Journal of Advances in Mathematics and Computer Science

28(6): 1-11, 2018; Article no.JAMCS.42819

ISSN: 2456-9968

(Past name: British Journal of Mathematics & Computer Science, Past ISSN: 2231-0851)

Mathematical Modelling of the Role of Interference on the Transmission Dynamics and Management of Hiv and Aids

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Authors' contributions

This work was carried out in collaboration between all authors. Author JMM designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors JKN and BOK managed the analyses of the study. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/JAMCS/2018/42819

Editor(s):

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Complete Peer review History: http://www.sciencedomain.org/review-history/26351

Received: 10 May 2018

Accepted: 28 July 2018

Published: 22 September 2018

Original Research Article

Abstract

In this paper, a deterministic mathematical model incorporating interference is developed and analysed to investigate the role of interference on the transmission dynamics and management of HIV and AIDS. The model is shown to be positively invariant as well as bounded. The endemic state is shown to exist provided that the reproduction number is greater than unity. Furthermore, by the use of Routh-Hurwitz criterion and suitable Lyapunov functions, the endemic states are shown to be locally and globally asymptotically stable. This implies that disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrences. Numerical simulations indicate that minimal interference against the disease lowers the rate of infection and enhances the disease management.

Keywords: Interference; HIV and AIDS management; reproduction number; stability analysis.

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1 Introduction

Human Immunodeficiency Virus (HIV) is a virus spread through certain body fluids. The virus attacks the body's immune system, specifically the CD4 cells, often called T cells. Over the time, HIV weakens the body's immune system [1], and this makes it difficult for the body to fight off infection from other diseases such as malaria, tuberculosis, pneumonia among others [2]. Unlike some other viruses, the human body can't get rid of HIV completely, even with treatment [3; 4].

The epidemic of AIDS has been steadily spreading for the past two decades, and now affects every country in the world. Each year, more people die, and the number of HIV+ persons continues to rise despite national and international HIV prevention policies and dedicated public healthcare strategies [5].

Numerous mathematical models have been developed to explore transmission dynamics and treatment of HIV and AIDS at population level. According to Adnan [6], the discovery of human immunodeficiency virus (HIV) as the causative organism of acquired immunodeficiency syndrome (AIDS) and inability of modern medicine to find a cure for it has placed HIV as one of the most dreaded pathogens of the 21^{st} century. However, with the advent of antiretroviral therapy (ART), it is now possible to control HIV. Adherence to ART helps to keep the viral load under control and prolong the time of progression to AIDS, resulting in near normal life expectancy. Even with the introduction of ART, a substantial number of patients fail to adhere due to a variety of reasons, including adverse side effects, drug abuse, mental disorders, socioeconomic status, literacy, and social stigma. Close monitoring, major social reforms, and adequate counseling should also be implemented to curb other challenges.

Homelessness people are amongst most vulnerable in the society and do not get the help they need to address their health, economic, and social issues. Homelessness and HIV/AIDS are intricately related as homelessness worsens HIV since they are doubly affected. The pressure of daily needs, exposure to violence, alcohol and mental health issues and other conditions of the homelessness make homeless and unstably housed people extremely vulnerable to HIV infection [7].

Despite of the many intervention strategies put in place, the impact of HIV and AIDS is pervasive and far-reaching, with devastating effects on individuals and communities psychologically, economically and socially. Families lose their most productive members due to this disease, leaving children and elderly people without means of support. This continued spread may be attributed to increased interference in the management of the disease. Interference is the act or the process of hindering, obstruction or impeding, that is, the action of interfering or the process of being interfered with. In this context, interference in the management and the control of HIV and AIDS are those factors which should be done to reduce the spread of HIV and AIDs infection but are, however, not being done. This includes little or no public awareness, sharing sharp objects, having unprotected sex, non adherence to antiretroviral therapy, homelessness among others which are different forms of human behaviour. Thus, this study develops a deterministic model to investigate the role of interference on the transmission dynamics and management of HIV and AIDS.

2 The Model

We formulate a model in which the total human population at any time t denoted by N is subdivided into classes, $S(t)$ the class of individuals susceptible to HIV and AIDS infection. Recruitment into susceptible class is done at a rate Λ. The class $I(t)$ consists of individuals who are asymptomatically infected with HIV infection, this infection occurs at the rate λ. In the

absence of treatment, individuals develop symptoms of HIV and AIDS and progress from the class $I(t)$ to the class $A(t)$ at the rate ρ. Treatment for HIV and AIDS is done at the rate α, thus the classes $I_t(t)$ and $A_t(t)$ consist of individuals who are on treatment. Non adherence to treatment may be lost at the rate β, thus the classes $I_t(t)$ and $A_t(t)$ slide back to class $I(t)$ and class $A(t)$ respectively. Mortality occurs among AIDS patients at the rate ν while natural death is assumed to occur in all classes at the rate μ.

At a given time the total population is given by:

$$N(t) = S(t) + I(t) + I_t(t) + A(t) + A_t(t) \tag{1}$$

The rate at which a susceptible individual acquire HIV and AIDS is defined as

$$\lambda = \frac{\pi\theta[I_t(t) + \omega I(t)]}{N(t)} \tag{2}$$

Where π is the probability that susceptible individuals will acquire HIV upon effective contact with an HIV infected individual and θ is the effective contact rate with HIV infected individuals While ω is a modification parameter accounting for the assumed increased infectivity due to dual infection.

This study sought to investigate the role of interference on the transmission dynamics and management of HIV and AIDS. Let, τ denote the interference term, the modified force of infection becomes:

$$\lambda = \frac{\pi\theta\tau[I_t(t) + \omega I(t)]}{N(t)} \tag{3}$$

Where, $\tau > 1$ since interference is correlated with infectiousness.

From the above definitions, the resulting diagram for the model is given in Fig. 1.

The dynamics described can be represented mathematically as;

$$\begin{aligned}
\dot{S}(t) &= \Lambda - (\mu + \lambda)S(t) \\
\dot{I}(t) &= \lambda S(t) + \beta I_t(t) - (\alpha + \rho + \mu)I(t) \\
\dot{I}_t(t) &= \alpha I(t) - (\mu + \beta)I_t(t) \\
\dot{A}(t) &= \rho I(t) + \beta A_t(t) - (\alpha + \mu + \nu)A(t) \\
\dot{A}_t(t) &= \alpha A(t) - (\beta + \mu + \nu)A_t(t)
\end{aligned} \tag{4}$$

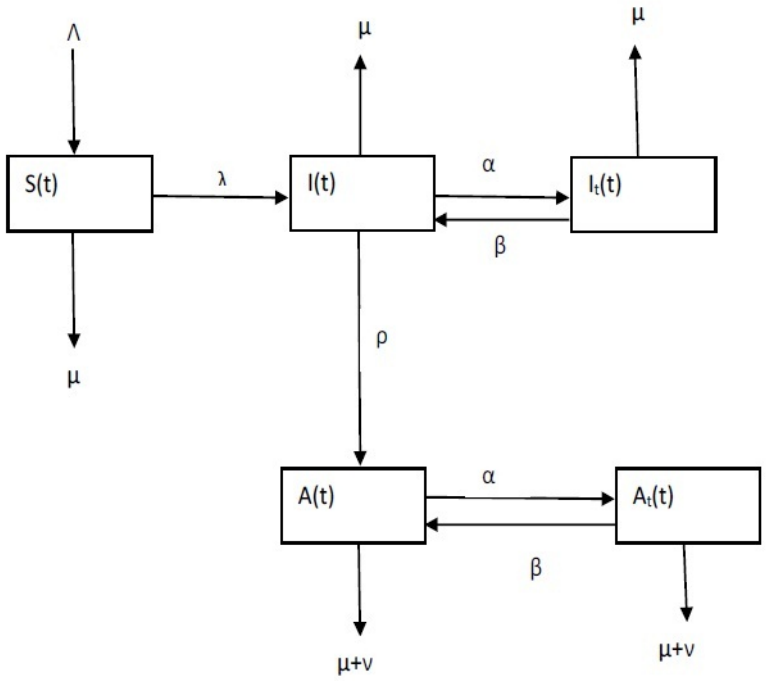

Fig. 1. Model flow diagram

3 Model Analysis

Based on the fact that the model deals with human population, all the state variables and parameters are assumed to be non-negative $\forall t \geq 0$. This model is studied in the feasible region $\mathbb{R}$ where $\{S(t), I(t), I_t(t), A(t), A_t(t)\} \epsilon \Omega \epsilon \mathbb{R}^5_+$ and it can be shown that as t tends to infinity;

$$0 \leq N(t) \leq \frac{\Lambda}{\mu} \tag{5}$$

Which shows that the set of solutions is bounded. Thus, the model Equation (4) is epidemiologically well posed in the region Ω.

The basic reproduction number R_0, computed using the next generation matrix method approach for Equation (4) is given by

$$R_0 = \pi\theta\tau\{\frac{\alpha + (\mu + \beta)\omega}{(\mu\alpha + (\mu + \beta)(\mu + \rho)}\} \tag{6}$$

.

The endemic state is defined as

$$I^*(t) = \frac{(\beta + \mu)\Lambda}{(\alpha\mu + (\beta + \mu)(\mu + \rho))R_0}\{R_0 - 1\} \tag{7}$$

For an infection to be endemic in a population, $I^*(t) > 0$. This inequality holds provided that $R_0 > 1$.

4 Disease-free Equilibrium Point

The disease-free equilibrium point is a steady-state solution for which there is no disease or infection in the population [8]. To obtain the disease-free equilibrium point we set the normalised model system (4) equal to zero as shown below, $E^0 = \{S(t), I(t), I_t(t), A(t), A_t(t)\} = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$

5 Local Stability of the Disease-free Equilibrium

The Jacobian matrix of Equation (4) is given by

$$J = \begin{pmatrix} -(\mu+\lambda) & 0 & 0 & 0 & 0 \\ \lambda & -(\alpha+\rho+\mu) & \beta & 0 & 0 \\ 0 & \alpha & -(\beta+\mu) & 0 & 0 \\ 0 & \rho & 0 & -(\alpha+\mu+\nu) & \beta \\ 0 & 0 & 0 & \alpha & -(\beta+\mu+\nu) \end{pmatrix} \tag{8}$$

Clearly $-(\lambda+\mu)$ is an eigenvalue. We analyse the reduced matrix

$$J_1 = \begin{pmatrix} -(\alpha+\rho+\mu) & \beta & 0 & 0 \\ \alpha & -(\beta+\mu) & 0 & 0 \\ \rho & 0 & -(\alpha+\mu+\nu) & \beta \\ 0 & 0 & \alpha & -(\beta+\mu+\nu) \end{pmatrix} \tag{9}$$

The trace of Equation (9) is negative and the determinant is given by

$$\det J_1 = (-\alpha-\mu-\nu)(-\beta-\mu-\nu)(\mu\alpha \ +\mu\rho+\rho\beta+\mu\mu+\beta\mu) - \alpha \ (\beta\mu\alpha+\beta\mu\beta+\beta\mu\mu+\beta\rho\mu+\beta\rho\beta)$$

Thus the $detJ_1 > 0$, provided $(-\alpha-\mu-\nu)(-\beta-\mu-\nu)(\mu\alpha+\mu\rho+\rho\beta+\mu\mu+\beta\mu) > \alpha(\beta\mu\alpha+\beta\mu\beta+\beta\mu\mu+\beta\rho\mu+\beta\rho\beta)$.

Therefore, the disease-free equilibrium is locally asymptotically stable.

6 Global Stability of the Disease-free Equilibrium

For global stability of the DFE, the technique by Castillo [9] is used. There are two conditions that if met, guarantee the global asymptotic stability of the disease free state. Equation (4) may be written in the form

$$\begin{aligned} \frac{dX}{dt} &= H(X,Z), \\ \frac{dZ}{dt} &= G(X,Z), G(X,0) = 0 \end{aligned} \tag{10}$$

Where $X \in \mathbb{R}^1$ and $X = \{S(t)\}$ denotes the number of uninfected individuals and $Z \in \mathbb{R}^4$ where $Z = \{I(t), I_t(t), A(t), A_t(t)\}$ denotes the number of infected individuals. $E^0 = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$

denotes the disease free equilibrium point of this system where

$X^* = \frac{\Lambda}{\mu}$

Conditions in (11) must be met to guarantee a local asymptotic stability:

$\frac{dX}{dt} = H(X,0), X^*$ is globally asymptotically stable (GAS)

$$G(X,Z) = PZ - \hat{G}(X,Z), \hat{G}(X,Z) \geq 0 for (X,Z) \in \Omega \tag{11}$$

Where, $P = D_z G(X^*, 0)$ is an M-matrix (the off-diagonal elements of P are non-negative) and Ω is the region where the model makes biological sense.

Theorem 1. *If system* (10) *satisfies conditions* (11)*, then the fixed point* $E^0 = (X^*, 0, 0, 0, 0)$ *is a globally asymptotically stable equilibrium of system* (10) *provided that* $R_0 < 1$ *and the assumptions in* (11) *are satisfied.*

Proof. Consider

$H(X,O) = \Lambda - \mu S$ and $G(X,Z) = PZ - \hat{G}(X,Z)$

Where $P = \begin{pmatrix} -(\alpha+\rho+\mu) & \beta & 0 & 0 \\ \alpha & -(\mu+\beta) & 0 & 0 \\ \rho & 0 & -(\alpha+\mu+\nu) & \beta \\ 0 & 0 & \alpha & -(\beta+\mu+\nu) \end{pmatrix}$

And

$$G(X,Z) = \begin{pmatrix} \hat{G_1}(X,Z) \\ \hat{G_2}(X,Z) \\ \hat{G_3}(X,Z) \\ \hat{G_4}(X,Z) \end{pmatrix} = \begin{pmatrix} -\pi\theta\tau[I_t(t) + \omega I(t)] \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Considering the Jacobian matrix, and replacing $S(t) = \frac{\Lambda}{\mu}$, $I(t) = 0$, $I_t(t) = 0$ $A(t) = 0$, $A_t(t) = 0$ we obtain $\hat{G_1}(X,Z) = 0$ and so the conditions in (11) are met so E^0 is globally asymptotically stable when $R_0 < 1$. This implies that we do not expect the disease outbreak for life. Thus, the epidemic will die out or will not develop in the population.

□

7 Local Stability of the Endemic Equilibrium

The total population N from Equation (4) is $N(t) = S(t) + I(t) + I_t(t) + A(t) + A_t(t)$. Thus we study the equations at the endemic state $E^*\{S^*(t), I^*(t), I_t^*(t), A^*(t), A_t^*(t)\}$. The Jacobian of Equation (4) at endemic state E^* is given by

$$J_2 = \begin{pmatrix} -\mu - F_1 & \frac{\pi\theta\tau\omega S^*}{N} & \frac{\pi\theta\tau S^*}{N} & 0 & 0 \\ F_1 & F_2 & \frac{\pi\theta\tau S^*}{N} + \beta & 0 & 0 \\ 0 & \alpha & -(\beta+\mu) & 0 & 0 \\ 0 & \rho & 0 & -(\alpha+\mu+\nu) & \beta \\ 0 & 0 & 0 & \alpha & -(\beta+\mu+\nu) \end{pmatrix} \tag{12}$$

Where $F_1 = \frac{\pi\theta\tau}{N}(I_t^* + \omega I^*)$ and $F_2 = \frac{\pi\theta\tau\omega S^*}{N} - (\alpha + \rho + \mu)$

An important creterion by Routh-Hurwitz gives the necessary and sufficient conditions for all the roots of the characteristic polynomial (with real coefficients) to lie in the left half of the complex plane. In other words, all the roots of the polynomial are negative or have negative real roots if the determinants of all Hurwitz matrices are positive [10]. From the Jacobian matrix (8) , for the trace to be negative

$$(\alpha + \rho + \mu) > \frac{\pi\theta\tau\omega S^*}{N} \tag{13}$$

The determinant will be given by

$$detJ(E^*) = -\alpha\beta(-\alpha(-\frac{\beta\pi\theta\tau\omega I^*}{N} - \beta\mu - \frac{2\beta\pi\theta\tau\omega I^*\pi\theta\tau S^*}{N^2} - \mu\frac{\pi\theta\tau S^*}{N} - \frac{\beta\pi\theta\tau I_t^*}{N} - \frac{2\beta\pi\theta\tau I_t^*\pi\theta\tau S^*}{N^2}) + (-\beta - \mu)(-\frac{\pi\theta\tau\pi\theta\tau\omega S^*(I_t^* + \omega I^*)}{N^2} + (\frac{\pi\theta\tau\omega S^*}{N} - (\alpha + \rho + \mu))(-\mu - \frac{\pi\theta\tau}{N}(I_t^* + \omega I^*)))) + (-\alpha - \mu - \nu)(-\beta - \mu - \nu)(-\alpha(-\frac{\beta\pi\theta\tau\omega I^*}{N} - \beta\mu - \frac{2\beta\pi\theta\tau\omega I^*\pi\theta\tau S^*}{N^2} - \mu\frac{\pi\theta\tau S^*}{N} - \frac{\beta\pi\theta\tau I_t^*}{N} - \frac{2\beta\pi\theta\tau I_t^*\pi\theta\tau S^*}{N^2}) + (-\beta - \mu)(-\frac{\pi\theta\tau\pi\theta\tau\omega S^*(I_t^* + \omega I^*)}{N^2} + (\frac{\pi\theta\tau\omega S^*}{N} - (\alpha + \rho + \mu))(-\mu - \frac{\pi\theta\tau}{N}(I_t^* + \omega I^*))))$$

The determinant $detJ(E^*) > 0$ provided that;

$$(-\alpha - \mu - \nu)(-\beta - \mu - \nu) > \alpha\beta \tag{14}$$

Since the trace is negative and the determinant is positive provided that inequality (13) and inequality (14) holds, then the eigenvalues of Equation (12) will have negative real parts. Therefore, the endemic equilibrium is locally asymptotically stable. This implies that the disease transmission levels can be kept quite low or manageable with minimal deaths.

8 Global Stability of the Endemic Equilibrium

The global stability of the equilibria is obtained by means of the Lyapunov's direct method and LaSalle's invariance principle [11]. Consider the non-linear Lyapunov function $V : (S, I, I_t, A, A_t) \in \Omega \subset \mathbb{R}_+^5 : S, I, I_t, A, A_t > 0$
defined as

$$V = S - S^* \ln S + I - I^* \ln I + I_t - I_t^* \ln I_t + A - A^* \ln A + A_t - A_t^* \ln A_t \tag{15}$$

Where V is in the interior of the region Ω. E^* is the global minimum of V on Ω and $V : (S, I, I_t, A, A_t) = 0$. The time derivative of Equation (15) is given by

$\frac{dV}{dt} = \dot{V} = \dot{S}(1 - \frac{S^*}{S}) + \dot{I}(1 - \frac{I^*}{I}) + \dot{I}_t(1 - \frac{I_t^*}{I_t}) + \dot{A}(1 - \frac{A^*}{A}) + \dot{A}_t(1 - \frac{A_t^*}{A_t})$
$\dot{V} = (\Lambda - (\mu + \lambda)S)(1 - \frac{S^*}{S}) + (\lambda S + \beta I_t - (\rho + \mu + \alpha)I)(1 - \frac{I^*}{I}) + (\alpha I - (\mu + \beta)I_t)(1 - \frac{I_t^*}{I_t}) + (\rho I + \beta A_t - (\mu + \nu + \alpha)A)(1 - \frac{A^*}{A}) + (\alpha A - (\mu + \nu + \beta)A_t)(1 - \frac{A_t^*}{A_t})$
$\dot{V} = (\Lambda - \Lambda\frac{S^*}{S} - (\mu + \lambda)S) + S^*(\mu + \lambda) + (\lambda S - \lambda S\frac{I^*}{I} + \beta I_t - \beta I_t\frac{I^*}{I} - (\rho + \mu + \alpha)I) + (\rho + \mu + \alpha)I^* + (\alpha I - \alpha I\frac{I_t^*}{I_t} - (\mu + \beta)I_t + (\mu + \beta)I_t^* + (\rho I - \rho I\frac{A^*}{A} + \beta A_t - \beta A_t\frac{A^*}{A} - (\mu + \nu + \alpha)A) + (\mu + \nu + \alpha)A^* + (\alpha A - \alpha A\frac{A_t^*}{A_t} - (\mu + \nu + \beta)A_t) + (\mu + \nu + \beta)A_t^*$

At endemic states: $\dot{V} = (\mu+\lambda)S^*(2-\frac{S}{S^*}-\frac{S^*}{S})+(\lambda S^*+\beta I^*)(1-\frac{I^*}{I})+\alpha I^*(1-\frac{I}{I^*}\frac{I_t^*}{I_t})+(\rho I^*+\beta A_t^*)(1-\frac{A^*}{A})+\alpha A^*(1-\frac{A}{A^*}\frac{A_t^*}{A_t})\leq 0$

Hence $V<0$. We see that $V=0$ iff $S=S^*, I=I^*$, $I_t=I_t^*$, $A=A^*$ and $A_t=A_t^*$. Thus the largest compact invariant set in $\{S,I,I_t,A,A_t\}\in\Omega : V=0$ is the Singleton E^*, where E^* is the endemic equilibrium. Thus E^* is globally asymptotically stable in the interior of the region Ω. This implies that the disease transmission levels can be kept quite low or manageable with minimal deaths at the peak times of the re-occurrence.

9 Sensitivity Analysis

Sensitivity analysis of R_0 with respect to the model parameters is carried out in order to determine the role of interference on the transmission dynamics and management of HIV and AIDS [12]. To perform sensitivity analysis, we use the normalised forward sensitivity index also known as elasticity [13]. The normalised forward sensitivity index of the reproduction number R_0 in Equation (6) with respect to interference parameter τ is given by;

$$\Gamma_{\tau}^{R_0} = \frac{\partial R_0}{\partial \tau}\times\frac{R_0}{\tau} = 1 \tag{16}$$

This implies that, the higher the interferences the higher the rate of infection.

10 Numerical Simulations

Numerical simulations were carried out to graphically illustrate the role of interference on the dynamics of HIV and AIDS transmission and management. To do this, some parameter values were used as indicated in Table 1.

Table 1. Parameter values used in simulation of HIV and AIDS model

Parameter	description	Value	Source
S(t)	susceptible individuals	4000	Estimate
I(t)	infected individuals	800	Estimate
A(t)	fully blown individuals	97	Estimate
Λ	Recruitment rate	600 per year	[14]
μ	mortality rate	0.85	[15]
ν	HIV/AIDS related death rate	0.94 per year	[16]
β	failure to adhere to treatment	0.2 per year	Estimate
α	Protection against HIV and AIDS	0.1 per year	Estimate
λ	Force of infection	2-100 per year	Estimate
ρ	Rate of progression to AIDS	2 per year	[17]

Based on the initial conditions and parameter values in Table 1 , the following graphs were obtained;

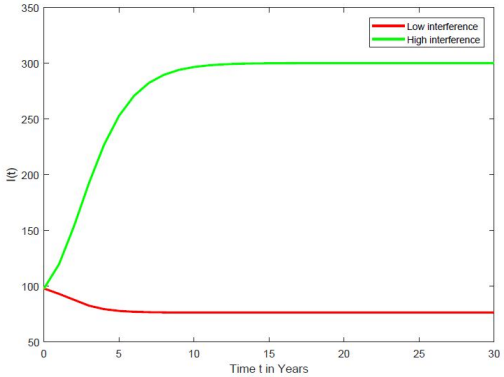

Fig. 2. Graph of I(t) against time t at low and high interference

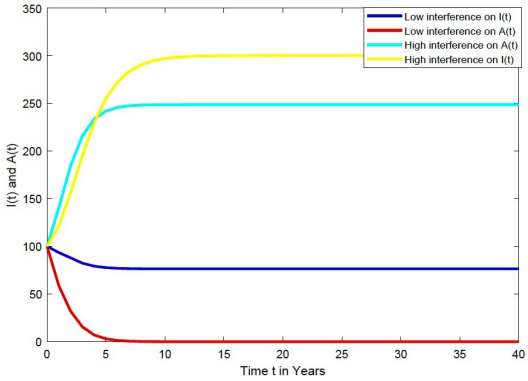

Fig. 3. Graph of I(t) and A(t) against time t at low and high interference

11 Discussion

From Fig. 2 , we observe that with a high interference rate the number of $I(t)$ rises sharply in a short span before remaining constant. The rise may be attributed to non adherence to condom use and antiretroviral therapy, little or no public awareness, high level of homelessness among others, which translates to high viral load, eventually leading to high HIV and AIDS infection. The constant trend is due to $I(t)$ channeling to $A(t)$ as a result of increased viral load for HIV persons which leads to a faster rate of progression to the symptomatic stage. From the same figure, low interference rate results to reduced rates of transmission and reduced viral load. This is as a result of people adhering to condom use and antiretroviral therapy, male circumcision, low level of homelessness, increased public awareness among others.

Additionally, Fig. 3 shows a combination of $I(t)$ and $A(t)$. With a high interference rate, more $I(t)$ are channeling to $A(t)$ and this leads to the sharp rise of $A(t)$ in a short span before remaining constant. The constant trend in $A(t)$ may be due to the death rate of $A(t)$ as a result of the infection and the natural death rate. From the same figure, low interference reduces $I(t)$ leading to a reduction in $A(t)$.

In order to reduce the number of new HIV and AIDS and reduce their impact on individuals, families and communities, there is a need to adhere and employ strategies such as increasing the public awareness drive to behaviour change and encourage openness, increasing access to voluntary HIV testing and counselling, promoting increased condom use to reduce the spread of HIV infection, male circumcision, improving access and adherence to antiretroviral therapy (ART) for people living with HIV and AIDS, practising proper hygiene and eating a balanced diet. These strategies will help in reducing the economic burden that is borne by a country in giving care and treating the infected individuals. As evidenced from these results, it is indeed true that low interferences on the transmission dynamics and management of HIV and AIDS impact positively on the control of the disease. Thus, more emphasis should be on adherence to all strategies that will lead to reduced levels of infection with HIV and progression to AIDS stage.

12 Conclusion

In this work, we formulated a model to investigate the role of interference on the transmission dynamics and management of HIV and AIDS. The model is shown to be positively invariant as well as bounded. The existence of the endemic equilibrium was established and the stability of the same was analysed. The endemic equilibrium was found to be globally asymptotically stable. This implies that at peak times of the reoccurrence of the diseases, the levels of infections are manageable with minimal interferences.

From the numerical simulations, we observe that minimal interference against the disease has the effect of reducing and managing the disease prevalence. This is in agreement with the mathematical analysis which showed that with low interferences, the endemic equilibrium was locally and globally asymptotically stable.

13 Recommendation

The government, through the relevant bodies such as the health sector, needs to sensitise the public about the need for reducing interferences with the view to reduce the disease prevalence in the population. Since interference has been used in a general sense, further research may be carried out to analyse the contribution of specific interference measures in the overall transmission dynamics and management of HIV and AIDS.

Competing Interests

Authors have declared that no competing interests exist.

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