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Mathematical Modelling and Analyzing the Dynamics of Condom Efficacy and Compliance in the Spread of HIV/AIDS

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

The study involves a mathematical analysis of condom efficacy and compliance in the transmission of HIV/AIDS. It explores how condom use impacts the prevention of HIV/AIDS and considers other epidemiological factors affecting the progression from HIV to full-blown AIDS. The model's

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existence and uniqueness of solution are established, with a focus on the basic reproduction number, representing the average new infections caused by an infected individual. The analysis reveals that the disease-free equilibrium is stable when the basic reproduction number is below unity i.e. $(R_0 < 1)$, but becomes endemic otherwise, i.e. $(R_0 > 1)$. Sensitivity analysis indicates parameters with positive values increase the reproduction number, emphasizing that the effective contact rate should not exceed 0.22 to avoid the endemic stage. Numerical simulations using MAPLE 18 software demonstrate that condom compliance reduces the dynamic spread of HIV, and targeted immunity boost controls the viral load.

Keywords: HIV/AIDS; treatment; condom efficiency; condom compliance; basic reproduction number and equilibrium points.

1. INTRODUCTION

"Human Immunodeficiency Virus (HIV) is a virus that targets the body's immune system, particularly the CD4-positive cells" [1-3]. "There were an estimated 39.0 million (33.1–45.7 million) people living with HIV at the end of 2022, two thirds of whom (25.6 million) are in the WHO African Region" [4]. Acquired Immunodeficiency Syndrome (AIDS) poses a significant risk to a substantial portion of the global population, impacting not only individuals infected with the virus but also their families and friends [5,6]. Global HIV estimates have been compiled by the Joint World Health Organization (WHO) and the United Nations Program on HIV/AIDS (UNAIDS) since the late 1980s. The identification of the first AIDS patient occurred in 1981, marking the beginning of the classification of AIDS as a global pandemic[7-9]. The initial stages of HIV infection are characterized by symptoms and signs such as flu-like symptoms, night sweats, cough, weight loss, headaches, diarrhea, sunburn-like rash, body aches, joint pain, and tonsillitis. In these early phases, the virus has a higher viral load in the bloodstream, making the spread of HIV infections more efficient throughout the body. HIV is transmitted through various body fluids (blood, tears, urine, saliva, etc.) and can infect an uninfected individual [4,10]. CD4-positive cells are crucial for fighting infections and play a significant role in modifying the immune system. Any disruption or reduction in the precision of these CD4-positive cells can have wide-ranging consequences, leading to the impairment of the immune system's functioning. The retention time of these lymphocytes is critical for maintaining a healthy immune response [5,11-13].Many researchers have worked on the dynamical spread of HIV/ AIDS using various assumptions.

In [14], a mathematical model to investigate the transmission dynamics of HIV in Nigeria is introduced, addressing a unique aspect by partitioning detected individuals into those receiving treatment and those not accessing treatment. This consideration, which has been absent in recent literature, adds depth to the understanding of HIV dynamics. The study's findings emphasize the pivotal role played by the fraction of detected individuals receiving treatment, affecting the population of latently-infected individuals and the AIDS class. The treatment's impact is highlighted as it hinders the progression of individuals into the AIDS class.

Work in [15] presented and analyzed "five nonlinear differential compartmental models to gain a deeper understanding of the parameters influencing the dynamic spread of HIV in society. The study involved numerical simulations to assess the effects of various parameters on the dynamic spread of the disease. The effective contact rate and the presence of fast progressors emerged as the primary key parameters that significantly influenced the dynamic spread of HIV in the community".

A mathematical model for the transmission of HIV/AIDS with early treatment was developed and analyzed by Akinwumi et al. [16]. The study calculated the basic reproduction number, a measure of the average new infections caused by an infected individual. Findings suggested that the disease-free equilibrium is stable when this number is below one. Numerical analysis showed that early treatment of latent infections decreases progression to AIDS. Additionally, substances enhancing immunity increased red blood cells. Sensitivity analysis indicated that the effective contact rate should stay below 0.3 to prevent the endemic stage.

Some other researchers that developed various variations of the HIV/AIDS model are outlined in [17-28].

In the realm of contemporary literature, a notable portion has tended to neglect the crucial aspect of condom efficacy and compliance when utilizing mathematical epidemic models for HIV/AIDS. What distinguishes this study is its specific focus on the efficiency and compliance to condom use as crucial factors in preventing the spread of HIV. This distinctive addition enhances the model's capacity to account for condom efficacy, providing a more comprehensive and insightful approach to the analysis of epidemics.

The study extends the work in Akinwumi et al. [16] by integrating condom efficacy and compliance considerations into a Five-compartment mathematical model for HIV/AIDS epidemics. This model, emphasizing condom efficiency and adherence, undergoes thorough analysis to enhance understanding of potential mitigation strategies. By addressing the often-overlooked variables of condom use, the research aims to provide a more realistic portrayal of dynamics in HIV spread and prevention. The inclusion of these factors in the model lays the groundwork for a comprehensive exploration of strategies, contributing to advancements in our understanding and approach to HIV/AIDS mitigation.

The paper follows the following structure: Section 2 outlines the methodology, encompassing the design and formulation of the model, along with its analysis. Sections 3 are devoted to presenting the results and engaging in a subsequent discussion of those findings. The paper is wrapped up with Section 4, which serves as the conclusion.

2. MATERIALS AND METHODS

In this research, the work done by Akinwumiet al. [16] is modified by incorporating condom efficiency and compliance on the dynamical spread of the disease HIV/AIDS.

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The population size $N(t)$ of human is sub–divided into sub–classes of individuals who are Susceptible $S(t)$, Latently Infected $Lt)$, Infected $I(t)$, Treated $T(t)$ and HIV/AIDS $A(t)$, So that;

$$N(t) = S(t) + L(t) + I(t) + T(t) + A(t) \quad (1)$$

The susceptible population is increased by the recruitment of individuals into the population either by birth or immigration at the rate (Λ). The population decreases by the newly infected individuals that move to latently infected class with condom efficacy and compliance at the rate $(1-\omega\alpha)$. The population also decreases by natural death at the rate (μ). Thus;

$$\frac{ds}{dt} = \Lambda - (1-\omega\alpha)\beta SI - \mu S \quad (2)$$

The population of the latently infected class consist newly infected individuals with the condom efficacy and compliancefollowing a contact with the infected human/object at the rate (β). The population later decreases due to progression to infectious class at the rate (κ), natural and disease induced death at the rate (μ) and (δ) respectively, also decreases due to early treatment at the rate (σ_1) .The population later increased by the help of immunity boosted from treated compartment when the condom used rise above 50%. Thus;

$$\frac{dL}{dt} = (1-\omega\alpha)\beta SI - (\kappa + \mu + \delta + \sigma_1)L + \theta T \quad (3)$$

The population of infected individuals increases by progression from latently infected individual, due to lack of treatment or treatment failure at the rate (κ). The population decreases due to treatment at the rate (σ_2), natural death at the rate (μ) and disease induced death at the rate (δ). Thus;

$$\frac{dI}{dt} = \kappa L - (\mu + \sigma_2 + \delta)I \quad (4)$$

The population of the treated individuals increases by the treatments of those that are latently and fully infected by HIV at the rate (σ_1) and (σ_2). The population decreases due to

natural death at the rate (μ), death due to the disease at the rate (δ), treatment failure due to drug resistance or inadequate dosing at the rate α and the immunity boosted after treatment and CD4T cell rises above 50% at the rate (θ). Then,

$$\frac{dT}{dt} = \sigma_1 L + \sigma_2 I - (\mu + \delta + \theta + \gamma)T \quad (5)$$

Full blown AIDS compartment increases by treated individuals that failed treatment due to one medical reason or the other at the rate α. The acquire immuno-deficiency syndrome individuals suffer natural death and death due to the disease at the rate (μ) and (δ) respectively. Hence;

$$\frac{dA}{dt} = \gamma T - (\mu + \delta)A \quad (6)$$

In summary, the following system of differential equation is proposed;

$$\begin{aligned}
\frac{ds}{dt} &= \Lambda - (1-\omega\alpha)\beta SI - \mu S \\
\frac{dL}{dt} &= (1-\omega\alpha)\beta SI - (\kappa + \mu + \delta + \sigma_1)L + \theta T \\
\frac{dI}{dt} &= \kappa L - (\mu + \sigma_2 + \delta)I \\
\frac{dT}{dt} &= \sigma_1 L + \sigma_2 I - (\mu + \delta + \theta + \gamma)T \\
\frac{dA}{dt} &= \gamma T - (\mu + \delta)A
\end{aligned} \quad (7)$$

For simplification, equation (7) becomes

$$\begin{aligned}
\frac{ds}{dt} &= \Lambda - (1-\omega\alpha)\beta SI - \mu S \\
\frac{dL}{dt} &= (1-\omega\alpha)\beta SI - C_1 L + \theta T \\
\frac{dI}{dt} &= \kappa L - C_2 I \\
\frac{dT}{dt} &= \sigma_1 L + \sigma_2 I - C_3 T \\
\frac{dA}{dt} &= \gamma T - C_4 A
\end{aligned} \quad (8)$$

Where;

$$C_1 = (\kappa + \mu + \delta + \sigma_1),\ C_2 = (\mu + \sigma_2 + \delta),$$
$$C_3 = (\mu + \delta + \theta + \gamma),\ C_4 = (\mu + \delta)$$

Theorem 1: The closed set $D = \left\{ (S, L, I, T, A) \in R_+^5 : N \le \frac{\Lambda}{\mu} \right\}$ is positively-invariant with non-negative initial values in R_+^5

Proof: Consider the feasible region D as defined above, then the rate of change of the total population is given by;

$$\frac{dN}{dt} = \Lambda - \mu N$$

It follows that $\frac{dN}{dt} \le \Lambda - \mu N$. Hence, if $N(0) \le \frac{\Lambda}{\mu}$, then $N(t) \le \frac{\Lambda}{\mu}$. Therefore, all solutions of the model with initial values in D remain in D for all time t > 0 and this implies that D is positively invariant.

2.1 Disease free Equilibrium Point

$$E_0^* = (S^* L^* I^* T^* A^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right)$$

2.2 The Basic Reproduction Number (R_o)

The Basic Reproduction Number (R_0) is a key epidemiological indicator quantifying the transmissibility of an infection. It represents the average number of new infections transmitted by an infected individual during the infectious period. $R_0 < 1$, suggests the disease will die off, $R_0 = 1$, indicates the presence of endemic, and $R_0 > 1$, implies potential for exponential growth and pandemic spread. Calculating R_0 is crucial for understanding and managing infectious diseases, informing public health strategies[17,18,29,30,26,31-34].

The Basic Reproduction Number (R_0) is given as $R_0 = \rho(FV^{-1})$

Given the matrices F and V below,

$$F=\begin{bmatrix} 0 & (1-\omega\alpha)\beta & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (9)$$

$$V=\begin{bmatrix} C_1 & 0 & -\theta & 0 \\ -\kappa & C_2 & 0 & 0 \\ -\sigma_1 & -\sigma_2 & C_3 & O \\ 0 & 0 & -\gamma & C_4 \end{bmatrix} \quad (10)$$

$$(FV^{-1})=\begin{pmatrix} 0 \\ 0 \\ 0 \\ -\dfrac{(-1+\omega\alpha)\beta\kappa C_3}{C_1C_2C_3-C_2\theta\sigma_1-k\theta\sigma_2} \end{pmatrix} \quad (11)$$

The ρ obtained by FV^{-1} which is known as basic reproduction number is given below

$$R_0=\frac{(\omega\alpha-1)\beta\kappa C_3\Lambda}{\mu(C_1C_2C_3-C_2\theta\sigma_1-k\theta\sigma_2)} \quad (12)$$

2.3 Local Stability of DiseaseFree Equilibrium

Theorem 2: The disease free equilibrium is locally asymptotically stable (LAS) if R_0< 1 and unstable if R_0>1.

Proof: To determine the local stability of E_0, the Jacobian matrix below is computed corresponding to Disease Free Equilibrium E_0. Considering the

$$J_1(E_0)=\begin{bmatrix} -\mu & 0 & \dfrac{-(1-\omega\alpha)\beta\Lambda}{\mu} & 0 & 0 \\ 0 & -C_1 & \dfrac{(1-\omega\alpha)\beta\Lambda}{\mu} & \theta & 0 \\ 0 & \kappa & C_2 & 0 & 0 \\ 0 & \sigma_1 & \sigma_2 & -C_3 & 0 \\ 0 & 0 & 0 & \gamma & -C_4 \end{bmatrix} \quad (13)$$

Since the first and the fifth column of the equation (13) have only the diagonal term that form the first two negative Eigen values i.e. $-\mu$ and $-C_4$, hence we have;

$$J_2(E_0)=\begin{bmatrix} -C_1-\lambda & \dfrac{(1-\omega\alpha)\beta\Lambda}{\mu} & \theta \\ \kappa & -C_2-\lambda & 0 \\ \sigma_1 & \sigma_2 & -C_3-\lambda \end{bmatrix}=0 \quad (14)$$

We obtained the Eigen values of (14) from the characteristics equation below;

$$A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0 \quad (15)$$

Where

$$A_3=1$$
$$A_2=C_1+C_2+C_3$$
$$A_1=\frac{\beta\omega\alpha\kappa\Lambda-\beta\kappa\Lambda-\mu\theta\sigma_1+\mu C_1C_2+\mu C_1C_3+\mu C_2C_3}{\mu}$$
$$A_0=\frac{-(\omega\alpha-1)\beta\kappa C_3\Lambda+\mu C_1C_2C_3-\mu C_2\theta\sigma_1-\mu\kappa\theta\sigma_2}{\mu}$$

From A_0;

$$\frac{-(\omega\alpha-1)\beta\kappa C_3\Lambda}{\mu}+(C_1C_2C_3-C_2\theta\sigma_1-\kappa\theta\sigma_2)>0$$

$$\frac{-(\omega\alpha-1)\beta\kappa C_3\Lambda}{\mu}>-(C_1C_2C_3-C_2\theta\sigma_1-\kappa\theta\sigma_2)$$

$$\frac{(\omega\alpha-1)\beta\kappa C_3\Lambda}{\mu(C_1C_2C_3-C_2\theta\sigma_1-\kappa\theta\sigma_2)}<1$$

Hence, $R_0<1$

According to Routh Hurwitz criterion, which states that all the roots of the polynomial will have negative real parts if and only if all the coefficients A_i(i=0, 1, 2, 3) are all positive and that the matrices

T_i(i=1, 2, 3) are all positive. Clearly from (13) $A_3>0, A_2>0, A_1>0$ and $A_0>0$ if $R_0>1$ Also, the Hurwitz matrix T_i is all positive which are given as below;

$T_i=A_2>0$, $T_2=\begin{bmatrix} A_2 & A_3 \\ A_0 & A_1 \end{bmatrix} > 0$,

$T_3=\begin{bmatrix} A_2 & A_3 & 0 \\ A_0 & A_1 & A_2 \\ 0 & 0 & A_0 \end{bmatrix} > 0$

Therefore, all the eigenvalues of the matrix (13) are negative which shows that the disease free equilibrium is locally asymptotically stable.

2.4 Global Stability of Disease Free Equilibrium

Theorem 2: If $R_0 \leq 1$, then the disease free equilibrium ε_0 of the system (8) is globally asymptotically stable (GAS).

Proof: To establish the global stability of the disease free equilibrium ε_0, we construct the following Lyapunov function;

$$V = AL + BI + CT \tag{16}$$

$$V = -C_3 L + \left(\frac{C_3 C_1 - \theta\sigma_1}{\kappa} \right) I + \theta T \tag{17}$$

Calculating the derivative of V along the solution of the proposed system, we obtain

$$V^I = -C_3 L^I + \left(\frac{C_3 C_1 - \theta\sigma_1}{\kappa} \right) I^I + \theta T^I \tag{18}$$

$$V^I = \left(C_3 \beta \frac{\Lambda}{\mu}(\omega\alpha - 1) - \frac{C_1 C_2 C_3}{\kappa} + \frac{C_2 \theta\sigma_1}{\kappa} + \theta\sigma_2 \right) I \tag{19}$$

Since $S = \frac{\Lambda}{\mu} \leq N$

Little perturbation from (19) with the basic reproduction number (12) gives;

$$V^I = [R_0 - 1](C_1 C_2 C_3 - C_2 \theta\sigma_1 - \kappa\theta\sigma_2) I \tag{20}$$

We see that

$V^I \leq 0$, for $R_0 < 1$.

If $R_0 < 1$ then $V^I = 0 \Leftrightarrow I = 0$.

Therefore by LaSalle's invariance principle (1987), the disease free equilibrium is globally asymptotically stable if $R_0 \leq 1$ [14].

2.5 Existence of Endemic Equilibrium

Here we analyse the condition for the existence of equilibrium for which the HIV/AIDS disease is endemic in the population. If we consider the model equation (8)

Where $\varepsilon_0^* = (S^* L^* I^* T^* A^*)$ are the respective endemic equilibrium points.

$$S^* = \frac{\Lambda C_1 C_2 C_3 - \Lambda(C_2 + \kappa\sigma_2)\theta}{C_3 \kappa\mu\Lambda - 2(C_2 - \kappa\sigma_2)\theta\mu + C_1 C_2 C_3} \tag{21}$$

$$L^* = \frac{C_2}{\kappa}\left(\frac{C_3 \kappa\eta\Lambda - \theta C_2 \mu - \theta\kappa\sigma_2\mu}{C_1 C_2 C_3 \mu - \theta C_2 \mu - \theta\kappa\sigma_2\mu} \right) \tag{22}$$

$$I^* = \frac{C_3 \kappa\eta\Lambda - \theta C_2 \mu - \theta\kappa\sigma_2\mu}{(C_1 C_2 C_3 \mu - \theta C_2 \mu - \theta\kappa\sigma_2\mu)} \tag{23}$$

$$T^* = \frac{C_2 C_3 \mu\kappa\Lambda - \theta C_2 \mu - \theta\kappa\sigma_2\mu + \kappa\sigma_2}{C_3 \kappa(C_1 C_2 C_3 \mu - \theta C_2 \mu - \theta\kappa\sigma_2\mu)} \tag{24}$$

For which $(C_3 \kappa\eta\Lambda - \theta C_2 \mu - \theta\kappa\sigma_2\mu) > 0$

To have a positive equilibria in the domain.
The endemic equilibrium point of the HIV/AIDS model equation (8) exists whenever the threshold quantity R_0 >1.

2.6 Sensitivity Analysis

Sensitivity analysis investigates the relationship between a model's parameters and the critical threshold, the basic reproduction number R_0, which determines disease spread or eradication in a community. It is widely used for tasks like research prioritization and identifying influential parameters in biological system models. The analysis involves calculating partial derivatives of R_0 with respect to its parameters, providing insights into the impact of each parameter on the dynamics of disease transmission.

$$p; \mathrm{X}_{\mathrm{P}}^{\mathrm{R}_0} = \frac{\partial R_0}{p} * \frac{p}{R_0} \tag{25}$$

The results of the sensitivity indices of R_0 are as shown in the Table2.
Sensitivity analysis is a vital tool in epidemiological modeling, helping to assess the impact of variations in model parameters on key outcomes. Parameters with positive and negative indexes are particularly scrutinized during this analysis.

Parameters with positive indexes, such as $\Lambda, \beta, \kappa, \gamma$ are examined to understand their influence on the model's outputs. An increase in these parameters beyond certain thresholds may signify a heightened risk of undesirable outcomes, such as the transition to an endemic stage or increased disease transmission. Identifying such critical parameters informs targeted interventions and preventive strategies.

On the other hand, parameters with negative indexes i.e. $\mu, \delta, \omega, \theta, \sigma_1, \sigma_2, \alpha$ are equally significant. These parameters, when altered, may have a mitigating effect on the spread of the disease. Sensitivity analysis helps recognize factors that, when manipulated in a favorable direction, can contribute to disease control and prevention.

By systematically exploring the sensitivity of both positively and negatively indexed parameters, researchers can refine strategies for disease management. This analytical approach is essential for improving the accuracy and reliability of epidemiological models, aiding in effective public health decision-making and response planning [24].

2.7 Numerical Simulation

The numerical behaviour of equation (8) is studied using MAPLE 18 software with parameters values is presented in the Table 1.

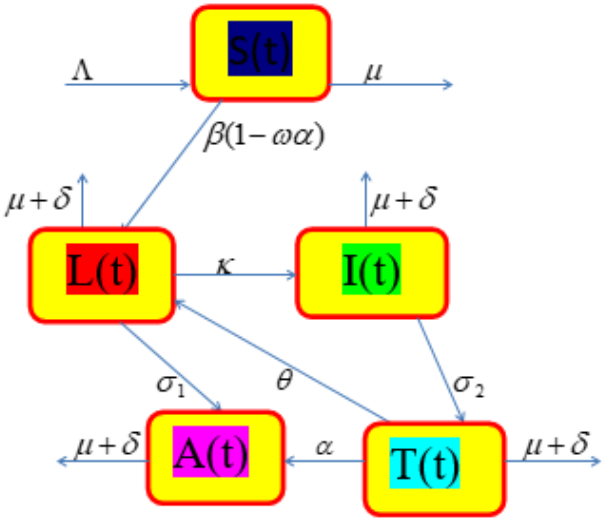

Fig. 1. Flow Diagram of HIV/AIDS

Table 1. Description of parameters with values

Parameter	Descriptions	Values	Source
Λ	Recruitment into Population	1.0	Assumed
β	Effective Contact Rate	0.2	[16]
μ	Natural Death Rate	0.019	[3]
δ	Disease Death Rate	0.01	[16]
α	Condom Compliance	0.04	Estimated
θ	Immunity Boost	0.1	[16]
κ	Progression Rate	0.068	Assumed
σ_1	Treatment Rate of Latent HIV	0.2	[16]

σ_2	Treatment Rate of active HIV	0.1	[3]
ω	Condom Efficiency	0.04	Estimated
γ	Treatment Failure	0.01	[16]

Table 2. Values and signs of sensitivity index (S. I) of R_0

Parameter	S. I.	Sensitivity values
R_0	Positive	0.9162098083
Λ	Positive	1.00000000
β	Positive	1.00000000
μ	Negative	- 0.576457747
δ	Negative	- 0.303398814
ω	Negative	-0.0016025641
θ	Negative	-0.4428337735
κ	Positive	0.7388980047
σ_1	Negative	- 0.487144319
σ_2	Negative	-0.7011837771
γ	Positive	.1135471214
α	Negative	- 0.001602564

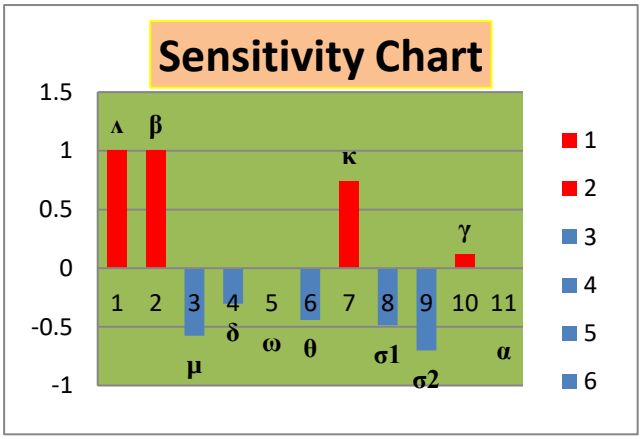

Fig. 2. Sensitivity chart

3. RESULTS

The numerical results of the research are presented in the graphs.

4. DISCUSSION

A five-compartmental mathematical model is presented to analyze the efficacy of condoms in the dynamic transmission of HIV/AIDS. The study aims to understand the impact of condom compliance and effective usage on preventing HIV and the progression to full-blown AIDS. The analysis includes an examination of the basic reproduction number (R_0), a determinant of disease spread or decline. Results indicate that the disease diminishes when R_0 is less than unity but spreads when R_0 exceeds one. Sensitivity analysis plays a pivotal role in identifying critical parameters within epidemic models. Specifically, parameters with a positive index, like the effective contact rate, are scrutinized. If the effective contact rate surpasses a threshold, such as 0.22, there is a heightened risk of transitioning to an endemic stage. This underscores the importance of understanding the influence of individual parameters on the overall dynamics of disease transmission. Sensitivity analysis guides the determination of thresholds and informs decision-making for effective control measures to prevent the emergence of endemicity in the population. Numerical analysis illustrates the dynamic behavior of epidemiological parameters within

the model, offering valuable insights for medical practitioners and health policymakers.

Figs. 3-5 examine the impact of compliance and effective condom usage on the dynamic spread of HIV/AIDS. The results highlight that correct and consistent condom usage, coupled with compliance, is a highly effective means of preventing the spread of HIV in society. The findings reveal a direct correlation: the higher the level of individual compliance with condom usage, the lower the incidence and prevalence of HIV. Consistent condom usage not only reduces the spread of HIV but also minimizes viral load among individuals, thereby preventing the progression to the advanced stages of the disease. This underscores the pivotal role of adherence to proper condom practices in mitigating the transmission and impact of HIV/AIDS.

Figs. 6 and 7 illustrate the preventive effect of immunity boosters on the progression of HIV to full-blown AIDS. HIV, which targets immune system T-cells, necessitates additional vitamins and minerals to support the repair of damaged cells in HIV-positive individuals. Immunity boosters play a crucial role in dynamically controlling the course of HIV/AIDS. Fig. 7 specifically demonstrates that when immunity is at its maximum, i.e., when it is full, there is a notable reduction in the progression from HIV to full-blown AIDS. This underscores the significance of maintaining a robust immune system through the use of immunity boosters as a strategic approach to impede the advancement of HIV to more severe stages.

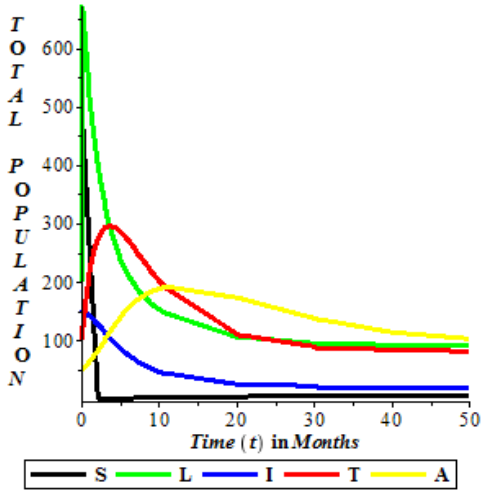

Fig. 3. Total population of SLITA with $\alpha, \omega = 0$

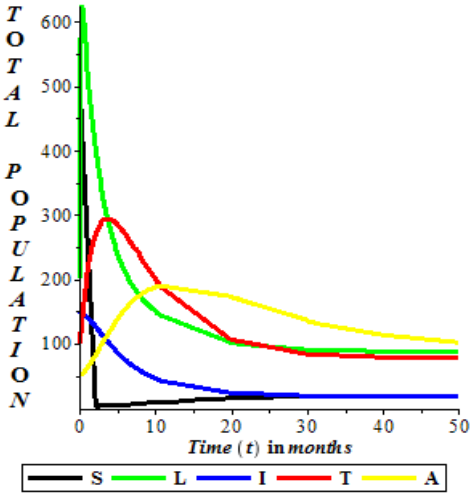

Fig. 4. Total population of SLITA with $\alpha, \omega = 0.5$

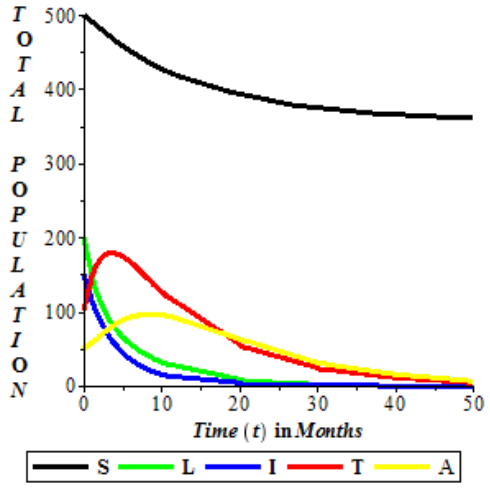

Fig. 5. Total population of SLITA with $\alpha, \omega = 1$

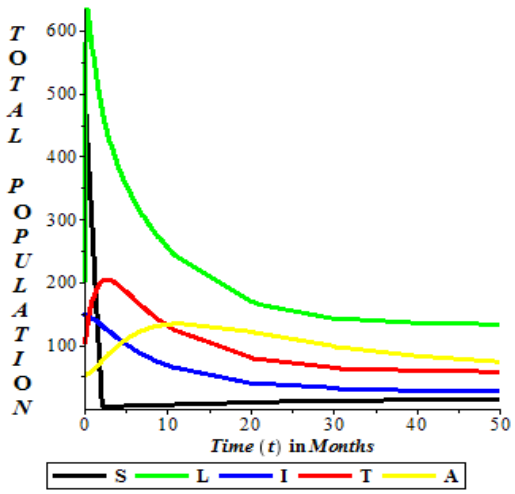

Fig. 6. Total population of SLITA with $\theta = 0.5$

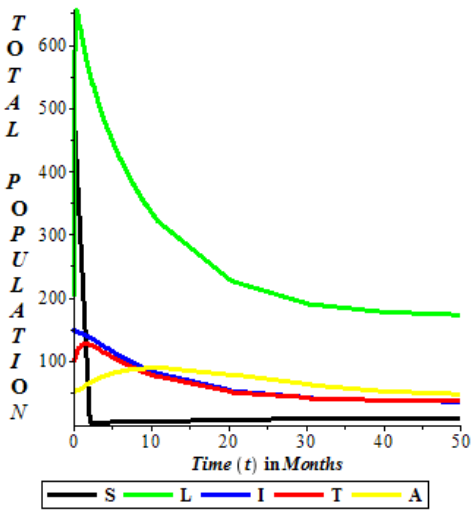

Fig. 7. Total Population of SLITA with $\theta = 1$

5. CONCLUSION

In conclusion, the study underscores the significance of condom compliance and efficiency in mitigating the transmission and spread of HIV. Consistent condom usage during sexual intercourse proves to be more effective than unprotected encounters. Additionally, interventions aimed at boosting immunity are crucial in preventing the progression to full-blown AIDS. To create an HIV/AIDS-free community, widespread encouragement for condom use is essential. Comprehensive efforts, including government-sponsored campaigns, should be implemented to educate both rural and urban populations about the importance of condom usage. Moreover, providing free condoms to citizens, supported by government initiatives, can significantly contribute to promoting safer practices and reducing the prevalence of HIV/AIDS in society.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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