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MATHEMATICAL ANALYSIS OF THE GLOBAL DYNAMICS MODEL FOR HIV INFECTION OF CD4$^+$ T CELLS WITH TREATMENT USING ADOMIAN DECOMPOSITION APPROACH

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ABSTRACT. A compartmental epidemic model proposed by Liancheng and Micheal [1] was investigated. A nonlinear incidence rate and treatment were taken into consideration. The bifurcation method introduced in [2] was being used to perform a bifurcation study, which is predicated on the use of the center manifold theory. The forward bifurcation was discovered. The Adomian Decomposition Method (ADM) was also used to approximate the solution of the problem's nonlinear system of differential equations. The computations were carried out using Maple, and the graphical results are given.

Keywords and phrases: HIV infection, Basic reproduction number, forward bifurcation. Adomian decomposition method
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1. INTRODUCTION

Human immunodeficiency virus (HIV) transmission is most commonly transmitted through sexual contact in society. It can also be spread through transfusions of HIV-infected blood or by injecting drugs into the bloodstream with a contaminated needle or syringe; however, this does not necessarily indicate that an individual has AIDS. HIV enters the bloodstream via immune system cells, especially T cells, which are white blood cells. These cells control a variety of disease-fighting mechanisms, with CD4+ T cells, a type of specialized helper T cell, being extremely prone to HIV permeability.
While HIV infects CD4+ T cells, it seizes the cell's genetic tools and uses them to create new HIV virus. After that, the newly formed HIV virus leaves the cell, destroying the CD4+ T cells. The loss of

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CD4+ T cells jeopardizes wellbeing as these are the cells aid in the response of several invading species with different types of immune cells.

When the number of reproductions is less than or equal to one, the HIV infection is clear from the individual; if the reproduction size is great than one, the HIV infection lingers.[19]. In addition, several reliable algorithms had been evolving, as well as an approximation solution of the differential system modeling of HIV infection CD4+ T cells was achieved using an extension of the standard variation iteration method (VIM), also known as the multi-state variational iteration method (MSVIM), this was compared to the fourth-order Runge-Kutta Method (RKV, Method), as well as a series solution. We do not plan to provide a fresh viewpoint or a thorough overview of these subjects, as this has already been done in a number of recent publications [1, 6, 19, 18, 20]. Instead, our goal is to use the existing numerical technique: Adomian decomposition approach to describe some relevant problems and explore in HIV care that is a major focus of this research, with the aim that this will help as an entry point for those interested in participating in the study.

In the worst-affected countries, HIV has decimated the men and women in their twenties and thirties, who are the labor force's strength. The majority of them die during the prime over their fertile years. Furthermore, the disease has overstretched medical systems, doubled the population of orphans, and lowered or dramatically lowered life expectancy rates, and no current medical procedure can fully eliminate the disease once it has contaminated human cells..

However, using a numerical approach, this study presents a HIV infection of CD4+ T cells mathematical model with treatment that minimizes the virus to a low or insignificant level of the virus for an individual infected with the virus to live a healthy life.

2. MATHEMATICAL MODEL

Based on compartmental deterministic model proposed in [1], we developed a model by assuming logistic growth of a cell population and treatment rate of the cells given by:

$$\frac{dT}{dt} = s - \alpha T + rT\left(1 - \frac{T + T^*}{T_{\max}}\right) - \frac{kVT}{1 + \alpha_1 V} + \rho T^*, \tag{2.1}$$

$$\frac{dT^*}{dt} = \frac{kVT}{1 + \alpha_1 V} - \beta T^* - \rho T^*, \tag{2.2}$$

$$\frac{dV}{dt} = N\beta T^* - \gamma V, \tag{2.3}$$

where T, T^* and V reflect the extent of the uninfected compartmental intensity CD_4^+TCells, infected CD_4^+T and Viruses that are free. s denotes the supply rate of $CD4^+$T cells from the thymus and bone marrow precursors, α which is the uninfected mortality rate $CD4^+$T cells, whereas k is the percentage rate of $CD4^+$T cells becomes infected with a virus that is available for free. T_{max} is the highest possible $CD4^+$T population of cells. $\rho$is the cells treatment rate. N represents the Viruses generated for free by lysing a $CD4^+$T cells, and β is the death proportion of infected $CD4^+$T cells. $\alpha_1$is saturated term. γ is the death rate of free viruses. The rate of change is increasing of the $CD4^+$T cell population is denoted by r. Qualitative analysis of the system described by Equations (2.1) - (2.3) reveals that long-term behavior can classify as either endemic or extinct. Whenever the illness is no longer present ordinarily, the response reaches a disease-free equilibrium of the structure asymptotically. The rate of change is increasing
ε_0 of the form,

$$\varepsilon_0 = \left(\frac{(r-\alpha)T_{\max} + \sqrt{T_{\max}\left[(r-\alpha)^2 T_{\max} + 4rs\right]}}{2r}, 0, 0 \right).$$

The threshold which always helps determine the stability of this equilibrium is the R_0, i.e. whether or not the infected cells will spread through the population

$$R_0 = \frac{kT_0 N\beta}{(\beta+\rho)\,\gamma}.$$

An endemic equilibrium of the form given exists if the disease-free equilibrium is unstable;

$$\varepsilon_1 = \left(\frac{(\beta+\rho)(\gamma+\alpha_1 N\beta\hat{T}^*)}{kN\beta}, \frac{-b \pm \sqrt{b^2-4ac}}{2a}, \frac{N\beta\hat{T}^*}{\gamma} \right). \tag{2.4}$$

where

$$a = \left(\frac{r\beta^2\alpha_1^2}{k^2 T_{\max}} + \frac{2r\beta\rho\alpha_1^2}{k^2 T_{\max}} + \frac{r\rho^2\alpha_1^2}{k^2 T_{\max}} + \frac{r\beta\alpha_1}{T_{\max}k} + \frac{r\rho\alpha_1^2}{T_{\max}k} \right),$$

$$b = \frac{r}{T_{\max}} \left(\left[\frac{\gamma^2}{k^2N^2} + \frac{2\rho\gamma^2}{k^2N^2\beta} + \frac{\rho\gamma^2}{k^2N^2\beta^2} \right] - \frac{r}{T_{\max}} \left(\frac{2\beta\gamma\alpha_1}{k^2N} + \frac{4\gamma\rho\alpha_1}{k^2N} + \frac{2\gamma\rho^2\alpha_1}{k^2N\beta} \right) \right.$$

$$\left. + \frac{\beta\alpha\alpha_1}{k} + \frac{\rho\alpha\alpha_1}{k} + \frac{r\beta\alpha_1}{k} + \frac{r\rho\alpha_1}{k} + \frac{r\gamma}{kT_{\max}kN} + \frac{r\rho\gamma}{kN\beta T_{\max}} - \beta \right),$$

$$c = \frac{r\gamma}{kN} + \frac{\alpha\rho\gamma}{kN} - \frac{\alpha\gamma}{kN} - \frac{\alpha\rho\gamma}{kN\beta} + \frac{r\gamma^2}{(kN)^2 T_{\max}} + \frac{2r\beta\rho\gamma}{(kN\beta)^2 T_{\max}} - \frac{2r\rho^2\gamma^2}{(kN\beta)^2 T_{\max}} - s.$$

Obviously Equation (2.4) will only exist provided $R_0 > 1$.

2.1. Positivity of solutions

Theorem 2.1: Let the initial facts $T(0) > 0$,$T^*(0) > 0$,$V(0) > 0$, the ideas follow after that $T(t)$,$T^*(t)$,$V(t)$,of HIV free model (2.1) are positive for all $t \geq 0$

Proof: It is clear from equation (2.1) that

$$\frac{dT}{dt} \geq -\left(\alpha + \frac{rT^*}{T_{\max}} + \frac{kV}{1+\alpha_1 V}\right) T, \tag{2.5}$$

$$\frac{dT}{T} \geq -\left(\alpha + \frac{rT^*}{T_{\max}} + \frac{kV}{1+\alpha_1 V}\right) dt. \tag{2.6}$$

Integrating both sides of (3.2)

$$InT \geq -\alpha t - \int_0^t \left[\frac{r}{T_{\max}} T^*(\tau) - kV(\tau)\right] d\tau + C_1. \tag{2.7}.$$

Take exponential of both sides, (2.7) gives

$$T(t) \geq C_2 \exp\left(-\alpha t - \int_0^t \left[\frac{r}{T_{\max}} T^*(\tau) - \frac{kV(\tau)}{1+\alpha_1 V(\tau)}\right] d\tau\right). \tag{2.8}$$

Applying the initial condition.
Hence

$$T(t) \geq T(0) \exp\left(-\alpha t - \int_0^t \left[\frac{r}{T_{\max}} T^*(\tau) - \frac{kV(\tau)}{1+\alpha_1 V(\tau)}\right] d\tau\right). \tag{2.9}$$

for all $t > 0$.
It is clear from equation (2.2) that

$$\frac{dT^*}{dt} \geq -(\beta + \rho) T^*. \tag{2.10}$$

So that,

$$T^*(t) \geq T^*(0) \exp\left[-\int_0^t (\beta + \rho)\, dz\right] > 0, \tag{2.11}$$

For all $t > 0$
It is clear from equation (2.3) that

$$\frac{dV}{dt} \geq -\gamma V. \tag{2.12}$$

So that,

$$V(t) \geq V(0) \exp\left(-\int_0^t \gamma dz\right) > 0, \tag{2.13}$$

for all $t > 0$

2.2. Bifurcation Analysis

Numerous traditional disease model thresholds, based on the fundamental reproductive numbers If indeed the reproductive number is one or less, in the feasible region, the resulting model only has the disease-free equilibrium, which would be globally stable. Whenever the most basic reproductive numbers are less than or equal to one, the model has a one-of-a-kind, globally stable endemic equilibrium in addition to the unstable disease-free equilibrium. When the disease's reproductive number reaches less than or equal to one, it dies. But if the number is greater than one, the disease is still present in the population**.** However, there is mounting evidence that perhaps the fundamental reproductive number alone is insufficient to completely ascertain the global dynamics of disease transmission. Even in simple epidemiology models, backward bifurcation with many endemic equilibria and or Hopf bifurcation yielding periodic solutions can occur [13,16,8,9,7,17,11], and the it's also possible that a Bogdanov-Takens singularity could occur [14,15]. In a model with only forward bifurcation, the sum (volume or fraction) of infective individuals is low when the reproductive number is greater than but similar to one. In a model of backward bifurcation, however, when the reproductive number is less than but similar to one, the model has two endemic equilibria, one of which is a saddle and the other of which is locally. While there is a special endemic equilibrium when the reproductive number is less than or equal to one, when the reproductive number is greater than but identical to one, the sum (number or fraction) of the infective individual is higher, according to the forwarding bifurcation model.

There are normally two thresholds in a scheme of backward bifurcation: $R_0 = R_c\,(0 < R_c < 1)$ and $R_0 = 1$at $R_0 = R_c$, a saddle-node bifurcation exists, and at $R_0 = 1$ has a bifurcation that is in the opposite direction. If the model has an endemic equilibrium, it's special, that is $R_0 \geq 1 \text{or} R_c = R_0 < 1$, we have an endemic equilibrium if there are two endemics, that is $R_c < R_0 < 1$, and if there is no endemic equilibrium, $R_c < R_0$,

Apply Theorem 2.2(See Appendix) to demonstrate the system (2.1)-(2.3) if there is a backward or forward bifurcation:

$$k = k^* = \frac{(\beta + \rho)\,\gamma}{T_0 N \beta}, \tag{2.14}$$

$$J(\varepsilon_0, k^*) = \begin{bmatrix} -\alpha + r - \frac{2rT_0}{T_{\max}} & \frac{rT_0}{T_{\max}} + \rho & -\frac{(\beta+\rho)\gamma}{N\beta} \\ 0 & -(\beta+\rho) & \frac{(\beta+\rho)\gamma}{N\beta} \\ 0 & N\beta & -\gamma \end{bmatrix}. \tag{2.15}$$

are given by: $\lambda_1 = -\frac{2Tr - rT_{\max} + \alpha T_{\max} + T_{\max}}{T_{\max}}$; $\lambda_2 = -\gamma - \rho - \beta$; $\lambda_3 = 0$ Thus $\lambda_3 = 0$ is a simple zero eigenvalue, and the rest are true negative eigenvalues. Conversely, when $k = k^*$(or a similar expression when$R_0 = 1$), the disease-free equilibrium ε_0 is an equilibrium that isn't hyperbolic: the presumption (A1) of Theorem 1.5 is then validated.

Denote now with $w = (w_1, w_2, w_3)^T$ the zero eigenvalue is associated with a right eigenvector $\lambda_3 = 0$

It follows:

$$\left(-\alpha + r - \frac{2rT_0}{T_{\max}}\right) w_1 - \left(\frac{rT_0}{T_{\max}} + \rho\right) w_2 - \left(\frac{(\beta+\rho)\gamma}{N\beta}\right) w_3 = 0, \tag{2.16}$$

$$-(\beta+\rho) w_2 + \left(\frac{(\beta+\rho)\gamma}{N\beta}\right) w_3 = 0, \tag{2.17}$$

$$(N\beta) w_2 - (\gamma) w_3 = 0. \tag{2.18}$$

So that;

$$w = \left(-\frac{\gamma\left(rT_0 + (\beta + 2\rho)T_{\max}\right)}{N\beta\left(2rT_0 + (r-\alpha)T_{\max}\right)}, \qquad \frac{\gamma}{N\beta}, \qquad 1\right)^T. \tag{2.19}$$

In addition, the left eigenvector $v = (v_1, v_2, v_3)$ upholding $v.w = 1$ is provided with:

$$v_1\left(-\alpha + r - \frac{2rT_0}{T_{\max}}\right) = 0, \tag{2.20}$$

$$v_1\left(-\frac{rT_0}{T_{\max}} + \rho\right) - v_2(\beta+\rho) + v_3(N\beta) = 0, \tag{2.21}$$

$$v_1\left(-\frac{(\beta+\rho)\gamma}{N\beta}\right) - v_2\left(\frac{(\beta+\rho)\gamma}{N\beta}\right) - v_3(\gamma) = 0. \tag{2.22}$$

Using $v_3 = 1$. As a result, the left eigenvector v is:

$$v = (0, 1, 1). \tag{2.23}$$

Let

$$f_1 = s - \alpha T + rT\left(1 - \frac{T + T^*}{T_{\max}}\right) - \frac{kVT}{V\alpha_1 + 1} + \rho T^*, \tag{2.24}$$

$$f_2 = \frac{kVT}{V\alpha_1 + 1} - (\beta+\rho) T^*, \tag{2.25}$$

$$f_3 = N\beta T^* - \gamma V. \tag{2.26}$$

In Theorem 1.5, the coefficients a and b are established, and this may be computed as follows: scriptsize

$$\begin{aligned} a = &\sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i \partial T_j}(\varepsilon_0, K^*) + \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i \partial V_j}(\varepsilon_0, K^*) \\ &+ \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i \partial T_j^*}(\varepsilon_0, K^*) + \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i^* \partial T_j^*}(\varepsilon_0, K^*) \\ &+ \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial V_i \partial V_j}(\varepsilon_0, K^*) + \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i^* \partial V_j}(\varepsilon_0, K^*), \end{aligned} \tag{2.27}$$

$$b = \sum_{k,i=1}^{3} v_k w_i \frac{\partial^2 f_k}{\partial T_i \partial K}(\varepsilon_0, K^*). \tag{2.28}$$

Taking to system's account (2.1)-(2.3) and taking into account all components of the left eigenvector v and also taking into account just the eigenvector v's nonzero components at virus free equilibrium;

It follows that:

$$a = \frac{\partial^2 f_1}{\partial T^2} + \frac{\partial^2 f_1}{\partial V^2} + \frac{\partial^2 f_2}{\partial V^2} + \frac{\partial^2 f_1}{\partial T \partial T^*} + \frac{\partial^2 f_1}{\partial T \partial V} + \frac{\partial^2 f_2}{\partial T \partial V}. \tag{2.29}$$

In view of (2.24) and (2.29), we then have:

$$a = -\frac{6\left(2NTr\beta - Nr\beta T_{\max} + N\alpha\beta T_{\max} + Tr\gamma - r\gamma T_{\max} + \alpha\gamma T_{\max} - \beta\gamma T_{\max}\right)^2}{T_{\max} N^2 \beta^2 \left(2Tr - rT_{\max} + \alpha T_{\max}\right)^2}, \tag{2.30}$$

where

$$T = (r-\alpha)\, T_{\max} + \frac{1}{2}\frac{\sqrt{T_{\max}^2 (r-\alpha)^2 + 4rs}}{r}.$$

And

$$b = \frac{6\left((r-\alpha)T_{\max} + \left(\sqrt{r^2 T_{\max}^2 - 2r\alpha T_{\max}^2 + \alpha^2 T_{\max}^2 + 4rs}\right)\right) G + \frac{1}{2}\sqrt{r^2 T_{\max}^2 - 2r\alpha T_{\max}^2 + \alpha^2 T_{\max}^2 + 4rs}}{\sqrt{r^2 T_{\max}^2 - 2r\alpha T_{\max}^2 + \alpha^2 T_{\max}^2 + 4rs}} \tag{2.31}$$

where

$$G = \gamma\left(\frac{1}{2}r - \frac{1}{2}\alpha + \beta\right) T_{\max}$$

Since the coefficient b is always positive, it is the sign of the coefficient a – which determines the local dynamics around the virus-free equilibrium, according to Theorem 2.2 for$k = k^*$.Thus the following result is established.

3. ADOMIAN DECOMPOSITION TECHNIQUE

Explicitly constructions of non-perturbative approximations to the system's non-perturbative solutions (2.1)-(2.3) were examining using Adomian decomposition method. This system's analogous canonical form is as follows:

$$T(t) = T(0) + \int_0^x s dt + (r-\alpha)\int_0^x T dt - \frac{r}{T_{\max}}\int_0^x T^2 + \frac{r}{T_{\max}}\int_0^x TT^* dt$$
$$-k(1+\alpha_1)^{-1}\int_0^x V dt.\int_0^x VT dt + \rho\int_0^x T^* dt, \tag{3.1}$$

$$T^*(t) = T^*(0) + k(1+\alpha_1)^{-1}\int_0^x V dt.\int_0^x VT dt - (\beta+\rho)\int_0^x T^* dt, \tag{3.2}$$

$$V(t) = V(0) + N\beta\int_0^x T^* dt - \gamma\int_0^x V dt. \tag{3.3}$$

Adomian decomposition methods for the solutions of equations (3.1)-(3.3) are considered as the following series;

$$T = \sum_{n=0}^{\infty} T_n, \tag{3.4}$$

$$T^* \sum_{n=0}^{\infty} T_n^*, \tag{3.5}$$

$$V = \sum_{n=0}^{\infty} V_n \tag{3.6}$$

The nonlinear terms in the system are then approximated (3.1)-(3.3) as follows;

$$TT = \sum_{n=0}^{\infty} \left(A_n(T_0....T_n, \qquad T_0....T_n)\right) \tag{3.7}$$

$$VT = \sum_{n=0}^{\infty} \left(B_n(V_0....V_n, \qquad T_0....T_n)\right) \tag{3.8}$$

$$TT^* = \sum_{n=0}^{\infty} \left(C_n(T_0....T_n, \qquad T_0^*....T_0^*)\right), \tag{3.9}$$

where

$$A_n = \frac{1}{n!}\left[\frac{d^n\left(\sum_{m=0}^{\infty} T_m\lambda^m\right)\left(\sum_{m=0}^{\infty} T_m\lambda^m\right)}{d\lambda^m}\right]_{m=0}, \tag{3.10}$$

$$B_n = \frac{1}{n!}\left[\frac{d^n\left(\sum_{m=0}^{\infty} V_m\lambda^m\right)\left(\sum_{m=0}^{\infty} T_m\lambda^m\right)}{d\lambda^m}\right]_{m=0}, \tag{3.11}$$

$$C_n = \frac{1}{n!}\left[\frac{d^n\left(\sum_{m=0}^{\infty} T_m\lambda^m\right)\left(\sum_{m=0}^{\infty} T_m^*\lambda^m\right)}{d\lambda^m}\right]_{m=0}. \tag{3.12}$$

The non-linear function A_n, B_n, C_n, are called Adomian's polynomials. Substituting equation (3.4)-(3.12) into (3.1)-(3.3) then we have;

$$\sum_{n=0}^{\infty} T_n = T(0) + sx + (r-\alpha)\int_0^x \sum_{n=0}^{\infty} T_n dt - \frac{r}{T_{\max}}\int_0^x \sum_{n=0}^{\infty} A_n dt$$
$$+\frac{r}{T_{\max}}\int_0^x \sum_{n=0}^{\infty} C_n dt - k\left(1+\alpha_1\right)^{-1}\int_0^x \sum_{n=0}^{\infty} V_n dt \int_0^x \sum_{n=0}^{\infty} B_n dt$$
$$+\rho\int_0^x \sum_{n=0}^{\infty} T_n^* dt, \tag{3.13}$$

$$\sum_{n=0}^{\infty} T_n^* = T^*(0) + k\left(1+\alpha_1\right)^{-1}\int_0^x \sum_{n=0}^{\infty} V_n dt \int_0^x \sum_{n=0}^{\infty} B_n dt$$
$$-(\beta+\rho)\int_0^x \sum_{n=0}^{\infty} T_n^* dt, \tag{3.14}$$

$$\sum_{n=0}^{\infty} V_n = V(0) + N\beta\int_0^x \sum_{n=0}^{\infty} T_n^* dt - \gamma\int_0^x \sum_{n=0}^{\infty} V_n dt. \tag{3.15}$$

Using the equation (3.13)-(3.15) The following scheme is described:

$$T_0 = T(0) + st, \tag{3.16}$$

$$T_0^* = T^*(0), \tag{3.17}$$

$$V_0 = V(0). \tag{3.18}$$

$$\sum_{n=0}^{\infty} T_n = (r-\alpha)\int_0^x \sum_{n=0}^{\infty} T_n dt - \frac{r}{T_{\max}}\int_0^x \sum_{n=0}^{\infty} A_n dt + \frac{r}{T_{\max}}\int_0^x \sum_{n=0}^{\infty} C_n dt$$
$$-k\left(1+\alpha_1\right)^{-1}\int_0^x \sum_{n=0}^{\infty} V_n dt \int_0^x \sum_{n=0}^{\infty} B_n dt + \rho\int_0^x \sum_{n=0}^{\infty} T_n^* dt$$
$$(\text{for } \ n \geq 0), \tag{3.19}$$

$$\sum_{n=0}^{\infty} T_n^* = k\left(1+\alpha_1\right)^{-1}\int_0^x \sum_{n=0}^{\infty} V_n dt \int_0^x \sum_{n=0}^{\infty} B_n dt - (\beta+\rho)\int_0^x \sum_{n=0}^{\infty} T_n^* dt$$
$$(for \ \ n \geq 0). \tag{3.20}$$

$$\sum_{n=0}^{\infty} V_n = N\beta\int_0^x \sum_{n=0}^{\infty} T_n^* dt - \gamma\int_0^x \sum_{n=0}^{\infty} V_n dt$$
$$(for \ \ n \geq 0). \tag{3.21}$$

Using Equation (3.7)-(3.12). The following are some of the Adomian polynomials:

$$F(t) = T^2 \tag{3.22}$$

We first set

$$T = \sum_{n=0}^{\infty} T_n. \tag{3.23}$$

Substituting (3.23) into (3.22) gives

$$[F(t) = (T_0 + T_1 + T_2 + T_3 + T_4 + T_5 + ...)^2 \tag{3.24}$$

When the right-hand side of the expression is expanded, it results in

$$F(t) = T_0^2 + 2T_0T_1 + 2T_0T_2 + T_1^2 + 2T_0T_3 + 2T_1T_2 + ... \tag{3.25}$$

The expansion in equation (3.25) can be rearranged by adding the sum of the subscripts of the components of the same to all terms. As a result, we can rewrite equations (3.25) as

$$\begin{aligned} F(t) = T_0^2 + 2T_0T_1 + 2T_0T_2 + T_1^2 + 2T_0T_3 + 2T_0T_2 + 2T_0T_4 + 2T_1T_2 \\ +T_2^2 + 2T_0T_5 + 2T_1T_4 + 2T_2T_3 + ... \end{aligned} \tag{3.26}$$

This gives Adomian polynomials for Equation (3.22) as

$$\begin{aligned} A_0 &= T_0^2, \\ A_1 &= 2T_0T_1 \\ A_2 &= 2T_0T_2 + T_1^2, \\ A_3 &= 2T_0T_3 + 2T_0T_2, \\ A_4 &= 2T_0T_4 + 2T_1T_2 + T_2^2, \\ A_5 &= 2T_0T_5 + 2T_1T_4 + 2T_2T_3, \end{aligned} \tag{3.27}$$

$$F(t) = VT \tag{3.28}$$

We first set

$$V = \sum_{n=0}^{\infty} V_n, \tag{3.29}$$

$$T = \sum_{n=0}^{\infty} T_n. \tag{3.30}$$

Substituting (3.29)-(3.30) into Equation (3.28) yields

$$\begin{aligned} F(T) = (V_0 + V_1 + V_2 + V_3 + V_4 + V_5 + ...) \times (T_0 + T_1 \\ +T_2 + T_3 + T_4 + T_5 + ...). \end{aligned} \tag{3.31}$$

When you multiply the two variables together, you get

$$\begin{aligned} F(t) = V_0T_0 + T_0V_1 + V_0T_1 + T_0V_2 + V_0T_2 + T_1V_1 + T_2V_0 \\ +T_0V_3 + T_1V_2 + T_2V_2 + T_3V_0 + T_0V_4 \end{aligned}$$

$$+V_0T_4 + T_1V_3 + V_1T_3 + V_2T_2 + ... \tag{3.32}$$

Obtaining every terms with the very same element's subscript amount T_n, We have the ability to rewrite
Equation (3.32) in the manner of:

$$F(t) = T_0V_1 + V_0T_1 + T_0V_2 + T_1V_1 + T_2V_0 + T_0V_3 + T_1V_2 + T_2V_1 + T_3V_0 + T_0V_4 + T_1V_3 + T_2V_2 + T_3V_1 + T_4V_0 + ... \tag{3.33}$$

As a result, the Adomian polynomials are denoted by

$$\left.\begin{array}{l} B_0 = T_0V_0 \\ B_1 = T_0V_1 + V_0T_1 \\ B_2 = T_0V_2 + T_1V_1 + T_2V_0 \\ B_3 = T_0V_3 + T_1V_2 + T_2V_1 + T_3V_0 \\ B_4 = T_0V_4 + T_1V_3 + T_2V_2 + T_3V_1 + T_4V_0 \end{array}\right\}, \tag{3.34}$$

$$T(t) = TT^* \tag{3.35}$$

We first set

$$T = \sum_{n=0}^{\infty} T_n, \tag{3.36}$$

$$T^* = \sum_{n=0}^{\infty} T_n^*. \tag{3.37}$$

Substituting (3.36 and 3.37) into Equation (3.35) yields

$$F(T) = (T_0 + T_1 + T_2 + T_3 + T_4 + T_5 + ...) \times ({T^*}_0 + {T^*}_1 + {T^*}_2 + {T^*}_3 + {T^*}_4 + T_5 + ...). \tag{3.38}$$

Multiplying the two factors gives

$$F(T) = T_0T_0^* + T_0^*T_1 + T_0T_1^* + T_0^*T_2 + T_1^*T_1 + T_2^*T_0 + T_0^*T_3 + T_1^*T_2 + T_2^*T_1 + T_3^*T_0 + T_0^*T_4 + T_0T_4^* + T_1^*T_3 + T_1T_3^* + T_2T_2^* + ... \tag{3.39}$$

Obtaining all terms with the very same sum of component subscripts T_n, We have the ability to rewrite equations (3.39) in the manner of:

$$F(T) = T_0T_0^* + T_0^*T_1 + T_0T_1^* + T_0^*T_2 + T_1^*T_1 + T_2^*T_0 + T_0^*T_3 + T_1^*T_2 + T_2^*T_1 + T_3^*T_0 + T_0^*T_4 + T_1^*T_3 + T_2^*T_2 + T_3^*T_1 + T_4^*T_0 + ... \tag{3.40}$$

Consequently, the Adomian polynomials, are given by

$$\begin{aligned} C_0 &= T_0T_0^*, \\ C_1 &= T_0^*T_1 + T_0T_1^*, \\ C_2 &= T_0^*T_2 + T_1^*T_1 + T_2^*T_0, \end{aligned} \tag{3.41}$$

$$C_3 = T_0^* T_3 + T_1^* T_2 + T_2^* T_1 + T_3^* T_0.$$
$$C_4 = T_0^* T_4 + T_1^* T_3 + T_2^* T_2 + T_3^* T_1 + T_4^* T_0.$$

$T = 719.57 + 1030.113124\mathrm{t} + 34.97820628\mathrm{t}^2 - 719.6610060\mathrm{t}^3 - 41.31829380\mathrm{t}^4 + ...$

$\mathrm{T}^* = 27 + 56.00465683\mathrm{t} - 39.08106622\mathrm{t}^2 - 6.185455807\mathrm{t}^3 + 11.57469526\mathrm{t}^4 + ...$

$V = 3341 - 7937.173347\mathrm{t} + 9608.980705\mathrm{t}^2 - 7735.947760\mathrm{t}^3 + 4631.970360\mathrm{t}^4 + ...$

4. NUMERICAL RESULTS AND DISCUSSION

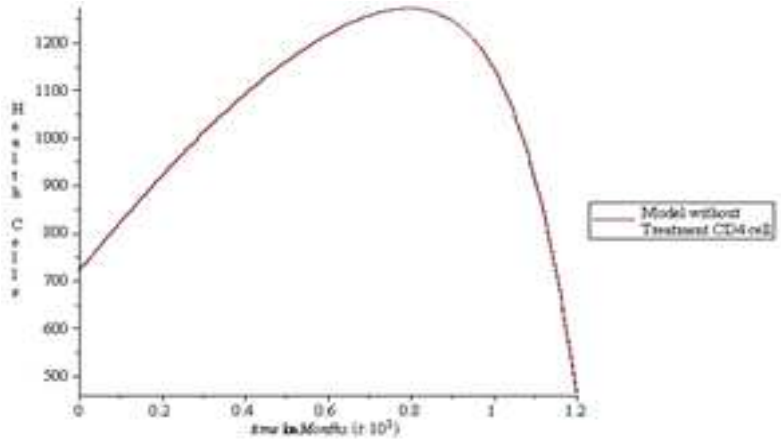

Figure 1: Uninfected $CD4^+$ T-cell count without treatment against time when $\beta = 0.3; \alpha = 0.002; r = 3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15.$

This figure shows the absence of chemotherapy in an individual's infected with HIV. The $CD_4{}^+$ T cell count reduces to zero hence it leads to full blown AIDS and give rooms for opportunist infection which leads to death.

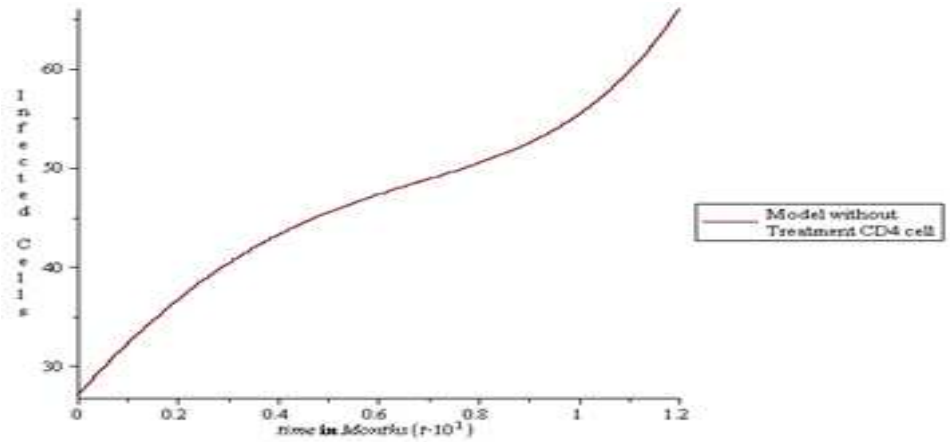

Figure 2: **I**nfected CD4$^+$ T-cell count without treatment against time when $\beta = 0.3; \alpha = 0.002; r = 3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15.$

This plot shows the absence of chemotherapy in an actively infectious $CD_4{}^+$ T cell in an individual's infected with HIV. It was observed that the cells were increasing with time.

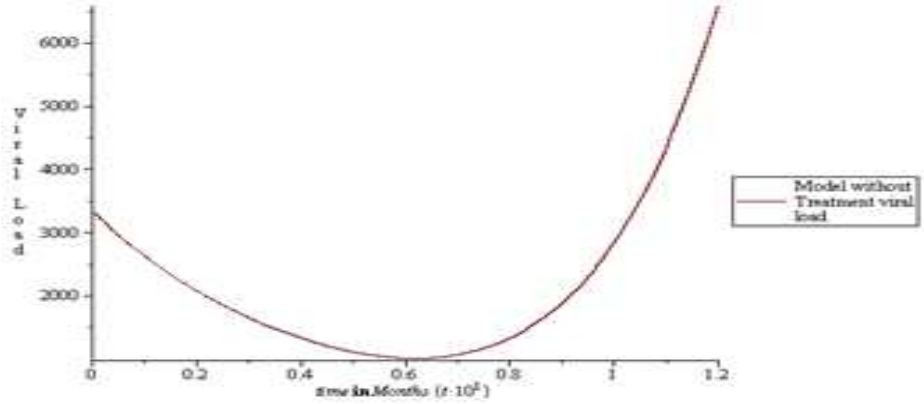

Figure 3: Viral load without treatment against time when $\beta = 0.3; \alpha = 0.002; r = 3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15.$

This graph shows the infectious viral particles in the absence of the chemotherapy during the infection. This leads to heavy viral loads (virus replication) in an individuals and it results to death.

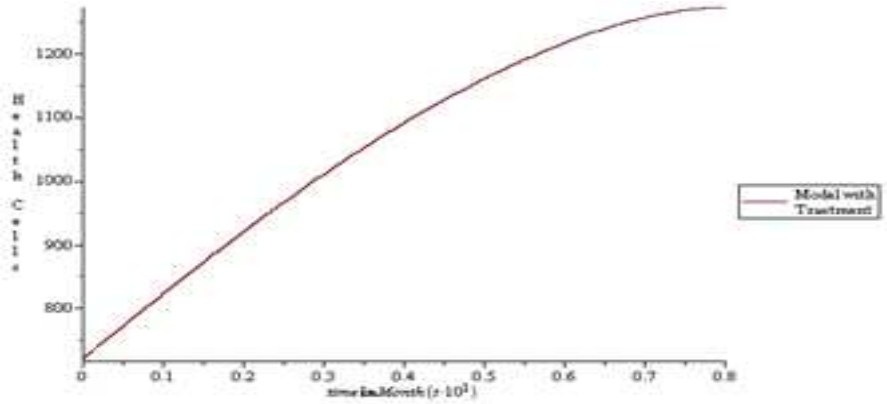

Figure 4: Uninfected CD4$^+$ T-cell count with treatment against time when $\beta = 0.3; \alpha = 0.002; r = 3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15$

This profile shows the presence of treatment in an individual. The effect of the drugs was shown by the number of $CD_4{}^+$ T cell count which is increasing with time and allows at least partial recovery of immunity. This suggests that an infected individual could live normal life.

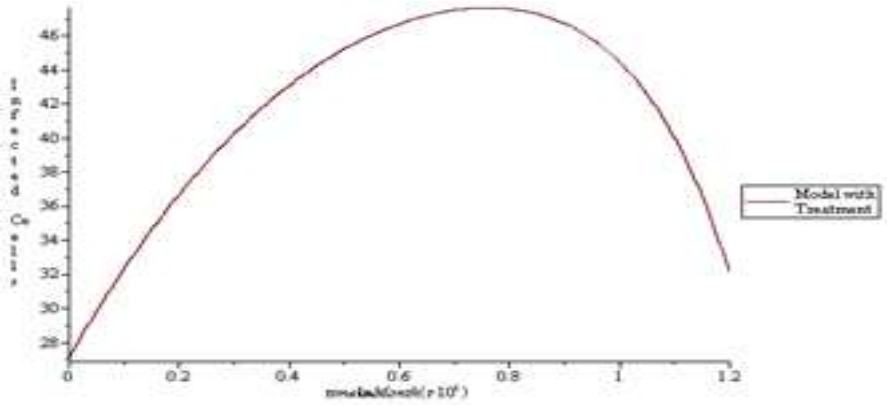

Figure 5: Uninfected CD4$^+$ T-cell count with treatment against time when $\beta = 0.3; \alpha = 0.002; r = 3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15$

This diagram shows the effect of presence of therapy by reducing the number of actively infectious $CD_4{}^+$ T cell not to progress to AIDS.

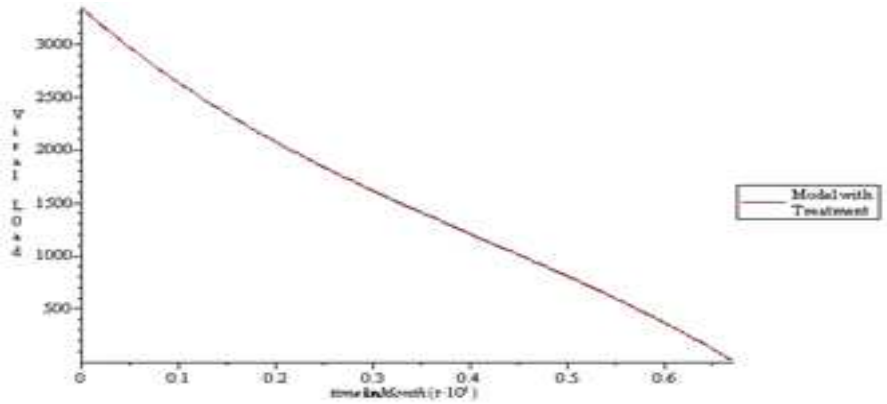

Figure 6: Viral load with treatment against time when $\beta = 0.3; \alpha = 0.002$;r= $3.0; \gamma = 2.4; k = 0.000027; T_{\max} = 1500; \rho = 0.01; s = 15$.

This figure shows the response to the drugs, suppresses activities of viral load, and this suggests that therapy could present replication of the virus.

5. CONCLUSION

We investigated a compartment's deterministic model that explains HIV viral dynamics with treatment. In this paper, our model shows that long-term activity can be divide into two categories: endemics and extinctions. We use the bifurcation method introduced in [10] and center manifold theory [18] to provide a precise indication of bifurcation thresholds.

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REFERENCES

[1] Liancheng, Wang and Michael, Y. Li. (2006): Mathematical Analysis of the global dynamics of a model for HIV infection CD4+ T cells. Mathematical Bioscience 200. 44-57.

[2] Castillo-Chavez. C and Song. B (2004): Dynamical model of tuberculosis and their application,Math. Biosci Eng., 1pp.361-404.

[3] Chowell, G. Hengartnerb, N. WQ., Castillo-chavez, C. Fenimorea, P. W. and Hyman, J. M. (2009): The basic reproduction number of Ebola and the effects of public health measures: the cases of Congo and Uganda Journal Theoretical Biology 2009.

[4] Robert L. Robertson (2005): A stage-structured model of HIV infection. International Journal of Pure and Applied mathematics vol. 25, No. 4 2005, 503-517.

[5] Patrick, W. Nelson and Alan, S. Perelson (2002): Mathematical analysis of delay differential equation models of HIV-1 infection. Mathematical Biosciences 179(2002) 73-94

[6] Merdan, M, Gokdogan, A and Erturk, V. S. (2011): An approximate solution of a model of HIV infection of $CD_4{}^+$ T cells. Iranian Journal of science and technology IJST (2011) A1:9-12.

[7] Li Jianguan and Ma Zhien (2004): Stability analysis for SIS epidemics model with vaccination and constant population size. Disc Cont Dyn Sys B 4:637-644.

[8] Hadeler K.P and Castillo-Chavez. C (1995): A core group model for disease transmission. Math Biosci 128:41-55.

[9] Dushoff J, Huang. W and Castillo-Chavez. C (1998): Backward bifurcation and catastrophe in simple models of fata disease, J Math Biol 36: 227-248.

[10] Buonomo. B and Lacitignola D. (2001). On the backward bifurcation of a vaccination model with nonlinear incidence. Nonlinear Analysis: Modelling and Control, Vol.16. No. 1, 30-46. Carr. J. Application of Centre Manifold Theory, Springe, New York, NY, USA, 1981

[11] Driessche, P. van den and Watmough James (2002): Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission. Mathematical Bioscience 180(2002) 29-48. www.elserier.com/locate/mbs

[12] Mei Yan and Zhongyi Xiang (2012): A delay-differential equation model of HIV infection of $CD_4{}^+$ T cells with cure Rate. International mathematical forum, vol. 7. 2012, no. 30, 1475-1481

[13] Alexander M.E and Moghadas S.M (2004): Periodicity in an epidemic model with a generalized nonlinear incidence. Math Biosci 189:75-96.

[14] Rusan and wang (2003): Dynamical behavior of an epidemic model with nonlinear incidence rate. J Diff Eqns 188:135-163.

[15] Zhous Xian, Donqmei and Li Yilong (2007): Bifurcations of an epidemics model with non-monotomic incidence rate of saturated mass action chaos solit fract 32: 1903-1915

[16] Alexander M.E and Moghadas S.M (2005): Bifurcation analysis of an SIRS epidemic model with generalized incidence. SIAM J Appl Math Biosci 65:1794-1816.

[17] Li Jianguan and Ma Zhien and Zhou Yicang (2006): Global analysis of SIS epidemic model with a simple vaccination and multiple endemic equilibra. Acta Math Sci 26B: 83-93. 17 2012 pp 154-159.

[18] Yongliang Li and Shunling Zhu (2011): Analysis of stability about the $CD_4{}^+$ T cells dynamics with nonlinear infection rate. Journal of Applied Mathematics and Bioinformatics, vol. 1, no.2, 75-83.

[19] Gukerhamer J and Homes P (1983); Nonlinear oscillations, dynamical systems and bifurcation of sector fields, Springer-Verlag.

[20] B.Gbadamosi, O. Adebimpe, I.O. Akinola and I.A. Olopade (2012): Solving Riccati Equation Using Adomia Decomposition Method. International Journal of Pure and Applied Mathematics.IJAM, Vol 78 No 3 (2012), 409-417

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Appendix

Let us consider a general system of ODEs with parameterϕ:

$$\frac{d\overset{\bullet}{x}}{dt} = f(x,\ \phi), \qquad f : \Re^n \times \Re \to \Re^n, \qquad f \in C^2(\Re^n \times \Re). \tag{2.32}$$

Without loss of generality, we assume that $x = 0$ is equilibrium for (2.32).

Theorem 2.2: Assume:

A1: $A = D_x f(0,0)$ is the linearization matrix of system (2.32) around the equilibrium $x = 0$ with ϕ evaluated at 0. Zero is a simple eigenvalue of A and all other eigenvalues of A have negative real parts;

A2: Matrix A has a (nonnegative) right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.
Let f_k denotes the k^{th} component of f,and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial T_i \partial T_j}(0,0), \tag{2.33}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial T_i \partial \phi_j}(0,0). \tag{2.34}$$

The local dynamics of system (2.32) around $x = 0$ are totally determined by a and b.

(1) $a > 0$, $b > 0$,when $\phi > 0$, with $|\phi| << 1$, $x = 0$ is locally asymptotically stable and there exists a positive unstable equilibrium; when $0 > \phi << 1$, $x = 0$, is unstable and there exists a negative and locally asymptotically stable equilibrium;
(2) $a < 0$, $b < 0$,when $\phi < 0$, with $|\phi| << 1$, $x = 0$ is unstable; when $0 < \phi << 1$, $x = 0$, is locally asymptotically stable and there exists a positive and unstable equilibrium;
(3) $a > 0$, $b > 0$,when $\phi < 0$, with $|\phi| << 1$, $x = 0$ is unstable and there exists a locally asymptotically stable negative equilibrium; when $0 < \phi << 1$, $x = 0$, is stable and a positive unstable equilibrium appears;

(4) $a > 0$, $b < 0$,when ϕ changes from negative to positive, $x = 0$ changes its stability from stable to unstable. Correspondently, a negative unstable equilibrium becomes positive and locally asymptotically stable.

Proof:

Let $\xi^c$and ξ^c be the generalized eigenspaces of A for the zero eigenvalue and all other eigenvalues, respectively. It is follows from the center manifold theory that center manifold W^c is one dimensional and $\Re^n = \xi^c \otimes \xi^s$. Parameterize the center manifold by $c\,(t)$ and decompose it into ξ^candξ^c, that is,

$$W = \{c(t)w + h(c,\phi) : v.h(c,\phi) = 0, |c| \leq c_0, c(0) = 0\}, \quad (2.35)$$

Where $c(t) \in \xi^c$and$h(c,\phi) \in \xi^c$. Because the center manifold is tangent to ξ^c at the origin, $h(c,\phi)$is a higher order term ($h(c,\phi)$has at least order 2). It also follows by the invariance of the center manifold under the flow that;

$$\frac{d}{dt}\left(c(t)w + h(c,\phi)\right) = f(c(t)w + h(c,\phi),\phi), \quad (2.36)$$

Applying Taylor expansion to the right hand side of equation (2.34) at $(0,0)$ and noticing that
$h\,(c,\phi)$ is higher order, we obtain that

$$\begin{aligned} f(c(t)w + h(c,\phi),\phi) &= f(0,0) + D_x f(0,0)((c(t)w + h(c,\phi) \\ &+ D_\phi f(0,0)\phi) + \tfrac{1}{2}(I_n \otimes (cw + h(c,\phi))^1)(D^2_{xx} f(0,0)) \\ &(c(t)w + h(c,\phi)) + \phi(D^2_{x\phi} f(0,0))(cw + h(c,\phi)) \\ &+ \tfrac{1}{2}\phi^2(D^2_{\phi\phi} f(0,0)) + \textit{higher order terms}, \end{aligned} \quad (2.37)$$

where $D^2_{x\phi}$ is the Hessian matrix; In is the identity matrix of order n; $\otimes$ is the Kronecker product. Using

$$f(0,0) = D_x f(0,0)c(t)w = D_\phi f(0,0) = D^2_{\phi\phi} f(0,0) = 0. \quad (2.38)$$

And the fact that $ch(c,\phi)$is of higher order, we simplify the above expansion for f as (higher order terms are dropped).

$$f(0,0) = (D_x f)h(c,\phi) + \frac{c^2}{2}(I_n \otimes w^1)(D^2_{xx} f)w + c\phi(D^2_{x\phi})w. \quad (2.39)$$

Multiplying both sides of equation (2.34) by v and using the fact that $v.h = 0$and $vD_x f(0,0) = 0$, we finally obtain the following equation for $c(t)$:

$$= \frac{c^2}{2} \sum_{k,i,j}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} + \sum_{k,i}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial x_\phi} c\phi, \tag{2.40}$$

$$\frac{dc}{dt} = \frac{c^2}{2} v(I_n \otimes w^1) D^2_{xx} f w + c\phi v D^2_{x\phi} f w. \tag{2.41}$$

$$= \frac{a}{2} c^2 + b\phi c. \tag{2.42}$$

Namely,

$$\frac{dc}{dt} = \frac{a}{2} c^2 + b\phi c. \tag{2.43}$$

Obviously, at $\phi = 0$ a transcritical bifurcation takes place in equation (2.43).