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ANALYSIS OF HIV-1 DYNAMICS UNDER VARIOUS HAART REGIMENS FOR STABILITY

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Abstract. This article delves into the realm of HIV research, focusing on mathematical models utilizing Delay Differential Equations (DDEs). Specifically, the study examines a distinct category of these models, emphasizing the identification of the bifurcation parameter within DDEs to ascertain the steady state of the system. The analysis extends to incorporate variable constant delays, addressing the critical issue of system stability. The primary objective is to establish a balanced condition by considering various factors such as local and global asymptotic states, the bifurcation parameter, and its sensitivity. The study employs a comprehensive approach, taking into account the intricate interplay of these factors to draw meaningful conclusions based on stability data. The investigation highlights the achievement of a disease-free equilibrium through the application of bifurcation analysis. The article showcases the improvement of the global stability of this equilibrium, underscoring the significance of the obtained results. By navigating through the complexities of HIV models using DDEs, this research contributes valuable insights into understanding the dynamics of the disease, with potential implications for informing intervention and treatment strategies.

Keywords: delay differential equation; bifurcation; stability; HIV.

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1. Introduction

Over time, numerous mathematical models have been employed to study human immunity and the immune system's reaction to HIV. Such a model tackles this problem from multiple angles using DDE [1]. Both linear and nonlinear models are used in the implemented DDE [2], with delay measures acting as an additional parameter. Cell kinds include susceptible, immune, and infectious cells, among others, which are included in the additional parameter. These models facilitate knowledge about HIV, how it spreads, and how to recover [3].

Consider the HIV model.

$$\frac{dv}{dt} = UP - cV \tag{1}$$

where c is the constant against V us variation and UP is the unknown virus production. Until a mathematical model describes the virus population and its growth, the virus growth pattern remains unclear. The model is used to measure the HIV growing population, and it is assessed by

$$P = r - dP + aP(1 - \frac{P}{P_{max}}) \tag{2}$$

where r is the population growth rate for the subsequent generation of new HIV cells. The population depth is represented by dP, and the maximum HIV-related death rate is represented by aP, or the bifurcation.The human immune system is almost always predictable since it responds quickly to alien diseases and the P max represents the greatest amount of disease that can spread in response to a critical attack on the immune system.

HIV [4] cannot spread since it is dependent on the health of the human immune system. A copy of DNA is present in every virus cell, and these copies are dispersed throughout the human immune system. Every human immune system acts as a host for the growth of viruses in human cells. The infection ratio is calculated using the increase of the virus population, with population P storing the greatest amount of virus growth. The virus's evaluation section makes use of the protein ratio present on the surface of human immune cells. Each HIV cell has an RNA that changes into the DNA of an immune cell in order to multiply. After the conversion of DNA, the virus started to multiply inside the host cell, producing more HIV cells. Although the procedure takes time to spread and convert, the spread ratio started to climb after the first conversion.

Thus, the HIV growth chart is evident and has an impact on the immune system. The immune system is compromised, which results in death. There are four phases from virus spread to human mortality. Stage 1 addresses the introduction of the virus into the body using the asymptotic steady state; Stage 2 addresses the transient using the bifurcation parameter; Stage 3 addresses the latency period using the parameter sensitivity; and Stage 4 addresses the spread ratio using the optimal control mechanism.

Anti-narcotics, which offer stability and balancing support to enable disease prevention against its overall spread, are a common component of current HIV therapy [5]. Anti-narcotics methods such as chemotherapy, which use the bifurcation strategy to turn the infected cell into a recovery cell, restrict the disease's rapid development to prevent disease duplication cells. In order to prevent HIV from spreading, a model is thus created to guarantee the time stability [6] of the cell.

Utilizing the delay system principle, which determines the overall disease spread based on input from the bifurcation technique, the disease spread is assessed [7]. The preventive medication works as an encroachment between the plasma and its total concentration, eventually diluting it in a week to maintain cell stability [8]. By assessing the disease cell spread ratio, stability is guaranteed and controlled during the early stages. When the previous phases are taken into account as comparison measures, the ratio tends to slow down as a result of the therapy that has been invoked. The stability varies according to the individual differences in the human immune system. The triggered cell has the potential to kill itself or be destroyed inside the individual as a result of this suppression. In order to eradicate the spreading virus, the plasma count must be higher. The RNA plasma recovered throughout the therapeutic process has the potential to destroy the spreading cells. Therefore, in order to invoke best practices for supplying an overall immune system, the model needs to be more dynamic in order to grasp cell activity and its growth phase. Collectively, these procedures fall under the category of effective dynamic model deactivation of the infected cell.

2. PROPERTIES OF DYNAMIC MODEL

The following requirements are met by the model that will be used: (i) stability; (ii) delay; (iii) affected rate; and (iv) cure rate. The following equation is used to set the infection ratio for the stability impact while proposing a model.

$$\frac{dP}{dt} = s - dP + aP(1 - \frac{P}{P_{max}}) - \beta PV + \rho I \tag{3}$$

$$\frac{dI}{dt} = \beta PV - (\delta + \rho)I \tag{4}$$

$$\frac{dv}{dt} = qI - cV \tag{5}$$

where I stands for the infected cell, V for volume, and P for the total population. βPV, where β is the infection rate, is the disease infection rate. The linear rate of virus spread is obtained by calculating the density of infected cells with δ.

Delay systems find extensive application, particularly in the field of engineering science. Variable discrete delays are used in this work to address DDE[9]. Based on the past and future variables, DDE is classified as bounded and unbounded, with a variable or fixed time delay that adapts to the changing state [10]. Such an application reflects the dynamics, control unit, and machine processing mechanism. Time delays affect all of these systems, which results in unstable systems and earlier performance [11], among other noteworthy harms brought on by instability.

Time delay systems are typically evaluated for performance using DDE and adhere to dynamics [12]. A stability lobe diagram, which evaluates the system's stability in conjunction with the control segment, is used to explain the stability flow of such a system. In order to address this problem and preserve the stability of all control systems, more analytical and numerical derivations are concentrated. The illness depth parameter and the disease spread speed are the stability control parameters.

2.1. Disease spread speed.

To classify stability based on its subdivisions, the stability criteria are elaborated utilizing scalar and frequency domain methods. The shifted polynomial approximation notion offers a novel method for analyzing the system by portraying stability through the monodromy matrix. The rate of spread is measured using the delay parameter, where β and

δ represent the spread rate and depth rate, respectively.

$$\frac{dP}{dt} = s - dP + aP(1 - \frac{P}{P_{max}}) - \beta P(t-i)V(t-i) + \rho I \tag{6}$$

$$\frac{dI}{dt} = \beta P(t-i)V(t-i) - (\delta + \rho)I \tag{7}$$

$$\frac{dv}{dt} = qI - cV \tag{8}$$

The delay and spread of the virus are assessed using the frequency virus spreading ratio, which is measured in relation to the general population. The non-infected cells are identified by tracing their spread value. In the given equation, t_i represents the ratio of times to infection in relation to the total population. The variable i represents the time interval between an infected cell and a non-infected cell, where i is a positive constant. The delay is also quantified by employing ti words that fulfill the initial condition.

3. Disease Depth Parameter

The depth is quantified by measuring the illness depth relative to the uninfected cells. The uninfected stage commences with the starting value θ. Therefore, P_0, V_0, and I_0 represent the beginning values of population, velocity, and the infection period respectively. These values are established during the initial inspection phase to presume that the cells are devoid of any viruses. The variable Bar calculates the ratio of infected cells based on its previously recorded values of $\bar{P_0}, \bar{V_0}, \bar{I_0}$. In order to prioritize the state, the beginning and end values are closely watched and the state is adjusted accordingly to ensure its stability.

Based on both local and global asymptotic states, the equilibrium is determined when assessing cell stability. Define the equilibrium as E. Next, the measurements for the local and global parameters are as follows:

$$E^0 = P_0, V_0, I_0 \tag{9}$$

$$E^1 = \bar{P_0}, \bar{V_0}, \bar{I_0} \tag{10}$$

Whereas the cell population is determined by

$$P^0 = (\frac{P_{max}}{2t})(t - d + \sqrt{(t-d)^2 + \frac{4ts}{P_{max}}}) \tag{11}$$

$$\bar{P_0} = \frac{c(\delta+\rho)}{\beta\rho} \tag{12}$$

$$\bar{V_0} = (\frac{1}{\delta})[s - d\bar{P_0} + a\bar{P_0}(1 - \frac{\bar{P_0}}{P_{max}})] \tag{13}$$

$$\bar{I_0} = (\frac{q}{c})\bar{V_0} \tag{14}$$

Let us suppose the relation R, where the base stability ratio for the relation is $\frac{P_0}{\bar{P_0}}$. In stage two, the freshly formed cells are observed and monitored using R. The ratio of non-infected cells to infected cells is determined by such observation and monitoring. Using the relation R, the change ratio is assessed. It calculates the total infection period by dividing the initial state by the final state's stability equilibrium change ratio.

Using the differential equation, the global bifurcation is measured, and the findings are obtained based on the observation.

Lemma1: Let the relation R $<$ i, $E_0 = P_0, V_0, I_0$ is set to be locally stable and when R $>$ 1, $E_0 = P_0, V_0, I_0$ is set to be globally stable.

Lemma2: Assume that the global and local variables are in a balanced form. P(t), V(t), and I(t), where t is the temporal percentage that varies between the initial and final state variables, are totally dependent on F $>$ 0.

Taking into account Lemma 1 and Lemma 2, the following supposition is used to construct Lemma 3.

(i) R $>$ 1

(ii) (c+ δ+ρ +d-t+$\frac{2t\bar{P}}{P_{max}}$)

(iii) (-d+t-$\frac{2t\bar{P}}{P_{max}}$)(c+ δ+ρ)+ $\beta\bar{I}(c+\delta) < 0$

4. Ensuring Delay Metrics in HIV Prediction

R>1 and R<1 are used to evaluate stability, while the DDE model is used to evaluate the disease-free equilibrium.

Lemma 1: The percentage of non-infected cells that become infected cells is known as the conversion rate, and this rate is used to predict the disease. R<1 is used to measure asymptotic stability, while R>1 is the unstable equilibrium.

To validate the aforementioned prediction, a few negative solutions with the characteristics that follow will be implemented.

$$\partial_1 = t - d - 2t\frac{P_0}{P_{max}} \tag{15}$$

The equilibrium E_0 announces the disease identification proportion that provides the eigen solution of the same using equation (15).

$$\partial^2 + (\delta+\rho+c)\partial + c(\delta+\rho) - q\beta P_0 e^{-\partial\tau} = 0 \tag{16}$$

The disease infection equilibrium τ

$$-\omega + i(\delta+\rho+c)\omega + c(\delta+\rho) - q\beta P_0\cos\omega\tau + iq\beta P_0\sin\omega\tau = 0 \tag{17}$$

In order to distinguish between the infected and noninfected cel1s, it is indicated by

$$-\omega^2 + c(\delta+\rho) = q\beta P_0\cos\omega\tau \tag{18}$$

$$(\delta+\rho+c)\omega = -q\beta P_0\sin\omega\tau \tag{19}$$

Squaring on both the equation (18) and (19) that results equation (20)

$$-\omega^4 + [(\delta+\rho+c)^2 + 2c(\delta+\rho)]\omega^2 + c^2(\delta+\rho)^2 - q^2\beta^2P_0^2 = 0 \tag{20}$$

The following quadratic functions with the following assumptions are the outcome when the results are narrowed down.

$$T_{10} = (\delta+\rho+c)^2 + 2c(\delta+\rho) \tag{21}$$

$$T_{20} = c^2(\delta+\rho)^2 - q^2\beta^2P_0^2 \tag{22}$$

The eqn (20) is rewritten as

$$x^2 + T_{10}x + T_{20} = 0 \tag{23}$$

the quadratic equation's coefficient by squaring T_{10}, which is dependent upon the squaring of T_{20}. The outcomes attained include

$$T_{10} = (\delta+\rho+c)^2+2c(\delta+\rho)>0 \tag{24}$$

$$T_{20} = c^2(\delta+\rho)^2-q^2\beta^2P_0^2 = c(\delta+\rho)[c(\delta+\rho)+\beta P_0][1-R] \tag{25}$$

The positive product of equation (24) is real, complex, and has the same sign. The equation lacks genuine roots with a positive sign, and the negative sign reflects anything that is real or negative. Ultimately, there isn't a negative equilibrium like that. Thus, equation (24) and equation (16) are equivalent.

5. Bifurcation Breakdown

The bifurcation breakdown is regarded as its parameter for assuring and assessing time delay, and it is quantified using the time delay proportion τ. When discussing the delay proposition, it is assumed that the equilibrium E, or stability parameter, has a value of R>1. E is regarded as the stability equilibrium from which the following linear equation is derived.

$$\partial^3+t_1\partial^2+t_2\partial+t_3 = e^{-\partial\tau}(u_1\partial^2+u_2\partial+u_3) \tag{26}$$

The equation's coefficients are shown by

$$t_1 = c+\delta+d-t+\frac{2t\bar{P}}{P_{max}}-\rho \tag{27}$$

$$t_2 = c(\delta+\rho)+(c+\delta+\rho)(d-a+\frac{2t\bar{P}}{P_{max}}-\rho) \tag{28}$$

$$t_3 = c(\delta+\rho)+(d-a+\frac{2t\bar{P}}{P_{max}}-\rho) \tag{29}$$

$$u_1 = -\beta\bar{I} \tag{30}$$

$$u_2 = -\beta\bar{I}(c+\delta) \tag{31}$$

$$u_2 = -\beta\bar{I}(c\delta) \tag{32}$$

Assuming $\tau=0$, the characteristics align with the DDE equation, as demonstrated by Lemma 1's assertion that all eigen values satisfy negative equilibrium. When equilibrium is reached through local stability, the asymptotic stability satisfies $\tau=0$. Based on equilibrium stability, a non-negative solution is not balanced when $\tau>0$. Thus, in order to support overall equilibrium,

$t_1 > 0, t_2 > 0, t_3 > 0$ will be followed by $u_1 > 0, u_2 > 0, u_3 > 0$. The results demonstrate that the result cannot be zero or negative, leading to the conclusion that the outcome must be non-negative to maintain equilibrium.

$$-i\omega^3 - t_1\omega^2 + it_2\omega + t_3 \tag{33}$$

$$\begin{aligned} &-u_1\omega^2\cos\omega\tau + iu_2\omega\cos\omega\tau + u_3\cos\omega\tau - \\ &iu_1\omega^2\sin\omega\tau - u_2\omega\sin\omega\tau + iu_3\sin\omega\tau \end{aligned} \tag{34}$$

When the real and imaginary parts of the equation are separated, it is derived by

$$t_3 - t_1\omega^2 = (u_3 - -u_1\omega^2)\cos\omega\tau - u_2\omega\sin\omega\tau \tag{35}$$

$$t_2\omega - \omega^3 = u_2\omega\cos\omega\tau + (u_3 - u_1\omega^2)\sin\omega\tau \tag{36}$$

The equation is transformed into a trigonometry function by squaring both sides, producing

$$\omega^6 + (t_1^2 - 2t_2 - u_1^2)\omega^4 + (t_2^2 - 2t_1t_3 + 2u_1u_3 - u_3^2)\omega^2 + t_3^2 - u_3^2 = 0 \tag{37}$$

Assuming that z = ω^2, the even power of ω is and the third order of equation is

$$z^3 + m_1z^2 + m_2z + m_3 = 0 \tag{38}$$

where

$$m_1 = t_1^2 - 2t_2 - u_1^2 \tag{39}$$

$$m_2 = t_2^2 - 2t_1t_3 + 2u_1u_3 - u_3^2 \tag{40}$$

$$m_3 = t_3^2 - u_3^2 \tag{41}$$

By squaring ω, which does not yield a positive imaginary solution, equation (37) has no negative solutions. Lemma below supports the outcomes described before and satisfies the condition

Lemma 1: The positivity of the real roots is verified under the condition that m_1, m_2, and m_3 >= 0

Let us assume

$$z = h(z) = z^3 + m_1z^2 + m_2z + m_3 = 0 \tag{42}$$

Then the derivative with respect to z proves that $h(z) = h^{'}(z)$. As z>0 then h>0 and hence prove that $h^{'}(z)$ which is also > 0. Therefore

$$h^{'}(z) \quad = \quad 3z^2 + 2m_1 z + m \tag{43}$$

where the lemma is proved since h(z) has no positive real roots.

The aforementioned lemma establishes the absence of ω in the case where $i\omega$ is considered the eigen value. Thus, it validates the thesis that states that when $\tau \geq 0$, all real eigen values are negative. The following assumption results from the consolidation of the aforementioned theorems and conclusions.

Assume that

$a) m_1, m_2, m_3 >= 0$

$b) R_0 > 1$

According to the first two assumptions, genuine positive values are guaranteed, and equilibrium stability is ensured. E is asymptotically stable based on delay $\tau \geq 0$.

When there is a delay, the assumption tends to meet both a) and b), where E is the equilibrium, and such a delay has no effect on the asymptotic stability because delay is guaranteed by $\geq$0.

Assume that the delay is measured and tends to change, failing to satisfy the requirement $\tau \geq 0$, if conditions a) and b) are not stable and do not respond to stability. When there is instability, the outcomes often fluctuate.

Based on the restriction $\lim_{z \to \infty} h(z) = \infty$, the value of h(0) is likewise < 0 when $m_3 < 0$. This is because, in order to obtain the least positive root from the equation (37), the resultant becomes $\omega = \sqrt{z}$.

In this case, the outcomes with the least positive root are proven through the introduction of bifurcation analysis.

Then the equation based on the stability condition it referred to

$$\frac{d}{dt}(Re\lambda(P))_{P=P_0} \quad > \quad 0 \tag{44}$$

Lemma 2: While considering we is the largest positive root then the equation is balanced with $i\omega(\tau_0) = i\omega_0$ satisfying the assumption $\partial(\tau) + i\omega(\tau)$ is differentiated with τ as neighbourhood of τ_0 .

After analysis of the above differential equation by assuming h_1, h_2 and h_3 are considered as the root of the equation then $f(h) = h^3 + m_1 h^2 + m_2 h = 0$ with $(m_2 < 0)$ and h_3 is the highest positive value.

$$(\frac{dh(x)}{dt} \quad > \quad 0) \tag{45}$$

Hence the above equation proof is omitted.

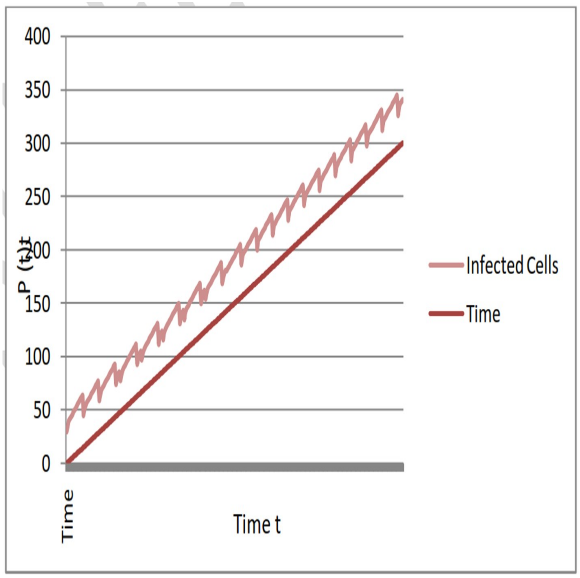

FIGURE 1. HIV virus infection rate vs time

The time correlation difference between the illness spread based on time is displayed in Figure 1. These steps will help counter the non-infection and infection rates over time by assuming the total number of virus cells.

As many models fail to address two parameters as valid aspects against disease wide spread, Figure 2 provides a depiction of the spread of the disease without adopting an overall mathematical model that suggests the virus growth rate with respect to acceleration and deceleration. The entire population is represented by the x and y axes, and is used to assess how widely the virus is spreading in comparison to its own spread.

Based on the evaluation of the overall population and the increasing virus spreading ratio, Fig. 3 displays the predicted HIV growth spread.

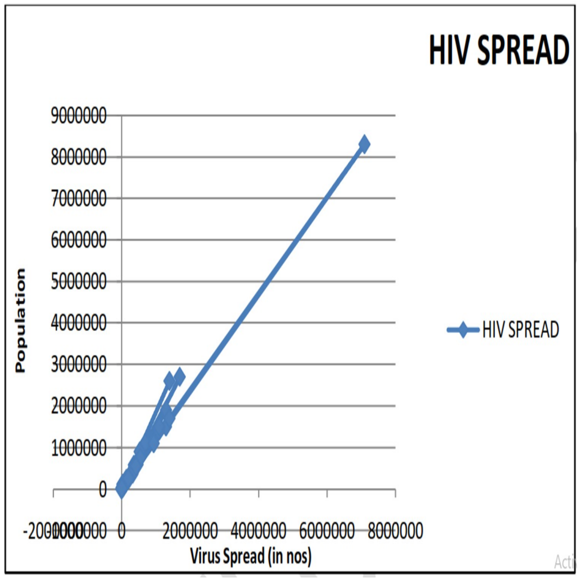

FIGURE 2. Disease spread ratio

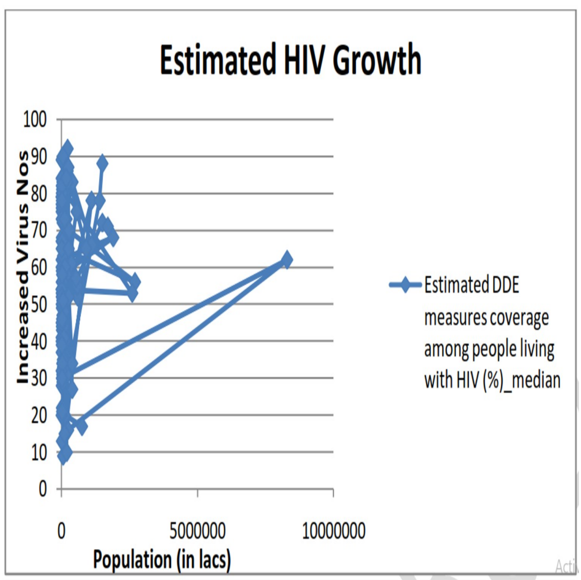

FIGURE 3. HIV growth against population and infection

The infection ratio is determined by calculating it against a particular population and then using the DDE mathematical model to address the rate of infection growth while taking into account the equilibrium of virus stability and ignoring the possibility of negative outcomes from growing virus volumes.

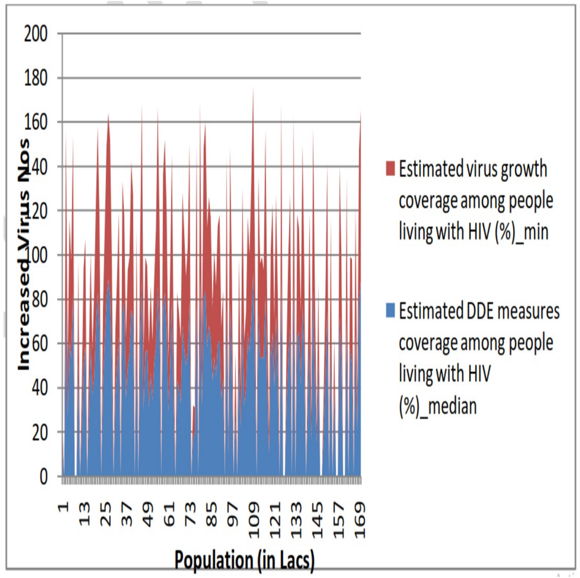

FIGURE 4. Min and Max virus growth rate

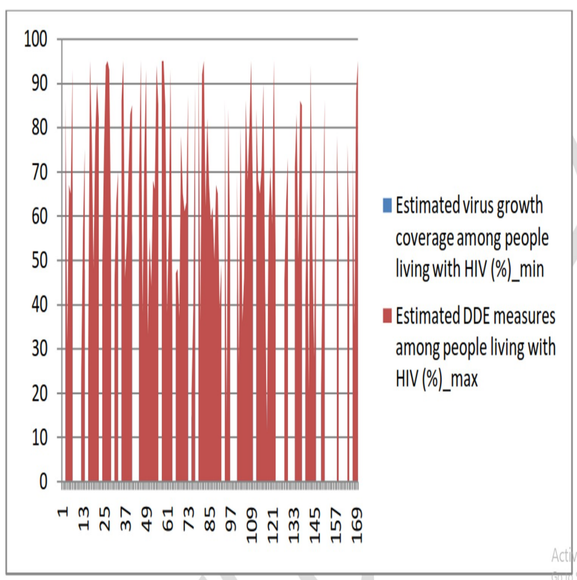

FIGURE 5. Virus asymptotic growth rate

The Minimum and Maximum growth rates, as well as a clear explanation of the growth stability study addressing the asymptotic phases of the viral spread and its classification, are provided in Figure 4. This ensures that the virus grows at a faster pace than the population as a whole.

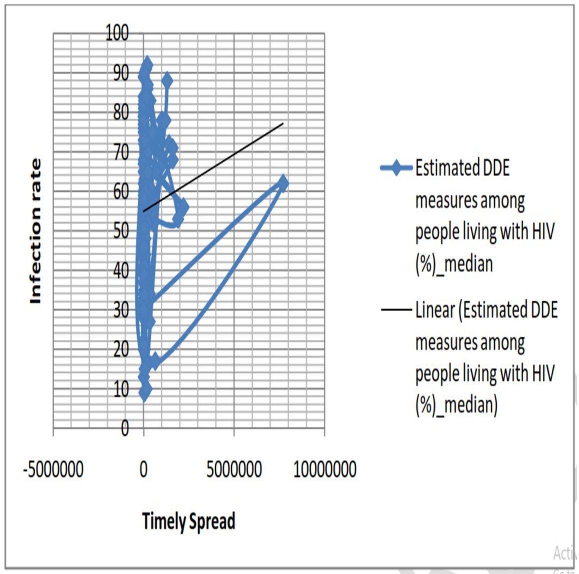

FIGURE 6. DDE HIV virus stability

The asymptotic stability rate of the viral growth versus population expansion is shown in Figure 5. For Vim's asymptotic growth rate, the results offer a trade-off analysis between virus growth and DDE model analysis, with the goal of maintaining maximum stability. When employing the DDE model, asymptotic stability analysis is used to establish stability based on the virus's growth against its expanding population. Asymptotic stability checks for maximal positive are used to guarantee the relationship, which provides constant virus growth and a clear analysis of its growth stability.

The stability comparison of a virus with an increasing population over time and an infection rate is shown in Figure 6. The stability is guaranteed against the spread of viruses against the population when these two coefficients are incorporated into the DDE model.

The stability against both the maximum and minimum rates of virus propagation is shown in Figure 7, and asymptotic stability is guaranteed based on the estimated growth and population size.

Bifurcation analysis is shown in Figure 8 to ensure stability by meeting equation (37). By assessing the viral population growth using the breakdown of the bifurcation analysis, the stability condition of the results is thus concluded.

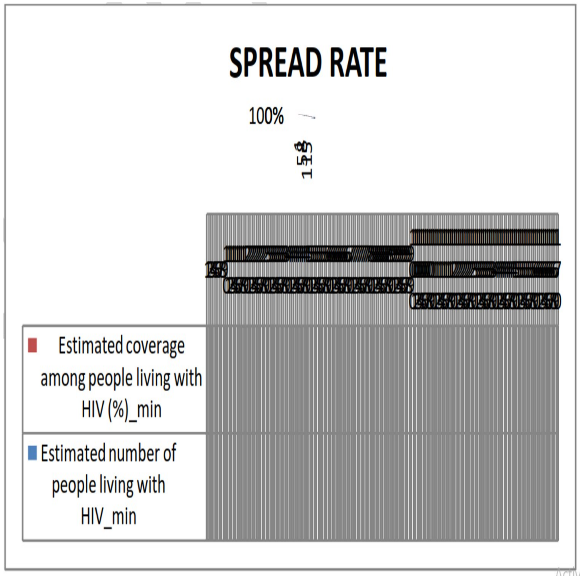

FIGURE 7. Virus Spread Rate with its Stability

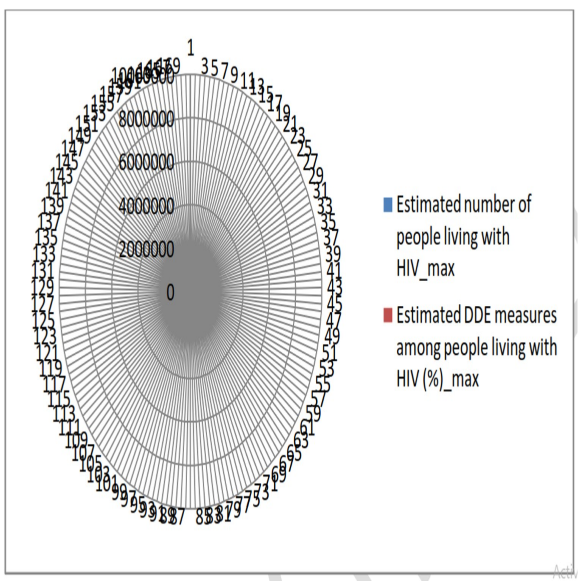

FIGURE 8. Bifurcation Breakdown

6. CONCLUSION

In conclusion, this research article delves into the crucial aspect of stabilizing HIV viral growth through the utilization of DDE's bifurcation parameter. The primary focus is on creating a steady state that not only prevents the occurrence of an overall delay but also guarantees stability in the growth rate of the virus. By incorporating both local and global characteristics, the study aims to establish equilibrium conditions that ensure not only the stability of the system but also a disease-free state.

One of the key contributions of this research lies in its exploration of how DDE's bifurcation parameter can effectively manage the dynamics of HIV viral growth. Bifurcation analysis is a powerful tool in understanding the behavior of complex systems, and in the context of this study, it proves instrumental in achieving stability without compromising the overall growth rate. The utilization of DDE's bifurcation parameter allows for a nuanced examination of the system's behavior, enabling researchers to identify and address potential instabilities.

The study emphasizes the importance of considering both local and global characteristics when analyzing the stability of HIV viral growth. Local characteristics provide insights into the immediate environment surrounding the virus, while global characteristics offer a broader perspective, taking into account the interactions and influences that extend beyond immediate surroundings. By comprehensively accounting for these factors, the research ensures a more holistic approach to understanding and managing HIV dynamics.

The establishment of equilibrium conditions is a pivotal aspect of the research findings. Achieving equilibrium is critical in maintaining stability in the system. The study not only identifies equilibrium points but also demonstrates their effectiveness in ensuring overall stability. The emphasis on both local and global characteristics in determining equilibrium conditions further enhances the robustness of the proposed model.

Furthermore, the outcomes of the research guarantee global stability, which is a significant achievement in the context of HIV viral growth management. Global stability implies that the system remains stable across a range of conditions, reinforcing the resilience of the proposed approach. The attainment of a disease-free equilibrium is another noteworthy outcome, signifying the potential to control and manage the virus without the occurrence of new infections.

A distinctive aspect of the research is its exploration of asymptotic bifurcation analysis and its implications. The failure of asymptotic bifurcation analysis, as indicated by the study, sheds light on the limitations and challenges associated with certain analytical approaches. This recognition of the method's shortcomings is valuable for future research, guiding scholars towards more effective tools and methodologies in understanding and managing the complexities of HIV dynamics.

In summary, this research article significantly contributes to the field by offering a comprehensive analysis of stabilizing HIV viral growth through the innovative use of DDE's bifurcation parameter. The study's emphasis on both local and global characteristics, the establishment of equilibrium conditions, and the guarantee of global stability and disease-free equilibrium collectively position it as a valuable resource in advancing our understanding of HIV dynamics and exploring effective strategies for its management. The acknowledgment of the limitations of asymptotic bifurcation analysis further underscores the need for ongoing research and the development of more robust analytical tools in the quest for effective solutions to the challenges posed by HIV.

Conflict of Interests

The authors declare that there is no conflict of interests.

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