution. Hence, the numerical results are in agreement with the theoretical result stated in Thm. 3.1 (ii).

TABLE I
DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	0.015
a	Rate of virus reproduction	224.06
b	Rate of death of viruses	2.4
c	Reproduction of uninfected cells	6
d	Death rate of uninfected cells	0.1
f	Death rate of infected cells	0.4
x	Initial x	100
y	Initial y	3
v	Initial v	4

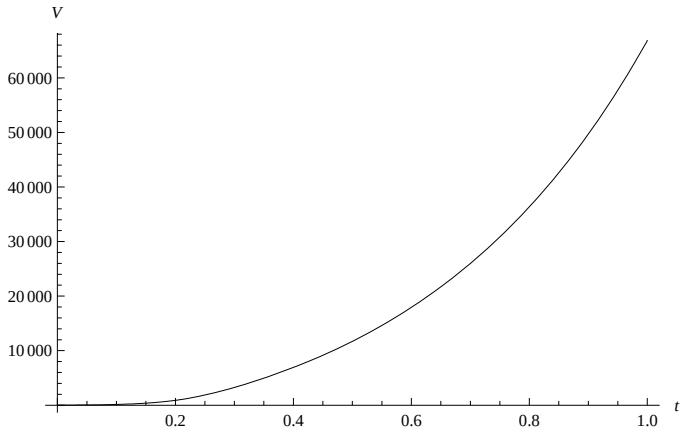

Figure 1: $V(t)$ solution

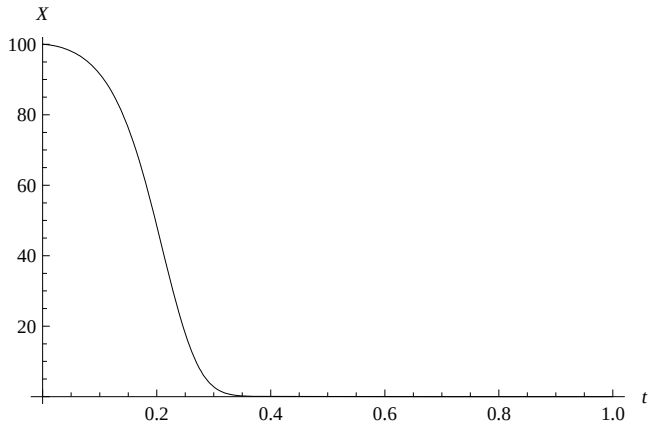

Figure 2: $X(t)$ solution

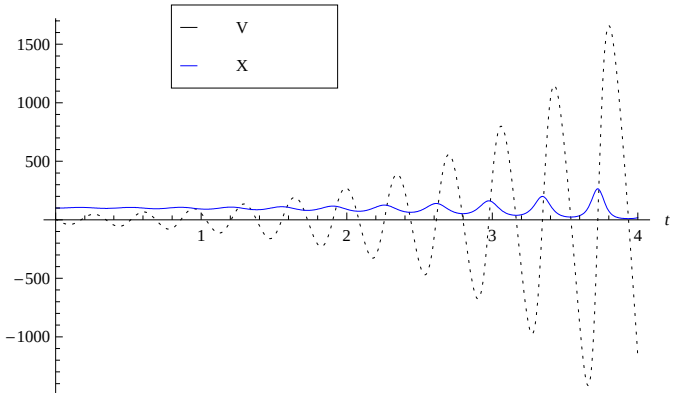

Figure 3: $V(t)$ & $X(t)$ solutions.

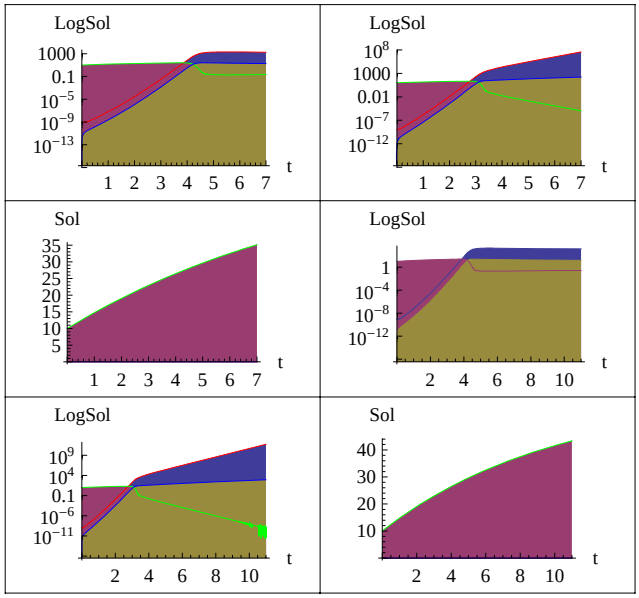

Figure 4: Snapshots of the solutions for different choices of the parameters given in Table I and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

TABLE II
DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	4.6E-4
a	Rate of virus reproduction	980
b	Rate of death of viruses	3
c	Reproduction of uninfected cells	1.3E-1
d	Death rate of uninfected cells	1.3E-2
f	Death rate of infected cells	4.E-1
x	Initial x	1.E+1
y	Initial y	0.00
v	Initial v	1.E-9

TABLE III
DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	3.6E-4
a	Rate of virus reproduction	1800
b	Rate of death of viruses	3
c	Reproduction of uninfected cells	2.E-1
d	Death rate of uninfected cells	2.E-2
f	Death rate of infected cells	8.E-1
x	Initial x	1.E+1
y	Initial y	0.00
v	Initial v	1.E-9

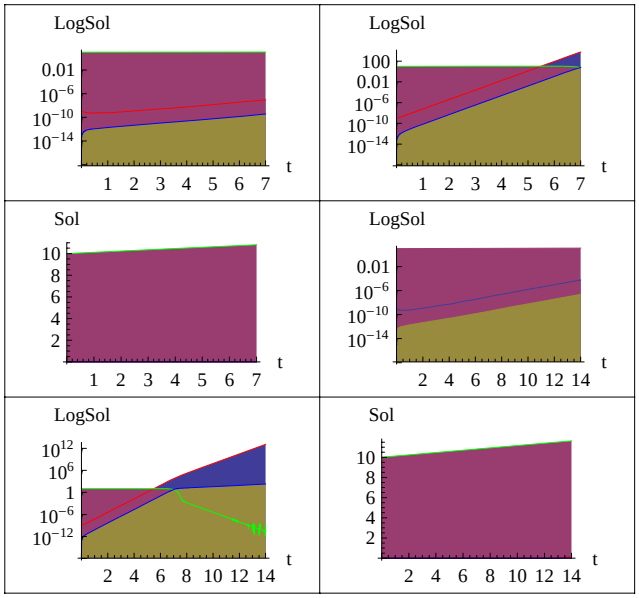

Figure 5: Snapshots of the solutions for different choices of the parameters given in Table II and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

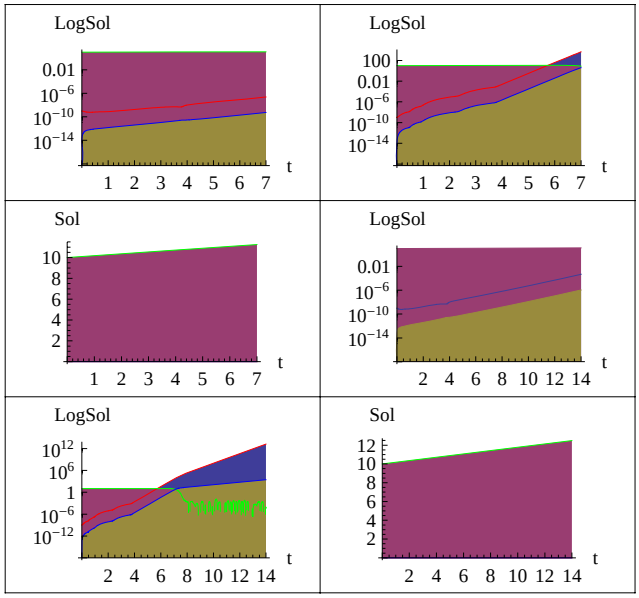

Figure 6: Snapshots of the solutions for different choices of the parameters given in Table III and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

TABLE IV
DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	6.3E-4
a	Rate of virus reproduction	870
b	Rate of death of viruses	3
c	Reproduction of uninfected cells	1.7E-1
d	Death rate of uninfected cells	1.7E-2
f	Death rate of infected cells	3.9.E-1
x	Initial x	1.E+1
y	Initial y	0.00
v	Initial v	1.E-9

TABLE V
DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	8.00E-4
a	Rate of virus reproduction	730
b	Rate of death of viruses	3
c	Reproduction of uninfected cells	1.7E-1
d	Death rate of uninfected cells	1.7E-2
f	Death rate of infected cells	3.1.E-1
x	Initial x	1.E+1
y	Initial y	0.00
v	Initial v	1.E-9

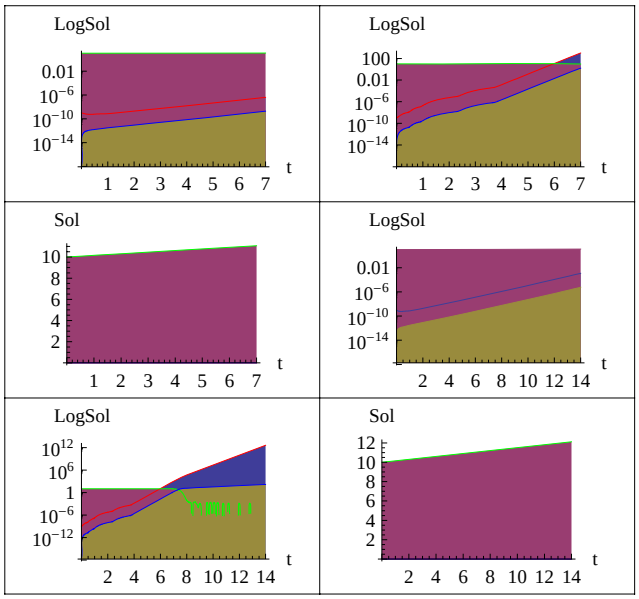

Figure 7: Snapshots of the solutions for different choices of the parameters given in Table IV and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

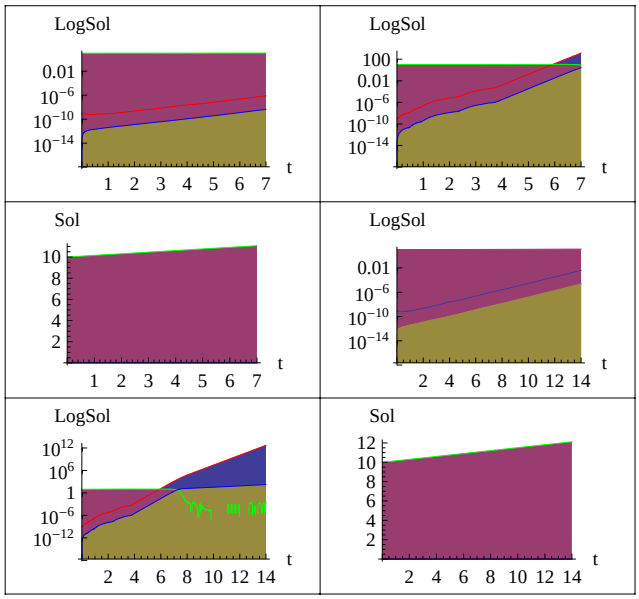

Figure 8: Snapshots of the solutions for different choices of the parameters given in Table V and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

TABLE V I

DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	6.6E-4
A	Rate of virus reproduction	830
B	Rate of death of viruses	3
C	Reproduction of uninfected cells	8.5E-2
D	Death rate of uninfected cells	8.5E-3
F	Death rate of infected cells	1.7.E-1
X	Initial x	1.E+1
Y	Initial y	0.00
V	Initial v	1.E-9

DEFINITION OF PARAMETERS

Parameter	Definition	Value
β	Rate at which uninfected cells becomes infected	2.5E-3
a	Rate of virus reproduction	110
b	Rate of death of viruses	3
c	Reproduction of uninfected cells	6.00E-2
d	Death rate of uninfected cells	6.00E-3
f	Death rate of infected cells	1.3E-1
x	Initial x	1.E+1
y	Initial y	0.00
v	Initial v	1.E-9

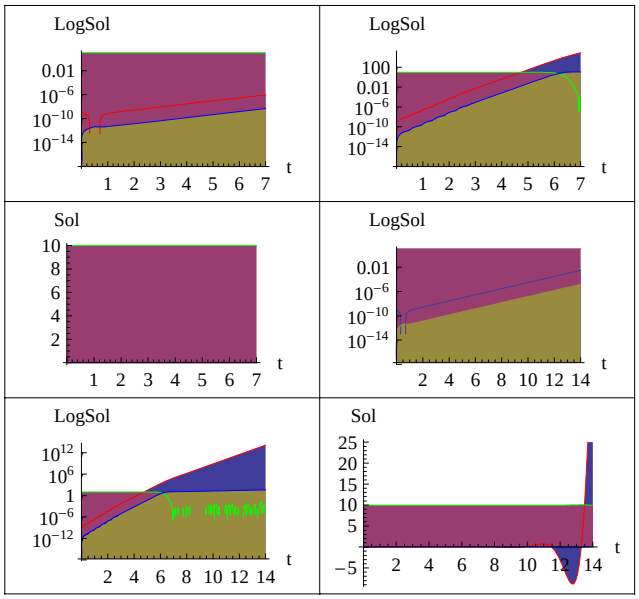

Figure 8: Snapshots of the solutions for different choices of the parameters given in Table VI and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

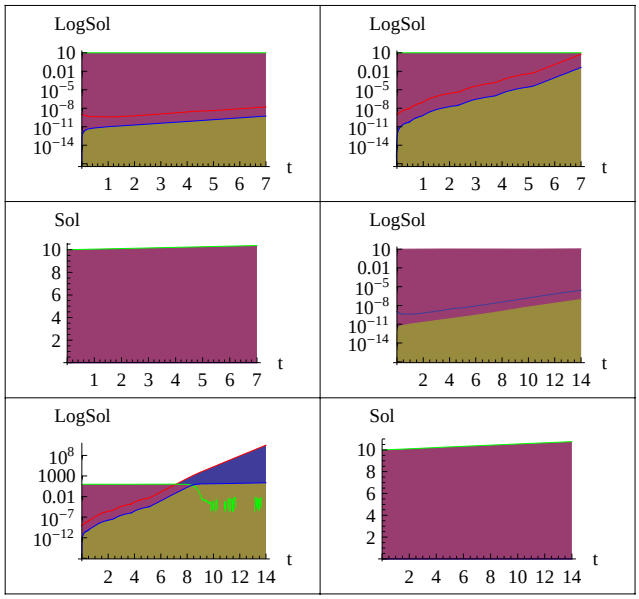

Figure 9: Snapshots of the solutions for different choices of the parameters given in Table VII and different values of time. The first shot corresponds to the case in Thm 3.1 (i). The second and third shots correspond to the cases in Thm 3.1 (ii) , respectively. Color Coding: Red (v(t)), Green (x(t)) and Blue (y(t)). The next three shots are repeats for different times. LogSol denotes the Log of the solution.

V. Conclusion

We have presented a simple HIV/AIDs finite dimensional model on interactions of the blood cells, the HIV/AIDs virus and the immune system. The primary objective of this paper is to give relevance of the well posedness of the finite dimensional evolution processes and to obtain a numerical validation of the theoretical results. Extension to the infinite dimensional case will be the focus of future work.

TABLE VII

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