

## ANALYSIS OF HIV-1 MATHEMATICAL MODEL USING TAYLOR'S SERIES METHOD

by

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*The main objective of this study is the use of Taylor's series method for approximate solution of HIV-1 infection model. This method explores to solve a system of ODE expressed as an infinite series. These series components are easily determined. The presented method's effectiveness and reliability are shown using a numerical example, and the consequences are evaluated to those acquired from different techniques in the research using tables and graphs. The proposed method has no assumptions about small or large parameters, and the technique's accuracy increases when the order of approximation is increased. The results reveal that the approximate solution obtained through the use of Taylor's series method is more reliable and accurate.*

Key words: *Taylor's series, approximate solution, HIV-1 model*

### Introduction

Infectious diseases, alternatively referred to as communicable diseases, have previously considered as a persistent menace to humans worldwide. The term *infectious diseases* refers to diseases that can be transferred between humans to humans, or humans to animals and *vs.* Bacteria, airborne viruses, and bodily fluid, such as urine, saliva, plasma, breast milk, and tears, all play a part in transmission.

It kills and destroys CD4 cells, which are commonly referred to as T cells. The HIV can damage a large number of these cells over time, impairing the body's ability to fight infection and illness. Those specific platelets support immune system in its battle against infections. If left undiagnosed HIV infection, reduces the body's CD4 cells (T cells). As the immune system deteriorates, the body's ability to fight infections and various diseases becomes increasingly challenging. Opportunistic illness or tumors take favor of a person's weakened immune condition and serve as a warning indicator of HIV infection. The HIV/AIDS is a serious worldwide health problem on a global level. Each year, millions of dollars are spent on the disease's treatment, yet there is currently no cure. Sub-Saharan Africa is home to around 25 million HIV-positive individuals, Center for Disease Control. A variety of mathematical models are discussed to aid in the understanding of HIV infection, progression of the disease, and antiretroviral ther-

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apy, among other things. Recent investigations have revealed that a considerable number of CD4+ T cells are diagnosed with the disease, suggesting how this subset among T cells is selectively affected. A researcher devised a model for HIV infection in 1989. Three variables are included in this model of virus spread: population numbers of plasma membranes, infected cells, and uncontrolled organisms. Combining mathematical and statistical analyses of human immune reaction viral infectious disease via clinical measures provides crucial information regarding HIV-1 pathogenicity and improved our understanding of the virus. As a result, it is an effective tool for constructing accurate mathematical models. Mathematical modelling is used to predict disease outbreaks. Additionally, it studies HIV-1 preventive and treatment strategies. When HIV enters into an organism, it infects a large number of CD4+ T cells and immediately duplicates. The blood possesses a heavy concentration of HIV virus fragments at this initial stage of infection, which expands across the organism. The HIV germs are transferred through bodily secretions such as urea, spit, breast feeding, blood, and tears. The HIV is detected in these body fluids in the form of free viral particles as well as virus contained among contaminated immune cells. The HIV is a type of virus that attacks CD4+ T-cell lymphocytes in humans, which relates to immune system's most numerous white blood cells. Due to the critical component of CD4+ T cells with immunological control, both loss and killing can get a vast range of detrimental impacts on the immune system's functioning, reducing its resilience. Indeed, the reduction in quantity of these cells is utilized for treatment trials like a marker in the process of AIDS. The immunodeficiency that characterizes AIDS is distinguished by a disruption in CD4+ T-cell function, for additional information, check [1-4].

As of late, various models based on body immune system have already been constructed with substantial exploration on HIV contamination of CD4+ T cells that has been accomplished to better knowledge of HIV dynamics, infection, disease movement, and immune system's interaction on HIV. Mathematical models with transmission of transferable diseases are exerting a rising effect on disease control and management practice and concept [5]. Perelson [1] introduced the model for primary HIV infection in 1989. Perelson *et al.* [2] refined model of Perelson and quantitatively demonstrated a portion of the model's way of behaving. It was noticed that model has a variety of AIDS, including CD4+ T-cell depletion, low degrees of free virus within the body, and a prolonged latency phase. Free viral particles, healthy CD4+ T cells, latently affected CD4+ T cells, and effectively tainted CD4+ T cells were the four divisions that they used to create the model. They showed the elements of these parts by the utilization of an arrangement of four differential equations. Perelson *et al.* [2] model has spawned a slew of HIV infection models, all of which gigantically affect the area of numerical demonstrating of HIV disease. Culshaw and Raun [6] reduce complexity of Perelson *et al.* [2] method to three components: free infection particles, affected and unaffected CD4+ T cells. Moreover, add a discrete time delay into the model and demonstrate time difference through viral particle expulsion and cellular disease. Ogunlaran and Noutchie [7] examined HIV infection system using factors: unaffected cells of CD4+ T and free virion incidence. Ogunlaran and Noutchie [7] sought to enhance the convergence of untreated CD4+ T cells while applying the fewest possible medicines and to prevent infected cells from reproducing. Galvao-Lima *et al.* [8] studied macrophages and their role through HIV virus. Yuzbasi and Karacayir [9] used Galerkin technique to deal with a model of HIV infection. They adopted a technique known as residual correction, the objective of which was to minimize the solution error. Li and Xiao [10] investigated global elements of an infection invulnerable framework in order to determine HIV viral load and the structure of treatment interferences. Additionally, they examined model's sliding and global dynamics which are determined by HIV disposal rate and the infected cells' development rate. Theys *et al.* [11] studied the impact of HIV-1 upon host cell with its spread.

They also sought to establish a link between the evolution of bacteria within the host cell and the host cell's fitness. Ransome *et al.* [12] conducted a study on the transmission, treatment, and anticipated HIV infection through socialization. They recognized that social investment plays a significant role in HIV transmission starting with one individual then on the next. Duro *et al.* [13] demonstrated tracking of CD4 cells for HIV-infected individuals using CD4 cell levels. Angulo *et al.* [14] established primary route of HIV-1 transmission among mother and kid. Hallberge *et al.* [15] defined developed period of data about the meaning of HIV exposure across collaborators. Omondi *et al.* [16] examined the transmission of HIV between two groups of people of varying ages using a mathematical model. Additionally, they demonstrated that males are less susceptible to infection than their female spouse. Ali *et al.* [17] discussed adomain decomposition method (ADM) for computing the solution of an HIV-infected model. The ADM demonstrates how to solve ODE concerning indefinite series variables. Alqudah *et al.* [18] developed a numerical system related to HIV infection within the sight of foundational microorganism medication and explored the influence of therapy on certain dynamical way of behaving of viral burden. As a medical intervention, stem cells are critical for the treatment of a variety of disorders. They found that while stem cell treatment can't give a fix, it can improve the quality of living and help people live longer in HIV-infected individuals. Numerous alternative analytical and numerical approaches have been used in the literature to determine approximate solution of HIV infection model, [19-25] for more information.

In this work, we have used Taylor series method (TSM), introduced by Brooke Taylor in 1715. Later, different researchers implemented the proposed method for solution of different problems arises in different field of science and technology. Recently, He and Ji [26] employed the proposed technique to find the solution of Lane-Emden equation, He *et al.* [27] obtained solution for fractal Bratu-type equation arising in electrospinning process, and Yu and Lin [28] implemented, Taylor series expansion for solving population balance equation. In the present study we have used proposed method for approximate solution of HIV-1 model.

*Taylor's series:* Let  $f(a + h)$  be an infinite differentiable function of variable  $h$  and  $a$  be a constant then there Taylor expansion:

$$f(a + h) = f(a) + hf'(a) + \frac{h^2}{2!} f''(a) + \frac{h^3}{3!} f'''(a) + \dots + \frac{h^n}{n!} f^n(a)$$

### Analysis of HIV-1 model

In this chapter we use TSM for approximate solution of HIV-1 model. Consider the model in the form of [29]:

$$\begin{aligned} \frac{dy(x)}{dx} &= a - by(x) - cy(x)z(x) \\ \frac{dw(x)}{dx} &= cy(x)z(x) - \delta w(x) \\ \frac{dz(x)}{dx} &= \varepsilon w(x) - \eta z(x) \end{aligned} \tag{1}$$

where  $y(x)$  represent uninfected and  $w(x)$  infected CD4+ T-cells. Also  $z(x)$  is the density of virus in its infectious state or virions in plasma. The rate of production of production of CD4+ T-cells denoted by  $a$ . The  $b$  and  $c$  represent the rate of natural death and rate of infected CD4+ T-cells from uninfected CD4+ T-cells. Furthermore,  $\delta$ ,  $\varepsilon$ , and  $\eta$  show infection creating cells death, generation of virion diseases due to infected cells and death rate of virus molecule, respectively.

To begin, we first consider the following numerical values for the aforementioned parameter in the HIV-1 model:

$$a = 0.272 \text{ (day per mm}^3\text{)}, b = 0.00136 \text{ (day per mm}^3\text{)}, c = 0.00027 \text{ (day per mm}^3\text{)}$$

$$\delta = 0.33 \text{ (day per mm}^3\text{)}, \varepsilon = 50 \text{ (virion per day)}, \eta = 2.0 \text{ (day)}$$

with these values eq. (1) become:

$$\begin{aligned} \frac{dy(x)}{dx} &= 0.272 - 0.00136y(x) - 0.00027y(x)z(x) \\ \frac{dw(x)}{dx} &= 0.00027y(x)z(x) - 0.33w(x) \\ \frac{dz(x)}{dx} &= 50w(x) - 2z(x) \end{aligned} \quad (2)$$

with initial condition

$$y(0) = 100, w(0) = 0, \text{ and } z(0) = 1$$

Now use Taylor series expansion for  $y(x)$ ,  $w(x)$ , and  $z(x)$ :

$$\begin{aligned} y(x) &= \sum_{n=0}^{\infty} \frac{x^n}{n!} y^n(0) \\ w(x) &= \sum_{n=0}^{\infty} \frac{x^n}{n!} w^n(0) \\ z(x) &= \sum_{n=0}^{\infty} \frac{x^n}{n!} z^n(0) \end{aligned} \quad (3)$$

For  $x = 0$  system (2) become:

$$\begin{aligned} y'(0) &= 0.272 - 0.00136y(0) - 0.00027y(0)z(0) \\ w'(0) &= 0.00027y(0)z(0) - 0.33w(0) \\ z'(0) &= 50w(0) - 2z(0) \end{aligned}$$

Using initial condition,  $y(0) = 100$ ,  $w(0) = 0$ , and  $z(0) = 1$ , one can get:

$$\begin{aligned} y'(0) &= 0.109 \\ w'(0) &= 0.027 \\ z'(0) &= -2 \end{aligned} \quad (4)$$

Now to find  $y''(0)$ ,  $w''(0)$ , and  $z''(0)$  we differentiate eq. (2), and get:

$$\begin{aligned} y''(x) &= -0.00136y'(x) - 0.00027(y(x)z'(x) + z(x)y'(x)) \\ w''(x) &= 0.00027(y(x)z'(x) + z(x)y'(x)) \\ z''(x) &= 50w'(x) - 2z'(x) \end{aligned} \quad (5)$$

Put  $x = 0$  in eq. (5), we get:

$$\begin{aligned} y''(0) &= -0.00136y'(0) - 0.00027(y(0)z'(0) + z(0)y'(0)) \\ w''(0) &= 0.00027(y(0)z'(0) + z(0)y'(0)) \\ z''(0) &= 50w'(0) - 2z'(0) \end{aligned} \quad (6)$$

Use initial condition and eq. (4) in eq. (6) one can get:

$$\begin{aligned} y''(0) &= 0.05382233 \\ w''(0) &= -0.06288057 \\ z''(0) &= 5.35 \end{aligned} \tag{7}$$

Now differentiate eq. (5) to get  $y'''(x)$ ,  $w'''(x)$ , and  $z'''(x)$ :

$$\begin{aligned} y'''(x) &= -0.00136y''(x) - 0.00027(y(x)z''(x) + z'(x)y'(x) + z(x)y''(x) + y'(x)z'(x)) \\ w'''(x) &= 0.00027(y(x)z''(x) + y'(x)z'(x) + z(x)y''(x) + y'(x)z'(x)) - 0.33w''(x) \\ z'''(x) &= 50w''(x) - 2z''(x) \end{aligned} \tag{8}$$

Put  $x = 0$  in eq. (8), we get:

$$\begin{aligned} y'''(0) &= -0.00136y''(0) - 0.00027(y(0)z''(0) + z'(0)y'(0) + z(0)y''(0) + y'(0)z'(0)) \\ w'''(0) &= 0.00027(y(0)z''(0) + y'(0)z'(0) + z(0)y''(0) + y'(0)z'(0)) - 0.33w''(0) \\ z'''(0) &= 50w''(0) - 2z''(0) \end{aligned} \tag{9}$$

Use initial condition, eqs. (4) and (7) in eq. (9) one can get:

$$\begin{aligned} y'''(0) &= -0.14442001397 \\ w'''(0) &= 0.165228188391 \\ z'''(0) &= -13.8440285 \end{aligned} \tag{10}$$

Now differentiate eq. (8) to get  $y^{iv}(x)$ ,  $w^{iv}(x)$ , and  $z^{iv}(x)$ :

$$\begin{aligned} y^{iv}(x) &= -0.00136y'''(x) - 0.00027(y(x)z'''(x) + z''(x)y'(x) + z'(x)y''(x) + y'(x)z''(x) + \\ &\quad + z(x)y'''(x) + y''(x)z'(x) + y'(x)z''(x) + z'(x)y''(x)), \\ w^{iv}(x) &= 0.00027(y(x)z'''(x) + z''(x)y'(x) + y'(x)z''(x) + z'(x)y''(x) + z(x)y'''(x) + \\ &\quad + y''(x)z'(x) + y'(x)z''(x) + z'(x)y''(x)) \\ z^{iv}(x) &= 50w'''(x) - 2z'''(x) \end{aligned} \tag{11}$$

Put  $x = 0$  in eq. (11), we get:

$$\begin{aligned} y^{iv}(0) &= -0.00136y'''(0) - 0.00027(y(0)z'''(0) + z''(0)y'(0) + z'(0)y''(0) + y'(0)z''(0) + \\ &\quad + z(0)y'''(0) + y''(0)z'(0) + y'(0)z''(0) + z'(0)y''(0)) \\ w^{iv}(0) &= 0.00027(y(0)z'''(0) + z''(0)y'(0) + y'(0)z''(0) + z'(0)y''(0) + z(0)y'''(0) + \\ &\quad + y''(0)z'(0) + y'(0)z''(0) + z'(0)y''(0)) \\ z^{iv}(0) &= 50w'''(0) - 2z'''(0) \end{aligned} \tag{12}$$

Use initial condition, eqs. (4), (7), and (10) in eq. (12) one can get:

$$\begin{aligned} y^{iv}(0) &= 0.3736033807339 \\ w^{iv}(0) &= -0.373442603578 \\ z^{iv}(0) &= 35.94946641955 \end{aligned} \tag{13}$$

By differentiating eq. (11), one can get:

$$\begin{aligned} y^v(0) &= -0.970393154435 \\ w^v(0) &= 0.9707000320873 \\ z^v(0) &= -90.571063018 \end{aligned} \tag{14}$$

The 5<sup>th</sup> order approximation of Taylor series as:

$$\begin{aligned} y(x) &= y(0) + xy'(0) + \frac{x^2}{2!} y''(0) + \frac{x^3}{3!} y'''(0) + \frac{x^4}{4!} y^{iv}(0) + \frac{x^5}{5!} y^v(0) \\ w(x) &= w(0) + xw'(0) + \frac{x^2}{2!} w''(0) + \frac{x^3}{3!} w'''(0) + \frac{x^4}{4!} w^{iv}(0) + \frac{x^5}{5!} w^v(0) \\ z(x) &= z(0) + xz'(0) + \frac{x^2}{2!} z''(0) + \frac{x^3}{3!} z'''(0) + \frac{x^4}{4!} z^{iv}(0) + \frac{x^5}{5!} z^v(0) \end{aligned} \quad (15)$$

Using initial values, eqs. (4), (7), (10), (13), and (14) in eq. (15), we get the 5<sup>th</sup> order approximate solution:

$$\begin{aligned} y(x) &= -0.008086609620x^5 + 0.0155668075305x^4 - 0.024070002328x^3 + \\ &\quad + 0.026911165x^2 + 0.109x + 100 \\ w(x) &= 0.0080891669340x^5 - 0.01556018482x^4 + 0.0275380313985x^3 - \\ &\quad - 0.031440285x^2 + 0.027x \\ z(x) &= -0.754758858483x^5 + 1.4978944341479x^4 - 2.307338083333x^3 + 2.675x^2 - 2x + 1 \end{aligned}$$

## Result and discussion

The analysis of biological infectious disease model is carried out through a well-known method called TSM. The proposed method's results are compared to those achieved using other methods in the literature.

Table 1 show the comparison of 5<sup>th</sup> order TSM solution of uninfected CD4+ T-cells  $y(x)$ , with Bessel Method, B-QLM, LWM, LADM, HAM, HPM, ABM, and RK4, results of TSM show close resemblance with the solution of these methods. Table 2 show the evaluation of 5<sup>th</sup> order TSM solution of infected CD4+ T-cells  $w(x)$ , with Bessel Method, B-QLM, LWM, LADM, HAM, HPM, ABM, and RK4, results of TSM express a local similarity with the solution of these methods. While results for the virus's density in its infective form,  $z(x)$ , are shown in tab. 3.

**Table 1. Comparison of 5<sup>th</sup> order TSM solution of  $y(x)$  with other methods [29]**

$x$	Bessel	B-QLM	LWM	LADM	HAM	HPM	ABM	RK4	TSM
0.2	100.023	100.022	100.023	100.023	100.023	100.023	100.023	100.023	100.023
0.4	100.047	100.046	100.047	100.046	100.047	100.047	100.048	100.047	100.047
0.6	100.071	100.071	100.071	100.070	100.071	100.071	100.073	100.071	100.071
0.8	100.097	100.097	100.097	100.092	100.097	100.097	100.098	100.097	100.096
1	100.121	100.121	100.122	100.112	100.122	100.123	100.123	100.122	100.119

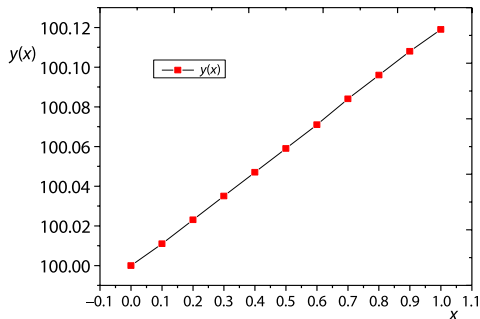
**Table 2. Comparison of 5<sup>th</sup> order TSM solution of  $w(x)$  with other methods [29]**

$x$	Bessel	B-QLM	LWM	LADM	HAM	HPM	ABM	RK4	TSM
0.2	0.00433	0.00433	0.00451	0.00436	0.00434	0.00434	0.00434	0.00433	0.00434
0.4	0.00715	0.00715	0.00727	0.00756	0.00714	0.00721	0.00715	0.00715	0.00721
0.6	0.00908	0.00908	0.00916	0.01074	0.00909	0.00934	0.00908	0.00908	0.00944
0.8	0.01049	0.01049	0.01055	0.01336	0.01063	0.01117	0.01049	0.01049	0.01185
1	0.01161	0.01161	0.01167	0.01866	0.01194	0.01276	0.01161	0.01161	0.01562

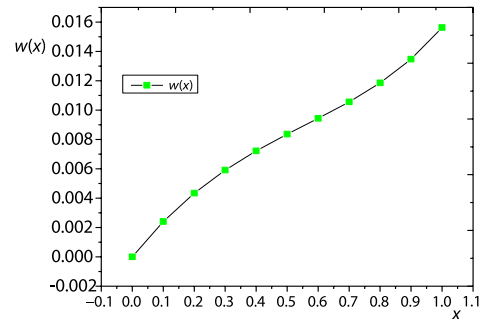
**Table 3. Comparison of 5<sup>th</sup> order TSM solution of  $z(x)$  with other methods [29]**

$x$	LWM	LADM	HAM	HPM	ABM	RK4	TSM
0.2	0.69074	0.69581	0.69059	0.69071	0.67900	0.69070	0.690696
0.4	0.51192	0.53851	0.51237	0.51208	0.50532	0.51190	0.510948
0.6	0.41103	0.46097	0.40994	0.41394	0.40745	0.41103	0.400052
0.8	0.35683	0.39607	0.35148	0.37749	0.35503	0.35684	0.296861
1	0.33072	0.27671	0.32869	0.42419	0.33252	0.33073	0.110797

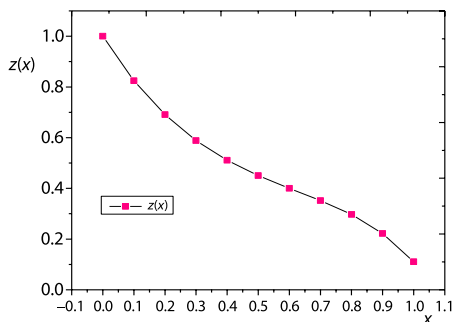
Figures 1-3 show the approximate solution for uninfected CD4+ T-cells  $y(x)$ , infected CD4+ T-cells  $w(x)$  and the virus's density in its infective form  $z(x)$ , respectively. While figs. 4-6 show the comparison of TSM solutions with other methods.



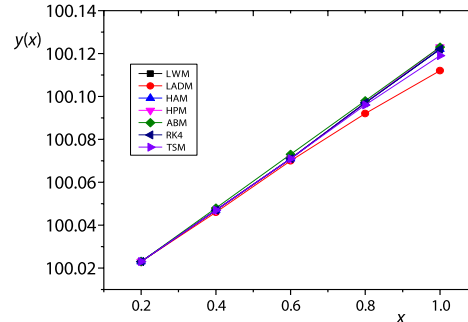
**Figure 1. Show approximate solution of 5<sup>th</sup> order TSM for  $y(x)$**



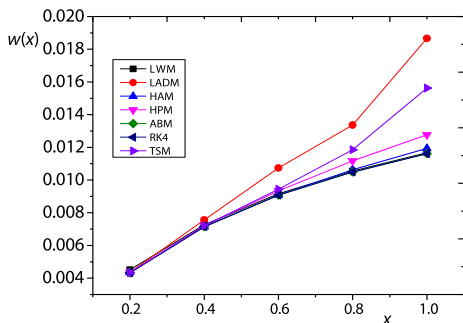
**Figure 2. Show approximate solution of 5<sup>th</sup> order TSM for  $w(x)$**



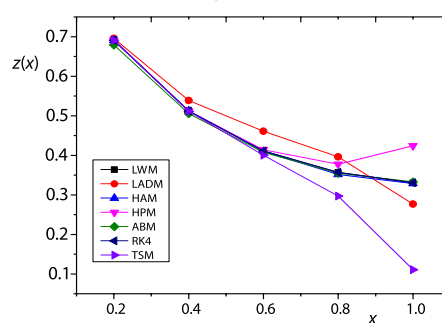
**Figure 3. Show approximate solution of 5<sup>th</sup> order TSM for  $z(x)$**



**Figure 4. Comparison of TSM with other methods for  $y(x)$**



**Figure 5. Comparison of TSM with other methods for  $w(x)$**



**Figure 6. Comparison of TSM with other methods for  $z(x)$**



## Conclusion

In this study the TSM is successfully applied for approximate solution of the mathematical model of HIV-1. Results obtained through the proposed scheme are very encouraging and interesting. The proposed method does not required discretization like other numerical method and also free from small or large parameter assumption. The presented method is simple in use, straight forward and required less computational work. Accuracy of the method increases with increase the order of approximation. It is cleared from the previous results and discussion that TSM is one of the powerful methods for obtaining approximate solution, as a result it will be more appealing for researchers to use this technique for different problems arises in different field of science and engineering.

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## Conflict of interest

No conflict of interest exists.

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