Impulsive HIV Model Using the *B*-Transform

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Abstract

In this paper, a model on the spread of HIV infection is developed using the impulsive theoretic. The HIV virus is noted to be instable as it changes from one phase to another, for this reason, the idea of B-stability is used to investigate the behaviors of the HIV cells, immune cells and non-immune cells in the blood plasma. Also, a strategic pattern for combating the disease vis-à-vis trapping the HIV cells down is made by administration of drugs to control the growth of HIV cells and to boost the population of CD_4 cells in the blood. We exploited the B-transform in [8] to obtain the solution to the model.

Keywords and Phrases

HIV, AIDS, CD_4 cells, Heterosexual population model, *B*-stable, *B*-transform, immune cells, impulsive differential Equation. Drug Administration.

AMS Subject Classification: 92C, 45G, 34A

1. Introduction

Impulsive differential equations are equations that are characterized by rapid changes at fixed or non-fixed moments during evolution time. These processes are noted to affect many physical and biological processes. Examples are population, biological rhythm system, military science etc (see [1], [2], [7 - 8]).

Acquired immunodeficiency syndrome (AIDS) is syndrome caused by a virus called Human Immuno deficiency virus (HIV). Most researchers have held that HIV directly kills the immune cells called helped T cells or CD_4 cells, eventually exhausting an immune system that is practically making replacements. Some researchers held that HIV changes the signals that sends immune cells migrating through the body directing CD_4 cells away from the body where they are normally circulate and towards sites where they may be destroyed ([3]).

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AIDS is said to have happened when a HIV patient's CD_4 cells in blood dropped drastically and the organ that produces then are completely incapacitated and cannot produce them again. The period from stage of injection of a patient with HIV to the time of full brown is called **incubation period**. AIDS has long incubation period ranging from 6 - 10 years.

AIDS has remained man's greatest disease that calls for concerted effort from researchers across many disciplines to eradicate.

In this paper, we present an impulsive deterministic model for studying the behaviour of immune cells and HIV/AIDS cells in the presence of antiretrovial drugs. The study is relatively new at least from impulsive point view, even though, several statistical and mathematical model for contagious disease including HIV and AIDS diseases are present in the literature. Those models include simple epidemiological models to drug compartment models.

Studies on HIV/AIDS are now receiving tremendous attention in recent times. Statistical model for forecasting the growth of HIV/AIDS population patients in the world has been reported (See [5]).

The mathematical model for the transmission dynamics of HIV-1, sex and sexual activity has made (see [4]). More recently, a multi-dimensional birth and death process model for the transmission and spread of HIV infection and AIDS has been developed ([6]) a kind of a stochastic model.

It is worthy of note to say HIV/AIDS are complicated diseases which defiles cure of Late. The dynamics and instability nature of the HIV virus as it moves from one stage to another makes it difficult to find a permanent cure for the disease.

Scientists upheld that CD_4 cells undergo rapid depletion as the HIV virus advances from stage to stage. For this reason, any chemotherapeutic approach to solving this problem must have to take into consideration the instability effect of the dynamics of HIV virus.

Instability in this context refers to all the structural changes the HIV virus undergoes from infection stage to full brown stage.

In order to have a concrete model to reflect the rapid change that occur in the development stage of the HIV, from infection stage to final stage of total immune collapse (full brown stage); we have decided to use the impulsive theoretic to model the transmission dynamics of the disease.

The HIV model we will consider is a non-linear impulsive differential equations couple with impulsive drug model. The solution of the model is obtained using B-transform developed in our earlier paper ([8]). The B-stability is applied to the model. The novelty of the work seeks also to illustrate one of the many beauties of the B-transform some of which have been emphasised in many publications (See [8-10] references therein).

2. The Impulsive HIV Model

The HIV model which we shall referred to here as model in (Box A) is given by:

$$\frac{dN(t)}{dt} = k_1 N(t) - k_2 N^2(t) - k_3 \frac{\partial X(a,t)}{\partial t} + f_1(t,z(t),N(t)), t \neq t_k, k = 1,2,3... \quad (1)$$

$$\frac{dZ(t)}{dt} = k_4 Z(t) - k_5 Z^p(t) + k_6 N(t) + f_2(t,z(t),N(t)), t \neq t_k, k = 1,2,... \quad (2)$$

$$\frac{dX(t,a)}{dt} = (1 - n_2 - n_3) \frac{\partial X(t,a)}{\partial t} + k_7 \frac{\partial X(t,a)}{\partial a}, t \neq t_k, k = 1,2,... \quad (3)$$

$$\Delta N(t)|_{t=t_k} = I_1(N(t_k)) \quad (4)$$

$$\Delta Z(t)|_{t=t_k} = I_2(z(t_k)) \quad (5)$$

$$\Delta X(a,t)|_{t=t_k} = I_3(X(a,t)) \quad (6)$$

$$o < t_o < t_1 < t_2 < ... < t_k, \lim_{k \to \infty} t_k = +\infty$$

where

$$k_7 = a_o + \sum_{l=1}^{n} a_i f_i(t), f_i(t) = \int_o^t f_i(a,t) N(a) da$$

a_i is the phase change at stage i

$N(t)$ is the population, of the immune cells present in the blood plasma.

$Z(t)$ is the population of non-immune cells in the blood plasma at time t.

$X(t,a)$ is the population of HIV cells at time t and are at the stage a_i, $i = 1,2$.

n_2 is the probability of the HIV cells to attract the immune cells.

n_3 is the probability of the HIV cells to repel the immune cells towards the nodes and the lymphatic cells.

$f(t,a)$ is fucidity function at time t stage a.

$f_i(t,z(t),N(t))$, $i = 1,2$ are non-linear functions that accounts for the contribution coexistence of the population $z(t)$ and $N(t)$.

K_i, $i = 1,2,...,7$ are some relevant rate constants.

Remark 1

In developing the above model the following are taking into consideration:

* HIV directly kill the immune cell called helper T cells or CD_4 cells.

* HIV changes the signal that sends an immune cell migrating through the body directly a CD_4 cell away from the blood towards the site where it may be destroyed.

* The HIV advances in various stages, this makes the understanding of the phase transition difficult and hence impulsive effect on the two cells concerned.

3. Impulsive Drug Model (BOX B)

$$\dot{x}(t) = k_8 x(t) + L_1(t, N(t), z(t) + g_1(X(a,t)), t \neq t_k. \tag{7}$$

$$\dot{y}(t) = k_9 x(t) + k_{10} y(t) + L_2(t, N(t), z(t)) + g_2(X(a,t)), t \neq t_k. \tag{8}$$

$$\Delta x(t = t_k) = I_1(x(t_k)). \tag{9}$$

$$\Delta y(t = t_k) = I_2(y(t_k)). \tag{10}$$

$$o < t_o < t_1 < t_2 < \ldots < t_k \lim_{k->\infty} t_k = \infty$$

$x(t)$ and $y(t)$ denote the amount of drug in take at time t into the gastrointestinal track and apparent volume of distribution (eg blood, muscle, tissue etc) at time t, k_8 and k_9 are some rate constants.

The non-linear function $L_i(t, N(t), z(t))$, $i = 1,2$ give account of the contribution effect on the changes, on the normal and immune cells.

The disease change from stage to stage and, hence, a study on the stability dynamics of the HIV Virus becomes necessary. B-stability is concept is developed with boundedness property of some transfer function derived from the impulsive system and some sets. A given set plays the role of region in which the virus is trapped down and consequently eliminate by the HIV drug.

Definition 1

Consider the impulsive system

(IDDE) $\dot{x} = f(t,x), t \neq t_k$

$\Delta x = I(x(t_k))$

$$o < t_o < t_1 < t_2 < \ldots < t_k, \lim_{k->\infty} t_k = \infty$$

The zero solution $x(t)$=0 in (IDDE) is said to be B-stable relative to a set C in the complex plain. If for every $\varepsilon > o$ there is a constant r >0 and a set C such that $|x(q)| < r$ implies that

$$|x(t)| = |\int_C x(q) e^{tq}\, dq| < \varepsilon \text{ for } t \geq t_o.$$

x(q) is the B-transform of x(t).
C is the set containing all the singularities of x(q) if there is any singularity.

4. Application of *B*-Transform to the Model in Box B

In [8] we introduced the *B*-transform of function $x(t)$ with impulse at some fixed moments during the evolutionary process as

$$B_{n'}x(t) = x_c(q) + x_I(q)$$

where

$x_c(q)$ and $x_I(q)$ are the components of the *B*-transform and are define as

$$x_c(q) = L_c x(t) = \int_o^\infty e^{-t/q^{n'}} x(t)dq,\ t \neq t_k, k = 0,1,2,... \tag{11}$$

and

$$x_I(q) = L_I x(t) = \sum_{t_o < t_k < t} e^{-t_k/q^{n'}} I(x(t_k)) \tag{12}$$

n' = 0,1,2 are the order of the transform. For sake of simplicity, we will make use of n'=1. In fact, there is no loss of generality if n' is restricted to one. For some kind of problems, an order $n' > 1$ may be exploited. The advantage of taken n'=1 lies in the derivation of the inverse transform.

We now return to the inverse transform of x(t).

This can be obtained from $x_c(q)$ and $x_I(q)$
as

$$x_c(t) = \frac{1}{2\pi i}\int_{V-i\infty}^{V+i\infty} x_c(q) e^{sq}\, dq \tag{13}$$

$$x_I(t) = \sum_{t_o < t_k < t} \psi(t_k, q) I(x(t_k)) \tag{14}$$

$$\psi(t_k, q) = \int_{V-i\infty}^{V+i\infty} dq\, e^{-t_k/q^{n'} + sq} \tag{15}$$

Before we proceed to the application of the *B*-transform to the model, it is pertinent to make use of the following substitutions.

$$u(t) = \frac{1}{N(t)}, u(t) = \frac{1}{Z(t)}$$

$$g(t, Z(t), \frac{1}{u(t)} = f_1(t, Z(t), N(t)) - k_3 \frac{\partial X(t,a)}{\partial t} \tag{16}$$

$$h_1(t, V(t)) = k_6 N(t) + f_2(t, Z(t), N(t)) Z^{-p}(t) \tag{17}$$

$$h_2(q, u(q)) = \int_o^\infty u^2(t) L_1(t, Z(t), \frac{1}{u(t)}) e^{-t/q} dt \tag{18}$$

$$h_3(q, u(q)) = k_6 \int_o^\infty N(t) Z^{-p}(t) e^{-t/q} dt \tag{19}$$

$$+ \int_{o}^{\infty} f_{2}(t,\ z(t),\ N(t))\ z^{-p}(t)\ e^{-t/q}\, dt$$

Finally, we denote the following constants by $\alpha = \frac{n^{2}+n^{3}}{k_{1}}, \beta = \frac{\alpha k_{1}}{q}$

n_2 and n_3 are defined above in the model while K_1 is constant q is a fixed constant defined in the *B*-transform. If we now substitute the value of $v(t)$ and $u(t)$ into the model are the following equations:

$$\frac{du(t)}{dt} = k_{2}u(t) - k_{3} + u^{2}(t)\, L_{1}(t, z(t), \frac{1}{u(t)}, t \neq t_{k}, k = 1,2,3,... \tag{20}$$

$$\frac{dv(t)}{dt} = - k_{4}v(t) - k_{5} + k_{6}\, h_{1}(t, v(t), t \neq t_{k} \tag{21}$$

$$\frac{dX(t,a)}{dt} = (1 - n_{2} - n_{3})\frac{\partial X(t,a)}{\partial t} + k_{7}\frac{\partial X(t,a)}{\partial a}, t \neq t_{k} \tag{22}$$

$$\Delta N(t)|_{t=t_{k}} = I_{1}(N(t_{k})) \tag{23}$$

$$\Delta z(t)|_{t=t_{k}} = I_{2}(z(t_{k})) \tag{24}$$

$$\Delta x(a,t)|_{t=t_{k}} = I_{3}(x(a,t_{k})) \tag{25}$$

$$o < t_{o} < t_{1} < t_{2} < ... < t_{k}, \lim_{k->\infty} t_{k} = \infty$$

If we apply *B*-transform to eqs (20 - 25) we get,

$$u(q) = \frac{q}{1 - k_{1}q}u_{o} - \frac{k_{2}q^{2}}{1 - k_{1}q} + \frac{q}{1 - k_{1}q}h_{2}(q,u)$$

$$+ \sum_{t_{o}<t_{k}<t} e^{-t_{k}/q}\, I\left(\frac{1}{u(t_{k})}\right). \tag{26}$$

$$z_{c}(q) = \frac{1}{\left[\frac{q}{1 - k_{4}q}v_{o} - \frac{k_{5}q^{2}}{1 - k_{5}q} + \frac{q}{1 - k_{4}q}h_{2}\left(q, \frac{1}{v(q)}\right)\right]^{\frac{1}{p}}} \tag{27}$$

where

$$z_{1}(q) = \sum_{t_{o}<t_{k}<t} e^{t_{k}/q}\, I\left(\frac{1}{v(t_{k})}\right) \tag{28}$$

$$Z(q) = Z_{C}(q) + Z_{1}(q)$$

Similarly, for eq(13), we have

$$\frac{\partial X_c(q,a)}{\partial a} = \frac{\alpha k_1}{q} X_c(q,a) + \alpha X_o \tag{29}$$

$$\boldsymbol{X_1(q,a) = \sum_{t_o<t_k<t} e^{-t_k/q} I(X(q,t_k))} \tag{30}$$

$$X_o = X(q,o)$$

5. Main Results

If we solve eq (29) for fixed *q*, we get

$$X(q,a) = \frac{\left(\frac{n_2+n_3}{k_1} + \left(\frac{\alpha k_1}{q} + \frac{n_2+n_3}{k_1}\right) e^{\frac{-a\alpha k_1}{q}}\right)}{\frac{\alpha k_1}{q}}$$

$$+ \sum_{t_o<t_k<t} e^{-t_k/q} I(X(q,t_k)) \tag{31}$$

Now that *B*-transform of the cells are obtained, it is pertinent to investigate the *B*-stability of the model.

Analysis of the Models in Box A and B

$$\begin{aligned} v(q) &\geq \frac{q}{1-k_1 q} u_o + \frac{q}{1-k_1 q} h(q,u) \\ N(q) &\leq \frac{1}{u(q)} = \frac{1}{\frac{q}{1-k_1 q} u_o + \frac{q}{1-k_1 q} h(q,u)} \\ &\leq \frac{1-k_1 q}{q u_o} = \frac{1}{q u_o} - \frac{k}{u_o} \\ &= C + \frac{k_6}{q u_o} - \frac{kk_6}{u_o} \\ &\to c + \frac{kk_6}{u_o} \, as \; q \to \infty. \end{aligned} \tag{32}$$

Let r_i, $i = 1,2,3$ be constant

$$|N(q)| \leq \left(\frac{1-k_1 q}{q}\right)^p u_o^{\frac{1}{p}} [1]^{-1} \tag{34}$$

$$|Z(q)| \leq \left(\frac{1 - k_1 q}{q} \right)^p \frac{u_o^{\frac{1}{p}}}{\left[1 + \frac{p(p-1)}{2u_o^2} h^2(q,a) \right]} < r_2 \tag{35}$$

Then

$$\left| \int_C N(q) e^{tq} \, dq \right| \leq \int_{C_1} |N(q)| e^{tq} \, dq$$

$$< r_2 \int_{C_1} e^{tq} \, dq$$

$$|N(t)| \leq \frac{r_3}{2\pi} \left[\frac{e^{tq}}{q} (e^{2\pi} - 1) + \frac{1}{2\pi} e^{2\pi(k_1 t - 1)} + \frac{e^{k_1 t - 1}}{t} + 1 \right] \tag{36}$$

If

$$h(q,a) > \sqrt{\frac{2u_o}{p(p-1)} \left(\frac{u_o^{\frac{1}{p}}}{r_2} - 1 \right)}$$

Then

$$|Z(t)| < r_2$$

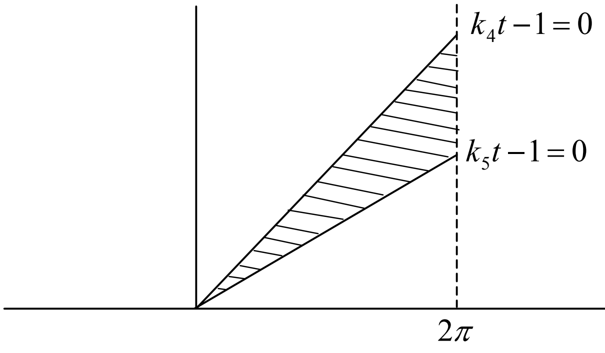

Fig.1: Integration along the Triangular Domain C_1

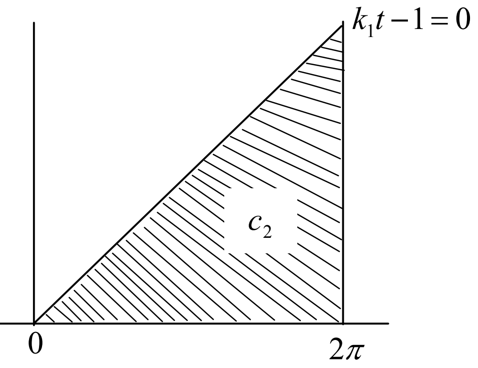

Fig.2: Integration along the Triangular Domain C_1

$$|X(q,a)| \leq \left[\frac{n_2 + n_3}{\alpha k_1^2} |q| + \left(1 - \frac{n_2 + n_3}{\alpha k_1} |q|\right) + e^{-\frac{a\alpha k_1}{q}} \right] |X_o| < r_3. \tag{37}$$

We make use of the following to investigate that stability of the model in the Box A

$$\begin{aligned} |Z(t)| &\leq \frac{1}{2\pi}\int_{c_1} e^{tq}\,|z(q)|\,dq \\ &\leq \frac{r_2}{2\pi}\int_{c} e^{tq}\,dq \\ &= \frac{r}{2\pi t}(k_4+k_5)(e^{2\pi q}-1) < \varepsilon \end{aligned} \tag{38}$$

Therefore

$$\left(\frac{2q\pi - 1}{t}\right) < \frac{\varepsilon_1}{r(k_4+k_5)}$$

Similarly, we have

$$\left| X(q,a)\right| \leq \frac{r_1}{2\pi t}(e^{2\pi t}-1) < \varepsilon_3$$

The result obtained above suggested the model is *B*-stable, hence, we can find a set *C* such the HIV cells can be trapped down.

We now study the *B*-transform of the drugs model in the box B as follows:
We now apply the *B*-transform for the drugs model as follows:

$$\begin{aligned} x(q) = \frac{q}{1-k_6 q}&[-x_o + L_1(q,Z(t),N(q)) + g_1(X(q,a)) \\ &+ \sum_{t_o<t_k<t} e^{-t_k/q} I_1(x(t_k))] \end{aligned} \tag{40}$$

$$\begin{aligned} y(q) = \frac{q}{1-k_8 q}&[y_o - k_4 x_c(q) - k_5 L_2(q,Z(q),N(q)) \\ &+ g_2(x(q,a)) + \sum_{t_o<t_k<t} e^{-t_k\alpha/q} I_2(y(t_k))] \end{aligned} \tag{41}$$

From equations (40) and (41) we have

$$\begin{aligned} L_1(q,N(q),Z(q)) = &\left(\frac{1-k_6 q}{q}\right) x(q) - x_o - \sum_{t_o<t_k<t} e^{-t_k/q} I_1(x(t_k)) \\ &+ g_1(X(q,a)) \end{aligned} \tag{42}$$

$$\begin{aligned} L_2(q,N(q),Z(q)) = &\left(\frac{1-k_6 q}{q}\right) y(q) - x(q) - \frac{1}{k_4} y_o \\ &- \frac{1}{k_4}\sum e^{-t_k/q} I_2(y(t_k)) \end{aligned} \tag{43}$$

$$+ g_2(x(q,a))$$

where nature $L_i(q, N(q), Z(q)),$ can be represented in various forms, following form are hereto refer to as case I and II.

Case I

$$g_i(q, N(q), Z(q)) = \alpha_i N(q) + \beta_i Z(q), i = 1,2 \quad \alpha_1 \text{ and } \beta_i$$

are constant

Case II

$$L_1(q, N(q), Z(q) = \alpha_1 N^2(q) - \alpha_1 N(q) - f_1(Z(q)) = - A_1 \tag{44}$$

$$L_2(q, N(q), Z(q)) = \beta_2 Z^2(q) - \beta_2 Z(q) - f_2(Z(q)) = - A_2 \tag{45}$$

$f_i(Z(q))$, $i = 1,2$. are functions that depend on from Case I.

$$\alpha_1 N(q) + \beta_1 Z(q) = \left(\frac{1 - k_6 q}{q}\right) x(q) + x_o - \sum_{t_o < t_k < t} e^{-t_k/q} I_1(x(t_k)) + g_1(x(q,a)) \tag{46}$$

$$\alpha_2 N(q) + \beta_2 Z(q) = \left(\frac{1 - k_8 q}{k_4 q}\right) y(q) - x(q) = \frac{1}{k_4} y_o + + g_2(x(q,a)) - \frac{1}{k_4} \sum_{t_o < t_k < t} e^{-t_k/q} I_2(y(t_k)). \tag{47}$$

In [7], the idea of exclusion and marginalization was elucidated, this can be extended to the models in Box A & B and the overall effect on $N(t)$ and $Z(t)$ be studied.

We consider the following situation if $N(t)$ exclusion $Z(t)$ that the drug is so effective that sero-negative situation of HIV patients, mathematically speaking, this is the situation wherein $Z(t) < N(t)$.

Therefore

$$\begin{aligned} N(q) = \frac{1}{\alpha_1 + \alpha_2} [(\frac{1 - k_6 q}{q}) x(q) + (\frac{1 - k_8 q}{k_4 q}) y(q) + \\ + g_1(x(q,a) + g_2(x,(q,a)) + \\ - \sum_{t_o < t_k < t} e^{-t_k/q} I_1(x(t_k)) + x_o - \frac{1}{k_4} y_o + \\ - \sum_{t_o < t_k < t} e^{-t_k/q} I_1(x(t_k)) - \frac{1}{k_4} I_2(y(t_k))] \end{aligned} \tag{48}$$

From case II, we have the following equations synonymous to the characteristic equation.

$$N^{\pm}(q) = \frac{\alpha_1 \pm \sqrt{\alpha_1^2 + 4\,\alpha_2\, f_1(Z(q)) - 4\,A_1}}{-2\,\alpha_1} \tag{49}$$

$$Z^{\pm}(q) = \frac{\beta_1 \pm \sqrt{\beta_1^2 + 4\,\beta_2\, f_2(Z(q)) - 4\,A_2}}{-2\,\beta_2} \tag{50}$$

Thus

$$N^{-}(q) = \frac{\alpha_1 - \sqrt{\alpha_1^2 + 4\,\alpha_2\, f_1(Z(q)) - 4\,A_1}}{-2\,\alpha_2} \tag{51}$$

$$Z^{+}(q) = \frac{\beta_1 + \sqrt{\beta_1^2 + 4\,\beta_2\, f_2(Z(q)) - 4\,A_2}}{-2\,\beta_2} \tag{52}$$

$$Z^{-}(q) = \frac{\beta_1 - \sqrt{\beta_1^2 + 4\,\beta_2\, f_2(Z(q)) - 4\,A_2}}{-2\,\beta_2} \tag{53}$$

$$Z(q) = C_1 Z^{-}(q) + C_2 Z^{+}(q) \tag{54}$$

$$N(q) = C_3 N^{-}(q) + C_4 N^{+}(q) \tag{55}$$

From above equation $Z(t)$ and $N(t)$ can be determined. Now define:

$$D_1 = \beta_1^2 + 4\,\beta_2\, f_2(z(q) - 4\,A_2$$

if $D_1 < 0$ then

$$\beta_1^2 + 4\,\beta_2\, f_2(z(q)) - 4\,A_2 = 4\,\pi^2 i^2\, \omega(q), i^2 : -1.$$

It follows that

$$Z^{-}(t) = \frac{\beta_1}{-4\pi\,\beta_2^1} \frac{e^{qt}}{t}\Big|_{C_1} + \frac{2\pi i}{2\,\beta_2}\,\omega_1(t). \tag{56}$$

The above equation can be utilize to monitor the effect of the drugs on the population $Z(t)$ of the patient where f_2 $(z(q))$ grows periodically with $Z(t)$.

The effective use of the drug depends on how $f_2(z(t))$ is boost. A very effective drug, will tend to increase the population of $Z(t)$ and at the same decrease the population of $X(t,a)$. Since from the derivation, there is no explicit relation that exists between $Z(t)$ and $X(t,a)$ from the model. Therefore, the drug to be developed must be able to tract down the $X(t,a)$ cells using appropriate set C. Because $X(t,a)$ is dynamically changing from phase to phase, the molecules of the drug should be able to attach itself to the cell of the HIV virus and gradually destroy it or at should destroy it at a very rapid rate.

Applying the inverse transform to eqs(54 & 55) we have

$$Z(t) = \frac{\beta_1}{-4\beta_2 \pi i} \cdot \frac{e^{qt}}{t}\Big|_C - \frac{2\pi i}{2\beta_2}[(\beta_1^2 - 4A_2)\frac{e^{qt}}{t}\Big|_C \tag{56b}$$

$$+ 4\beta_2 f_2(z(t)) + \frac{2\pi i}{-2\beta_2}\omega_1(t)$$

If $D_2 > 0$ then

$$D_2 = 4\pi^2 \omega^2(q)$$

$$Z^{\pm}(q) = \frac{\beta_1 \pm 2\pi\,\omega_2(q)}{-2\beta_2} = \frac{\beta_1}{-2\beta_2} \pm \frac{\pi}{\beta_2}\omega_2(q)$$

Therefore

$$Z^{\pm}(t) = \frac{1}{2\pi i}\int_C \left(\frac{\beta_1}{2\beta_2} + \frac{\pi}{\beta_2}\omega_2(q)\right) e^{qt}\, dq$$

$$= \frac{\beta_1}{4\pi\beta_2 i}\int_C e^{qt}\, dq + \frac{\pi}{4\pi\beta i}\omega_2(t)$$

The solution to the model is therefore

$$Z(t) = C_1 Z^-(t) + C_2 Z^+(t)$$

C_1 and C_2 are constants.

We are now in the position to study the behaviour of $N(t)$ and $Z(t)$ and $X(a,t)$ using the *B*-stability concepts relative to some sets. A simple application of study of the drug effect on $Z(t)$ can be obtained by setting the first and second terms in eq (56) to zero.

Therefore, we have

$$Z(t) = 4\beta_2 f_2(z(t)) - \frac{2\pi i}{2\beta_2}\omega_1(t) \tag{57}$$

We can monitor the growth $Z(t)$ from eq. (57) once $f_2(Z(t))$ and $w(t)$ are known. $f_2(Z(t))$ can roughly be highly be inferred from experimental data.

7. $P_Z(t)$ and $P_N(t)$ Functions

We define two vital functions as

$$P_z(t) = \frac{Z(t)}{X(t,a)}$$

$$P_N(t) = \frac{N(t)}{X(t,a)}$$

We compute the following ratio

$$\frac{Z(q)}{X(q,a)} = \frac{\left[\frac{(n_2+n_3)q}{\alpha k_1^2} + (1 - \frac{(n_2+n_3)q}{\alpha k_1^2})e^{-\frac{a\alpha k_1}{q}}\right]x_o + \sum_{t_o<t_k<t} e^{-t_k/q} I_(x(q,a))}{\left[\frac{q}{1-k_4 q}v_o - \frac{k_5 q}{1-k_5 q} + \frac{q}{1-k_4 q}h_2\right]^{\frac{1}{p}}} +$$

$$+\sum e^{-t_k/q} I\left(\frac{1}{V(t_k)}\right)\left[\frac{(n_2+n_3)q}{\alpha k_1^2} + (1 - \frac{(n_2+n_3)}{\alpha k_1^2}q)e^{-\frac{a\alpha k_1}{q}}\right]x_o +$$

$$+ \sum_{t_o<t_k<t} e^{-t_k/q} I(X(q,t_k))]$$

Similarly

$$\frac{N(q)}{X(q,a)} = \frac{\left(\frac{1-k_1 q}{q}\right)^p U^{\frac{1}{p}}\left[1 + \frac{p(p-1}{2u_o^2}h^2(q,a)\right]^{-1}}{\left[\frac{(n_2+n_3)q}{\alpha k_1^2} + \left(1 - \frac{(n_2+n_3)}{\alpha k_1^2}q\right)e^{\frac{-a\alpha k_1}{q}}\right]X_o}$$

$$+ \sum_{t_o<t_k<t} e^{-t_k/q} I(X(q,t_k))\}$$

If follow that

$$\frac{Z(t)}{X(t,a)} = \frac{1}{2\pi i}\int_c \frac{Z(q)}{X(q,a)} e^{qt}\, dq = P_z(t)$$

$$\frac{N(t)}{X(t,a)} = \frac{1}{2\pi i}\int_c \frac{N(q)}{X(q,a)} e^{qt}\, dq = P_N(t)$$

For any patient with HIV/AIDS infection, the functions $P_Z(t)$ and $P_N(t)$ must be closely monitored with time. If P_Z is very smaller than one (i.e., $P_z(t) << 1$) then the population of HIV cells is increasing at an alarming proportion which does not augur well for the patients infected with the disease. The drug that will cure or alleviate the disease must have the tendency to reverse the trend (i.e., $P_{.}(t)>>1$) and the property that ($P_N(t)<<1$). In physical term, this will involve developing a drug whose molecules will attracts that of HIV virus get attached to it and destroy it at very fast rate.

The $P_N(t)$ function must also be strictly be monitored so as to keep the population of non-immune cells at an appreciable level so as not to create another physiological problem.

The ratio $P_{.}(t)$ to $P_N(t)$ i.e., ($P_{.}(t)/P_N(t)$ can also offer available information about the chemotherapeutic effect of the HIV/AIDS drugs. At certain threshold value of this ratio, a full brown stage is attained this to subject to clinical enquiries.

If $P_{.}(t)>\frac{1}{4}$ or there about; there is a strong indication that the patients with HIV will progress into full brown AIDS as professor David Ho predicted (see [3]).

8. Conclusion

HIV and AIDS are man's greatest disease that appears to have defiled cure. Many research works are going worldwide to develop drugs whether natural or synthetic to fight the virus.

The present paper gives a strategic pattern for combating the disease vis-a-vis to trap the HIV down and the administration of drugs to control the growth of HIV cells; and to boost the population of CT_4 the Lymphocyte cells.

The instability nature of HIV disease as it changes from phase to phase makes the combat of the disease insurmountable for quite some time. Researchers are hereby encourage to see how impulsive theoretic can be further be explored to study the mode of transmission of the disease, epidemiological impact and drug administration of the disease.

The theory of impulsive system with variable structure and variable times impulsive process will offer a lot of un-match able opportunity to study the dynamical nature of many real life process for which the HIV and AIDS problems are an examples.

Finally, we like to reiterate that the B-transform is a new method developed for impulsive systems, more applications are expected to be explored for the method.

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