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Global Stability and Sensitivity Analysis of the Dynamics of Human Population Subjected to HIV/AIDS with Treatment

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Abstract : *In this study, a mathematical model of human population dynamics pertaining to the HIV/AIDS has been formulated. This study categorized human population into five compartments as Susceptible, Primary, Asymptomatic, Symptomatic, and AIDS (SPAJV). The well-possedness of the formulated models proved. The equilibrium points of the model are identified. Additionally, parametric expression for the basic reproduction number is constructed following next generation matrix method and analyzed its stability using Routh Hurwitz criterion. From the analytical and numerical simulation studies it is observed that if the basic reproduction is less than one unit then the solution converges to the disease free steady state i.e., disease will wipe out and thus the treatment is said to be successful. On the other hand, if the basic reproduction number is greater than one then the solution converges to endemic equilibrium point and thus the infectious humans continue to increase i.e., disease will persist in the community and thus the treatment is said to be unsuccessful. Sensitivity analysis of the model parameters is conducted and their impact on the reproduction number is analyzed. Finally, numerical solutions of the model equations are simulated using MATLAB. The results and observations have been included in the text of this paper lucidly.*

Keywords: *HIV, Basic Reproduction Number, Stability Analysis, Routh Hurwitz criterion, well-possedness*

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I. Introduction

Starting from the discovery of AIDS (Acquired Immunodeficiency Syndrome) and its causing virus HIV (Human Immunodeficiency Virus) in 1980s they have been showing a great impact on human kind around the world. As fatal diseases they have become sources of stigma and discrimination around the world [1].Since the appearance of the epidemic, it is estimated that 76.1 million people have been infected with HIV and about 35 million individuals have been died of AIDS related illness [1, 2, 3].

In 2016, it was estimated that 36.7 million people were living with HIV disease. This figure includes 1.8 million of newly infected people during that year. Also, nearly 1 million people have died during the same year because of AIDS related illness [4].

HIV is a virus that slowly attacks human immune system. This immune system functions as the natural defense department of human body to fight against all kinds of illnesses. Whenever a person gets infected with HIV virus then her or his immune system gets weaker and weaker. It leads to the poor health of such a patient and eventually it becomes hard to the immune system to fight over infections and diseases [1, 2].

Based on WHO clinical staging of HIV/AIDS disease, the HIV infection is classified into four distinct stages viz.,(i) Primary/Acute stage(ii) Asymptomatic stage (iii) Symptomatic stage and (iv) Advanced AIDS stage [2, 4]. These four stages of the disease have been briefly described in what follows:

Stage 1(Primary HIV infection): This stage of HIV infection is a first stage and known as primary infection stage. Primary infection begins after an individual becomes infected with HIV for the first time. This stage lasts for a few weeks. During this period, individuals experience Flu fever like symptoms. However, the test of HIV infection is invisible at this stage. Thus, who have been exposed to HIV should check themselves again after six months.

*Stage 2(Asymptomatic HIV infection):*This stage of HIV is a second stage and is known as Asymptomatic HIV infection , individuals become free from all types of symptoms of HIV although there will be some swollen glands. The HIV appears in blood in very low level, but it can be detectable. If an HIV test is performed, the result will come out to be positive. While the individuals remain asymptomatic, the HIV in their blood continues to reproduce constantly. This stage lasts for about ten years. However, the period of second stage can be much longer or shorter depending on the individual. This period is also characterized by a CD4+count whose count is around 500 cells per μl.

*Stage 3(Symptomatic HIV infection):*This stage of HIV is called *Symptomatic HIV infection*, at this stage symptoms start appearing and human immune system gets damaged by HIV. Further, it leads to high

destruction ofCD4+ cells and human immune system become very weak to replace them. Thus, the immune system stops properly functioning and this leads to developing of disease symptoms [3, 4].

Stage 4(Acquired Immune Deficiency Syndrome AIDS): In fourth and last stage, a person can be medically tested positive as having AIDS. The progression to AIDS can be characterized by CD4+ count which is 200 or below per ml in a patient, while it is around 1000 per ml in a normal person. At this stage, the infected individual is likely to develop opportunistic infections in their respiratory system, gastro-intestinal system, central nervous system and on the skin as well. Once a person is diagnosed with AIDS, the AIDS status remains permanent.

Organization of the paper: In Section 2, assumptions of the model are stated and based on which a mathematical model for describing the population dynamics of human population related to HIV/AIDS disease is formulated. In section 3, well possedness of the model formulation, stability analysis of the equilibrium points and reproduction number are included. In Section 4, numerical simulation studies of the model equations are performed by assigning various sets of numerical values to the model parameters. In Section 5 sensitivity analysis of model parameters towards the reproduction number is carried out. In section 6 Result and Discussion are presented. Finally, the paper ends with concluding remarks in Section 7.

II. Model Formulation

The model proposed in this study divides total human population into five compartments to describe the dynamics of human population pertaining to HIV (Human Immunodeficiency Virus)using ART as treatment. These compartments are described as follows: (i) Susceptible human's compartment. It is denoted by $S(t)$. These susceptible humans are capable of becoming infected in the future. This include new human that not infected yet. (ii) Primary infected human's compartment. It is denoted by $P(t)$.This compartment consists of exposed or infected humans that do not know their status but transmit disease to others with effective contact (iii) Asymptomatic human's compartment. It is denoted by $A(t)$. This compartment consists of those who know that they are infected with virus but shows no signs of infections and abstain from transmitting virus to others. They undergo ART and Herbal medicine treatments (iv) Symptomatic human compartment. It is denoted by $J(t)$. This compartment consists of infectious humans and they show signs of infections. Such humans manifest their weakness as they harmed by virus and abstain from transmitting virus to others and undergo Herbal medicine treatment (v) AIDS human's compartment. It is denoted by $V(t)$. This consists of humans that highly contains virus that weakens T-helper cells. This categorical human requires high medications and cares.

Here, a mathematical model of the Human Immunodeficiency virus (HIV) is constructed based on the following assumptions made on the human populations:

(i) The total population size of human's populations is assumed to be constant.
(ii) Both the numbers of births and deaths of humans are equal.
(iii) Human Immunodeficiency virus (HIV) model classifies the human population into five compartments as SPAJV at any time.
(iv) Susceptible humans are recruited into the compartment $S(t)$ at a constant rate τ.
(v) Susceptible humans are infected when they come into effective contact with primary infected humans that do not know their status and join primary infected compartment at a rate β.
(vi) Asymptomatic humans join symptomatic humans at a rate θ.
(vii) The symptomatic humans join asymptomatic human's compartment at the rate ω and AIDS human's compartment at a rate γ.
(viii) All types of human's compartments suffer natural mortality with a rate μ.
(ix) AIDS humans die of infection at a rate ρ.
(x) Asymptomatic and Symptomatic humans are treated
(xi) All parameters used in the model are positive.

Table 1 Notations and description of model variables

Variable	Description
$\mathbf{S(t)}$	Population size of susceptible humans
$\boldsymbol{P(t)}$	Population size of primary infected humans
$\mathbf{A(t)}$	Population size of asymptomatic humans
$\mathbf{J(t)}$	Population size of symptomatic humans
$\mathbf{V(t)}$	Population size of AIDS humans

Table 2 Notations and description of model parameters

Parameter	Description
τ	Recruitment rate of susceptible humans. With this rate new humans will born and they will enter into susceptible compartment

$\boldsymbol{\beta}$	Transmission rate of infected humans. With this rate humans transfer from compartment S to P
$\boldsymbol{\kappa}$	Rate of humans transferring from compartment P to A
$\boldsymbol{\eta}$	Rate of humans transferring from compartment P to J
$\boldsymbol{\omega}$	Rate of humans transferring from compartment J to A because of treatment with ART
$\boldsymbol{\theta}$	Rate of humans transferring from compartment A to J because of ART misusing or stopping
$\boldsymbol{\mu}$	Natural death rate. With this rate humans in all compartments die naturally
$\boldsymbol{\gamma}$	Rate of humans transferring from compartment J to V.
$\boldsymbol{\rho}$	Death rate of AIDS humans

Based on the basic assumptions together with the description of both model variables and parameters the schematic diagram of the compartmental model is drawn and is given in Figure 1.

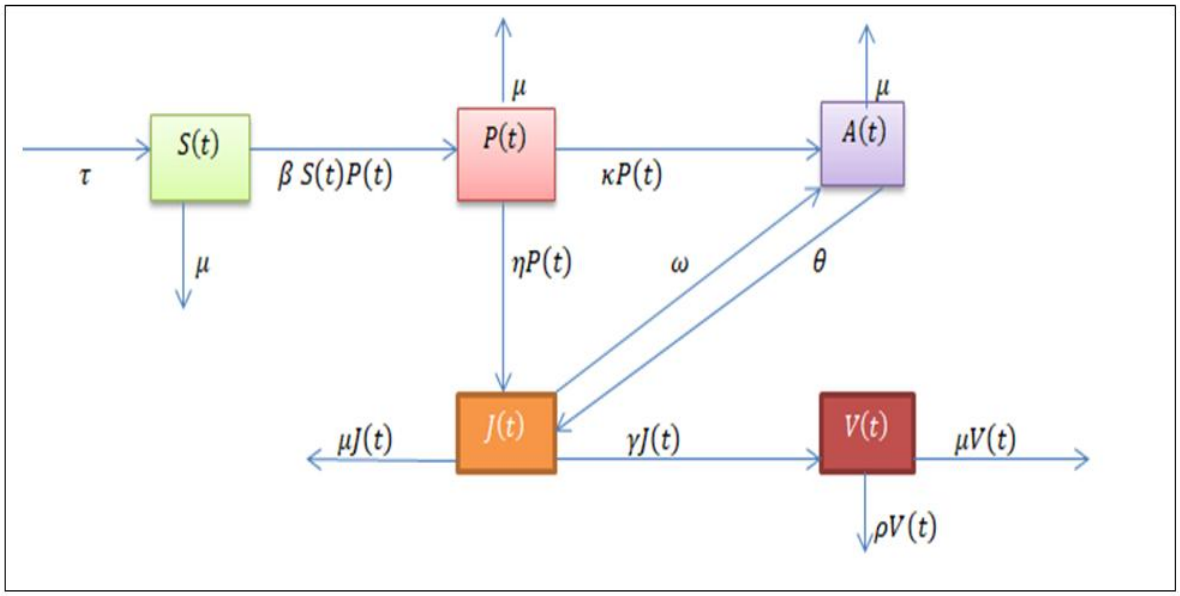

Figure 1 Schematic diagram of compartmental structure of the model

Based on the model assumptions, the notations of variables and parameters and the schematic diagram, the model equations are formulated and are given as follows:

$$dS/dt = \tau - \beta S(t)P(t) - \mu S(t) \quad (1)$$
$$dP/dt = \beta S(t)P(t) - (\kappa + \eta + \mu)P(t) \quad (2)$$
$$dA/dt = \kappa P(t) + \omega J(t) - (\theta + \mu)A(t) \quad (3)$$
$$dJ/dt = \eta P(t) + \theta A - (\omega + \gamma + \mu)J \quad (4)$$
$$dV/dt = \gamma J(t) - (\rho + \mu)V \quad (5)$$

The non-negative initial conditions of the model equations (1) – (5) are denoted by $S(0) > 0,\ P(0) \geq 0,\ A(0) \geq 0,\ J(0) \geq 0,\ V(0) \geq 0$. This system consists of five first order non-linear ordinary differential equations.

III. Mathematical analysis of the model

In this section mathematical analysis of the present, improved and modified model is conducted. The analysis consists of the features including (i) existence, positivity and boundedness of solutions (ii) steady states (iii) disease free equilibrium points (iv) endemic equilibrium points (v) basic reproduction number (vi)stability analysis of the disease free equilibrium points(vii)local stability of disease free equilibrium point (viii) global stability of disease free equilibrium point. These mathematical aspects of the model are presented and discussed in the following sub-sections respectively.

3.1 Existence, Positivity and Boundedness of solution

In order to show that the model is biologically valid and mathematically well-posed, it is required to prove that the solutions of the system of differential equations (1) – (5) exist, non-negative and bounded for all time t . It is done starting with proving Lemma 1.

Lemma 1 (Positivity) Solutions of the model equations (1) – (5) together with the initial conditions$S(0) \geq 0$, $P(0) \geq 0$, $A(0) \geq 0$, $J(0) \geq 0$, $V(0) \geq 0$ are always non-negative (OR) the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$,and $V(t)$are non-negative for all t and will remain in$\mathbb{R}_+^5$.

Proof Positivity of the solutions of model equations is shown separately for each of the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$ and $V(t)$

Positivity of $S(t)$*:* The model equation (1) given by $dS/dt = \tau - \beta S(t)P(t) - \mu S$ can be expressed without loss of generality, after eliminating the positive termτ appearing on the right hand side, as an inequality as $dS/dt \geq -[\beta P(t) + \mu]S(t)$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $S(t) \geq S(0)e^{-\mu t - \beta \int P(t)dt}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function $e^{-\mu t - \beta \int P(t)dt}$ is a non-negative quantity. Hence, it can be concluded that $S(t) \geq 0$.

Positivity of $P(t)$*:* The model equation (2) given by $dP/dt = \beta S(t)P(t) - (\eta + \theta + \mu)P(t)$ can be expressed without loss of generality, after eliminating positive term $\beta S(t)P(t)$ which is appearing on the right hand side, as an inequality as$dP/dt \geq -(\eta + \theta + \mu)P(t)$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $P(t) \geq P(0)e^{-(\eta+\theta+\mu)t}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function $e^{-(\eta+\theta+\mu)t}$ is a non-negative quantity. Hence, it can be concluded that $P(t) \geq 0$.

Positivity of $A(t)$*:* The model equation (3) given by $dA/dt = \kappa P(t) + \omega J(t) - (\theta + \mu)A(t)$can be expressed without loss of generality, after eliminating the positive terms$\kappa P(t)$and $\omega J(t)$ which are appearing on the right hand side, as an inequality as $dA/dt \geq -(\theta + \mu)A(t)$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $A(t) \geq A(0)e^{-(\theta+\mu)t}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function $e^{-(\theta+\mu)t}$ is a non-negative quantity. Hence, it can be concluded that $A(t) \geq 0$.

Positivity of $J(t)$*:* The model equation (4) given by$dJ/dt = \eta P(t) + \theta A - (\omega + \gamma + \mu)J$ can be expressed without loss of generality, after eliminating the positive terms $\eta P(t)$and θA which are appearing on the right hand side, as an inequality as $dJ/dt \geq -(\omega + \gamma + \mu)J$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $J(t) \geq J(0)e^{-(\omega+\gamma+\mu)t}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function$e^{-(\omega+\gamma+\mu)t}$is a non-negative quantity. Hence, it can be concluded that $J(t) \geq 0$.

Positivity of $V(t)$*:* The model equation (5) given by $dV/dt = \gamma J(t) - (\rho + \mu)V$ can be expressed without loss of generality, after eliminating the positive term $\gamma J(t)$ which is appearing on the right hand side, as an inequality as $dV/dt \geq -(\rho + \mu)V$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $V(t) \geq V(0)e^{-(\rho+\mu)t}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function $e^{-(\rho+\mu)t}$ is a non-negative quantity. Hence, it can be concluded that $V(t) \geq 0$.

Thus, the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$ and $V(t)$ representing population sizes of various types of human population are positive quantities and will remain in $\mathbb{R}_+^5$for all$t$.

Lemma 2 (Boundedness) The non-negative solutions of the system of model equations (1) – (5) are bounded. That is the model variables$S(t)$, $P(t)$, $A(t)$,$J(t)$ and $V(t)$ are all bounded for all t [4, 7,8].

Proof: Recall that each population size is bounded if and only if the total population size is bounded. Hence, in the present case it is sufficient to prove that the total population size $N(t) = S(t) + P(t) + A(t) + J(t) + V(t)$ is bounded for all t. It can be begun by showing that all feasible solutions are uniformly bounded in a proper subset $R \in \mathbb{R}_+^5$ where the feasible region R is given by $R = \{(S, P, A, J V) \in \mathbb{R}_+^5 ; \mathrm{N} \leq \tau\mu+\rho$.

Now, summation of all the five equations (1) – (5) of the model gives $\mathrm{dN(t)/dt} = \tau - \mu\mathrm{N(t)} - \rho\mathrm{V}(t)$. Again considering total population N(t) and subpopulation V(t) further we can write the equation as inequality of the form $\mathrm{dN(t)/dt} \leq [\tau - (\mu + \rho)\mathrm{N(t)}]$. Equivalently this inequality can be expressed as a linear ordinary differential inequality as $[\mathrm{dN(t)/dt}] + [(\mu + \rho)\mathrm{N(t)}] \leq \tau$ giving general solution upon solving as $\mathrm{N(t)} \leq [\tau/(\mu + \rho)] + ce^{-(\mu+\rho)t}$. But, the term $N(0)$ denotes the initial values of the respective variable $\mathrm{N(t)} = \mathrm{N(0)}$at $\mathrm{t} = 0$. Thus, the particular solution can be expressed as $\mathrm{N(t)} \leq [\tau/(\mu + \rho)] + [\mathrm{N(0)} - (\tau/(\mu + \rho))]e^{-(\mu+\rho)t}$. Further, it can be observed that $N(t) \rightarrow$

$[\tau/(\mu+\rho)]$as $t \to \infty$. That is, total population size $N(t)$takes off from a value N(0) at the initial time t = 0 and ends up with a bounded value $[\tau/(\mu+\rho)]$as the time t progresses to infinity. Thus, it can be concluded that $N(t)$ is bounded within a pair of values as $0 \le N(t) \le [\tau/(\mu+\rho)]$.

Therefore, $[\tau/(\mu+\rho)]$is an upper bound of $N(t)$. Hence, feasible solution of the system of model equations (1) – (5) remains in the region Ω which is a positively invariant set. Thus, the system is biologically meaningful in the domainΩ. Further, it is sufficient to consider the dynamics of the populations represented by the model system (1) – (5) in that domain.

Therefore, it can be summarized the result of Lemma 2 as "the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$ and $V(t)$ are bounded for all t".

Lemma 3 (Existence) Solutions of the model equations (1) – (5) together with the initial conditions $S(0) > 0$, $P(0) \ge 0$, $A(0) \ge 0$, $J(0) \ge 0$, $V(0) \ge 0$ exist in $\mathbb{R}^5_+$ i.e. the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$, and $V(t)$ exist for all t and will remain in $\mathbb{R}^5_+$.

Proof: Let the right hand sides of the system of equations (1) – (5) are expressed as follows:

$$dS/dt = \tau - \beta S(t)P(t) - \mu S(t) \equiv g_1(S,\ P,\ A,\ J\ V)$$
$$dP/dt = \beta S(t)P(t) - (\kappa+\eta+\mu)P(t) \equiv g_2(S,\ P,\ A,\ J,\ V)$$
$$dA/dt = \kappa P(t) + \omega J(t) - (\theta+\mu)A(t) \equiv g_3(S,\ P,\ A,\ J,\ V)$$
$$dJ/dt = \eta P(t) + \theta A - (\omega+\gamma+\mu)J \equiv g_4(S,\ P,\ A,\ J,\ V)$$
$$dV/dt = \gamma J(t) - (\rho+\mu)V \equiv g_5(S,\ P,\ A,\ J,\ V)$$

According to Derrick and Groosman theorem, let R denote the region $R = \{(S,\ P,\ A,\ J,\ V) \in \mathbb{R}+5$; N≤τ$\rho+\mu$. Then equations (1) – (5) have a unique solution if $\partial gi\partial xj,\ \forall\ i,\ j=1,\ 2,\ 3,\ 4,\ 5$ are continuous and bounded in R. Here, the notations $x_1 = S,\ x_2 = P,\ x_3 = A,\ x_4 = J,\ x_5 = V$ are employed. The continuity and the boundedness of $g_1,\ g_2,\ g_3,\ g_4$, and g_5 are verified as here under:

Table 3 Verification of Continuity and Boundedness of the Functions

Function	Existence and Continuity	Boundedness
g_1	$(\partial g_1)/(\partial S) = -[\beta P(t)+\mu]$ $(\partial g_1)/(\partial P) = -\beta S(t)$ $(\partial g_1)/(\partial A) = 0$ $(\partial g_1)/(\partial J) = 0$ $(\partial g_1)/(\partial V) = 0$	$\lvert(\partial g_1)/(\partial S)\rvert = \lvert-[\beta P(t)+\mu]\rvert < \infty$ $\lvert(\partial g_1)/(\partial P)\rvert = \lvert-\beta S(t)\rvert < \infty$ $\lvert(\partial g_1)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_1)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_1)/(\partial V)\rvert = 0 < \infty$
g_2	$(\partial g_2)/(\partial S) = \beta P(t)$ $(\partial g_2)/(\partial P) = \beta S(t) - (\kappa+\eta+\mu)$ $(\partial g_2)/(\partial A) = 0$ $(\partial g_2)/(\partial J) = 0$ $(\partial g_2)/(\partial V) = 0$	$\lvert(\partial g_2)/(\partial S)\rvert = \lvert\beta P(t)\rvert < \infty$ $\lvert(\partial g_2)/(\partial P)\rvert = \lvert\beta S(t) - (\kappa+\eta+\mu)\rvert < \infty$ $\lvert(\partial g_2)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_2)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_2)/(\partial V)\rvert = 0 < \infty$
g_3	$(\partial g_3)/(\partial S) = 0$ $(\partial g_3)/(\partial P) = \kappa$ $(\partial g_3)/(\partial A) = -(\theta+\mu)$ $(\partial g_3)/(\partial J) = \omega$ $(\partial g_3)/(\partial V) = 0$	$\lvert(\partial g_3)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_3)/(\partial P)\rvert = \kappa < \infty$ $\lvert(\partial g_3)/(\partial A)\rvert = \theta+\mu < \infty$ $\lvert(\partial g_3)/(\partial J)\rvert = \omega < \infty$ $\lvert(\partial 3)/(\partial V)\rvert = 0 < \infty$
g_4	$(\partial g_4)/(\partial S) = 0$ $(\partial g_4)/(\partial P) = \eta$ $(\partial g_4)/(\partial A) = \theta$ $(\partial g_4)/(\partial J) = -(\omega+\gamma+\mu)$ $(\partial g_4)/(\partial V) = 0$	$\lvert(\partial g_4)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_4)/(\partial P)\rvert = \eta < \infty$ $\lvert(\partial g_4)/(\partial A)\rvert = \theta < \infty$ $\lvert(\partial g_4)/(\partial J)\rvert = \omega+\gamma+\mu < \infty$ $\lvert(\partial g_4)/(\partial V)\rvert = 0 < \infty$
g_5	$(\partial g_5)/(\partial S) = 0$ $(\partial g_5)/(\partial P) = 0$ $(\partial g_5)/(\partial A) = 0$ $(\partial g_5)/(\partial J) = \gamma$ $(\partial g_5)/(\partial V) = -(\rho+\mu)$	$\lvert(\partial g_5)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial P)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial J)\rvert = \gamma < \infty$ $\lvert(\partial g_5)/(\partial V)\rvert = \rho+\mu < \infty$

Thus, all the partial derivatives $(\partial f_i)/(\partial x_j)$: $i,\ j = 1,\ 2,\ 3,\ 4$ exist, and are both continuous and bounded in R. Hence, by Derrick and Groosman theorem, a solution for the model (1) – (5) exists and is unique. Therefore, the formulated model is biologically meaningful and mathematically well-posed.

3.2 Steady state solutions

In order to understand the dynamics of the model, it is necessary to determine equilibrium points of the solution region. An equilibrium solution is a steady state solution of the model equations (1) – (5) in the sense that if the system begins at such a state, it will remain there for all times. In other words, the population sizes remain unchanged and thus the rate of change for each population vanishes. Equilibrium points of the model are

found, categorized, stability analysis is conducted and the results have been presented in the following sub-sections:

3.2.1 Disease free equilibrium point

Disease free equilibrium point is a steady state solution where there is no disease in the population. Now, absence of disease implies that $P(t) = A(t) = J(t) = V(t) = 0$ and also setting the right hand sides of the model equations (1) – (5) equal to zero results in giving $\tau - \mu S = 0$, solution of which is the population size of the susceptible humans at the disease free equilibrium and is given by$S^0 = (\tau/\mu)$.Thus, the disease free equilibrium point of the model equations(1) – (5) is given by

$$E_0 = (S^0, \quad P^0, \quad A^0 \quad J^0, \quad V^0)$$

3.2.2 Endemic equilibrium point

The endemic equilibrium point $E_1 = \{S^1, \quad P^1, \quad A^1, \quad J^1, \quad V^1\}$ is a steady state solution when the disease persists in the population. The endemic equilibrium point is obtained by setting rates of changes of variables with respect to time of model equations (1) – (5) to zero. That is, setting $dS/dt = dA/dt = dI/dt = dV/dt = 0$ the model equations take the form as

$$\tau - \beta S(t)P(t) - \mu S(t) = 0 \quad (6)$$
$$\beta S(t)P(t) - bP(t) = 0 \quad (7)$$
$$\kappa P(t) + \omega J(t) - aA(t) = 0 \quad (8)$$
$$\eta P(t) + \theta A - cJ(t) = 0 \quad (9)$$
$$\gamma J(t) - dV(t) = 0 \quad (10)$$

Here in (6) – (10), the quantities $a, \ b, \ c$ represent the parametric expressions as $b = \theta + \mu, \ a = \kappa + \eta + \mu, \ c = \omega + \gamma + \mu, \ d = \rho + \mu$. Clearly, solutions of (6) – (10) will provide endemic equilibrium of the model equations and that is obtained as follows:

(i) The equations (7) can be rearranged as $[\beta S - a]P = 0$ leading to the solutions$\beta S - a = 0$ or $P = 0$ or both. However, P does not vanish since the disease is assumed to persist. Thus, it leads to the only meaningful solution$\beta S - b = 0$ or equivalently $S = (b/\beta)$. That is, the S^1 component of$E_1$is given by

$$S^1 \equiv S = (a/\beta) = (\tau/\mu R_0) \quad (11)$$

(ii) Now the solution for P can be obtained by substituting equation (11) into equation (7) and rewriting the resulting equation as $\tau - \beta(\tau/\mu R_0)P - \mu(\tau/\mu R_0) = 0$ giving

$$P^1 \equiv P = (\mu/\beta)(R_0 - 1) \quad (12)$$

(iii) Again the solution for A can be obtained by substituting P value from (11) into equations (8) and (9). Now, the reduced expression has the form

$$A^1 \equiv A = \{[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - bc)\} \quad (13)$$

(iv) By substituting A and P values into equation (8) the solution for J can be obtained and is given as

$$J^1 \equiv J = \left\{\{b[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - ac)\} - \kappa(\mu/\beta)(R_0 - 1)\right\}/\omega \quad (14)$$

(v) Finally, using the value of J from (14) and solving for V from equation (10) we have

$$V' \equiv V = \gamma J^1/d = \left\{\{\gamma\, b[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - bc)\} - \kappa(\mu/\beta)(R_0 - 1)\right\}/d\omega \quad (15)$$

3.3 Basic Reproduction Number

The basic reproduction number is denoted by R_0 and is defined as the expected number of people getting secondary infection because of infected person enters into wholly susceptible population [11]. This number determines the potential for the spread of disease within a population. When$R_0 < 1$ each infected individual produces on average less than one new infected individual so that the disease is expected to die out. On the other hand if $R_0 > 1$ then each individual produces more than one new infected individual so that the disease is expected to continue spreading in the population. This means that the threshold quantity for eradicating the disease is to reduce the value of R_0 to less than one.

The basic reproductive number R_0 can be determined using the next generation matrix. In this method$R_0$is defined as the largest eigenvalue of the next generation matrix. The formulation of this matrix involves classification of all compartments of the model in to two classes: infected and non-infected. That is, the basic reproduction number cannot be determined from the structure of the mathematical model alone but depends on the definition of infected and uninfected compartments.

Assume that there are n compartments in the model and of which the first m compartments are with infected individuals [3]. From the system (1) – (5) the four equations of infected individuals are considered and decomposed into two groups: F contains newly infected cases and v contains the remaining terms. Let

$X = [S,\ P,\ A,\ J,\ V]^t$ be a column vector and the differential equations of the first four compartments are rewritten as $F(X) - T(X)$.

Now, let $F(X) = [F_1,\ F_2,\ F_3,\ F_4]^t$. Here (i) $F_1 = (\beta SP)$ denotes newly infected cases which arrive into primary infected compartment (ii) $F_2 = 0$ denotes newly infected cases arrived into the infectious asymptomatic compartment (ii) $F_3 = 0$ denotes newly infected cases arrived into the infectious symptomatic compartment, and (iii) $F_4 = 0$ denotes newly infected case from susceptible compartment into AIDS compartment. Further, let $T(X) = [T_1,\ T_2,\ T_3,\ T_4]^t$. Here $T_1 = aP,\ T_2 = -\kappa P - \omega J + bA,\ T_3 = -\eta P - \theta A + cJ$, and $T_4 = -\gamma J + dV$. Here, the parameters $a,\ b,\ c,\ d$ represent the expressions as $b = \theta + \mu,\ \ a = \kappa + \eta + \mu,\ \ c = \omega + \gamma + \mu,\ \ d = \rho + \mu$.

The next step is the computation of square matrices F and T of order $m \times m$, where m is the number of infected classes, defined by $F = \left[\partial F_i(E_0)/\partial x_j\right]$ and $T = \left[\partial T_i(E_0)/\partial x_j\right]$ with $1 \le i,\ j \le m$, such that F is non-negative, V is a non-singular matrices and E_0 is the disease free equilibrium point DFE. If F and T are non-negative and T is non-singular then T^{-1} is non-negative and thus FT^{-1} is also non-negative. Also, the matrix FT^{-1} is called the next generation matrix for the model. Finally, the basic reproduction number R_0 is given by $R_0 = \rho(\mathrm{F}T^{-1})$. In general, $\rho(A)$ denotes the spectral radius of matrix A and the spectral radius is the biggest non-negative eigenvalue of the next generation matrix.

The Jacobian of F and T at the disease free equilibrium point E_0 takes the form respectively as

$$F \equiv J_F(E_0) = \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } T \equiv J_T(E_0) = \begin{bmatrix} a & 0 & 0 & 0 \\ -\kappa & b & -\omega & 0 \\ -\eta & -\theta & c & 0 \\ 0 & 0 & -\gamma & d \end{bmatrix} \qquad (16)$$

It can be verified that the matrix $J_T(E_0)$ is non-singular as its determinant is non-zero and after some algebraic computations the next generation matrix is constructed as

$$[J_F(E_0)][J_V(E_0)]^{-1} = \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} a & 0 & 0 & 0 \\ -\kappa & b & -\omega & 0 \\ -\eta & -\theta & c & 0 \\ 0 & 0 & -\gamma & d \end{bmatrix}^{-1} = \begin{bmatrix} \beta\tau/\mu\, a & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Now, it is possible to calculate the eigenvalues of the matrix $[J_F(E_0)][J_T(E_0)]^{-1}$ to determine the basic reproduction number R_0 which is the spectral radius or the largest eigenvalue. Thus, the eigenvalues are computed by evaluating the characteristic equation $det\left[[J_F(E_0)][J_T(E_0)]^{-1} - \lambda I\right] = 0$ or equivalently solving

$$\begin{vmatrix} (\beta\tau/\mu\, a) - \lambda & 0 & 0 & 0 \\ 0 & -\lambda & -\lambda & 0 \\ 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & -\lambda \end{vmatrix} = 0$$

It reduces to the equation as $\lambda^3[(\beta\tau/\mu\, a) - \lambda] = 0$ giving the four eigenvalues as

$$\lambda_1 = (\beta\tau/\mu\, a), \quad \lambda_2 = 0, \quad \lambda_3 = 0, \quad \lambda_4 = 0$$

However, the largest eigenvalue here is and is the spectral radius or the threshold value or the basic reproductive number. Thus, the reproduction number of the model is $R_0 = (\beta\tau/\mu\, a)$.

3.4 Stability analysis of the disease free equilibrium

In absence of the infectious disease, the model populations have a unique disease free steady stateE_0. To find the local stability of E_0, the Jacobian method of the model equations evaluated at DEF $E_0$is used. Also, to determine the global stability at E_0 M-matrix method given in [10] is used. It is already shown that the DFE of model (1) – (5) is given by $E_0 = \{\tau/\mu,\ \ 0,\ \ 0,\ \ 0\}$. Now, following [5, 6, 7, 8] the stability analysis of DFE is conducted and the results are presented in the form of theorems and proofs in the following:

3.4.1 Local Stability of Disease Free Equilibrium point

Theorem 1: The DFE E_0 of the system (1) – (5) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: Consider the right hand side expressions of the equations (1) – (5) as functions so as to find the Jacobian matrix as follows:

$$dS/dt = \tau - \beta S(t)P(t) - \mu S(t) \equiv g_1(S,\ P,\ A,\ J\ V)$$
$$dP/dt = \beta S(t)P(t) - (\kappa + \eta + \mu)P(t) \equiv g_2(S,\ P,\ A,\ J,\ V)$$
$$dA/dt = \kappa P(t) + \omega J(t) - (\theta + \mu)A(t) \equiv g_3(S,\ P,\ A,\ J,\ V)$$
$$dJ/dt = \eta P(t) + \theta A - (\omega + \gamma + \mu)J \equiv g_4(S,\ P,\ A,\ J,\ V)$$
$$dV/dt = \gamma J(t) - (\rho + \mu)V \equiv g_5(S,\ P,\ A,\ J,\ V)$$

Let $J(S,\ P,\ A,\ J,\ V)$ be a Jacobian matrix of $g_1,\ g_2,\ g_3,\ g_4,\ g_5$ with respect to $S,\ P,\ A,\ J,\ V$. Thus,

$$J(S,\ P,\ A,\ J,\ V) = \begin{bmatrix} -\beta P - \mu & -\beta S & 0 & 0 & 0 \\ \beta P & \beta S - (\kappa + \eta + \mu) & 0 & 0 & 0 \\ 0 & \kappa & -(\theta + \mu) & \omega & 0 \\ 0 & \eta & \theta & -(\omega + \gamma + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -(\rho + \mu) \end{bmatrix}$$

Now, the Jacobian matrix of $g_1,\ g_2,\ g_3,\ g_4,\ g_5$ with respect to $S,\ P,\ A,\ J,\ V$ at the disease free equilibrium E_0 is given by

$$J(E_0) = \begin{bmatrix} -\mu & -\beta\tau/\mu & 0 & 0 & 0 \\ 0 & \beta\tau/\mu - a & 0 & 0 & 0 \\ 0 & \kappa & -b & \omega & 0 \\ 0 & \eta & \theta & -c & 0 \\ 0 & 0 & 0 & \gamma & -d \end{bmatrix}$$

Now, the eigenvalues of $J(E_0)$ are required to be found. The corresponding characteristic equation $det[J(E_0) - \lambda I] = 0$ is expanded and simplified as follows:

$$\begin{vmatrix} -\mu - \lambda & -\beta\tau/\mu & 0 & 0 & 0 \\ 0 & \beta\tau/\mu - a - \lambda & 0 & 0 & 0 \\ 0 & \kappa & -b - \lambda & \omega & 0 \\ 0 & \eta & \theta & -c - \lambda & 0 \\ 0 & 0 & 0 & \gamma & -d - \lambda \end{vmatrix} = 0$$

$$-(\mu + \lambda)(-d - \lambda)(\beta\tau/\mu - a - \lambda)[(-b - \lambda)(-c - \lambda) - \theta\omega] = 0$$

Thus, the five eigenvalues of the matrix $J(E_0)$are determined as

$$\lambda_1 = -\mu$$
$$\lambda_2 = -d$$
$$\lambda_3 = (\beta\tau/\mu) - a$$
$$\lambda_{4,5} = \frac{-(\theta + \omega + \gamma + 2\mu) \pm \sqrt{\theta^2 + 2\theta\omega - 2\theta\gamma + \omega^2 + 2\omega\gamma + \gamma^2}}{2}$$

It can be observed that all eigenvalues are absolutely negative quantities. Therefore, following the procedure given in [1, 10], it can be concluded that the DFE E_0 of the system of differential equations (1) – (5) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

3.4.2 Global Stability of Disease Free Equilibrium Point

Following the procedure given in [11] let $x \in R^n$ is disease compartment and $y \in R^m$ be disease free compartment the disease transmission model (1) – (5) can be written in the form:

$$\dot{x} = -(T - F)x - h(x, y) \qquad (17)$$
$$\dot{y} = g(x, y) \qquad (18)$$

Here in (17) the notations $F = J_F(E_0)$ and $T = J_T(E_0)$ are used.

Theorem 2: If $T - F$ is a nonsingular M-matrix and $h \geq 0$ then the disease-free equilibrium point of model equations (1) – (5) is globally asymptotically stable.

Proof: Using the procedure given in [10] the rate of change of the variables in the model equations (1) – (5) can be rewritten as

$$\dot{x} = -(T - F)x - \begin{bmatrix} \beta(S_0 - S)P \\ \\ 0 \end{bmatrix}$$
$$\dot{S} = \tau - \beta SP - \mu S$$

Now, it is to be shown that $T - F$ is non-singular M-matrix. From the previous computations (16) we have

$$F = J_F(E_0) = \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } T = J_T(E_0) = \begin{bmatrix} a & 0 & 0 & 0 \\ -\kappa & b & -\omega & 0 \\ -\eta & -\theta & c & 0 \\ 0 & 0 & -\gamma & d \end{bmatrix}.$$

$$T - F = \begin{bmatrix} a - \beta\tau/\mu & 0 & 0 & 0 \\ -\kappa & b & -\omega & 0 \\ -\eta & -\theta & c & 0 \\ 0 & 0 & -\gamma & d \end{bmatrix} = sI - \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 \\ \kappa & 0 & \omega & 0 \\ \eta & \theta & 0 & 0 \\ 0 & 0 & \gamma & 0 \end{bmatrix} = sI - B.$$

Here, $s = \max(a, \quad b, \quad c, \quad d)$ and $B = \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 \\ \kappa & 0 & \omega & 0 \\ \eta & \theta & 0 & 0 \\ 0 & 0 & \gamma & 0 \end{bmatrix}$

Now, $\det(T - F) = (\text{bcd} - \text{d}\theta\omega)[a - \beta(\tau/\mu)]$ and $\rho(B) = max\{\beta(\tau/\mu), \quad \sqrt{\theta\omega}\}$ and $T - F$ is nonsingular matrix provided that the conditions $\text{bcd} \neq \text{d}\theta\omega$ and $a \neq \beta\tau/\mu$ are satisfied. Further, off diagonal elements of $T - F$ are non-positive numbers. Thus, $T - F$ is non-singular M-matrix if $s \geq \rho(B)$.

Next, to show that the disease-free equilibrium is globally asymptotically stable for $R_0 < 1$, it is sufficient to show that $S \leq S_0$. The total population $N(t) = S(t) + P(t) + A(t) + J(t) + V(t)$ satisfy the condition $N' = \Pi - \text{dN} - dV \leq \Pi - \text{dN}$, so that $N(t) \leq S_0 - \big(S_0 - N(0)\big)e^{-\mu t}$, with $S_0 = \pi/\mu$. If $N(0) \leq S_0$, then $S(t) \leq N(t) \leq S_0$ for all time, if, on the other hand, $N(0) > S_0$, then $N(t)$decays exponentially to S_0, and either $S(t) \to S_0$, or there is some time T after which $S(t) < S_0$. Thus, from time T' onward, $x(t)$ is bounded above, in each component, by $e^{-(t-T')(T-F)}x(T')$ which decays exponentially to zero. Note that for the argument of global stability we are not concerned with the size of $x(t)$.

In fact, if $N(0) > S_0$, $x(T')$ may be much larger than $x(0)$. In this case the exponential bound on $x(t)$ concerns a decay following an epidemic, not an immediate elimination of the disease. In contrast, if $N(0) < S_0$, then the bound on $x(t)$ is $e^{-(t-T')(T-F)}x(0)$and no epidemic occurs. Therefore, from the above hypothesis disease-free equilibrium point of model equations (1-5) is globally asymptotically stable for$R_0 < 1$.

3.5 Stability Analysis of Endemic Equilibrium Point

By definition it is true that at the endemic equilibrium point $E_1 = \{S^1, \quad P^1, \quad A^1, \quad J^1, \quad V^1\}$ is the point where the disease persists or exists. To analyze the local stability ofE_1, Jacobian matrix of the model that evaluated at this equilibrium point is used. Further, remember that the endemic equilibrium point$E_1 = \{S^1, \quad P^1, \quad A^1, \quad J^1, \quad V^1\}$ of the given model (1) – (5) is already computed.

3.5.1 Local Stability of Endemic Equilibrium Point

The local stability of endemic equilibrium point is stated and proved in Theorem 3.

Theorem 3: The endemic equilibrium point is locally asymptotically stable if$R_0 > 1$ and unstable if$R_0 < 1$.

Proof: The stability analysis of $E_1$is conducted by following the similar procedure adopted as in the case ofE_0. Thus, the procedure starts with the construction of Jacobian matrix atE_1. Now, the Jacobian matrix of the model given in (15) at endemic equilibrium point E_1 takes the form as

$$S^1 \equiv S = b/\beta = (\tau/\mu R_0)$$
$$P^1 \equiv P = (\mu/\beta)(R_0 - 1)$$
$$A^1 \equiv A = \{[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - ac)\}$$
$$J^1 \equiv J = \big\{\{a[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - ac)\} - \kappa(\mu/\beta)(R_0 - 1)\big\}/\omega$$
$$V' \equiv V = \gamma J^1/d = \big\{\{\gamma\, a[(\kappa c + \eta\omega)(\mu/\beta)(R_0 - 1)]/(\theta\omega - ac)\} - \kappa(\mu/\beta)(R_0 - 1)\big\}/d\omega$$

$$J = J(S,\ P,\ A,\ J,\ V) = \begin{bmatrix} -\beta P - \mu & -\beta S & 0 & 0 & 0 \\ \beta P & \beta S - (\kappa + \eta + \mu) & 0 & 0 & 0 \\ 0 & \kappa & -(\theta + \mu) & \omega & 0 \\ 0 & \eta & \theta & -(\omega + \gamma + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -(\rho + \mu) \end{bmatrix}$$

$$J = J(E_1) = \begin{bmatrix} -\mu R_0 & -\beta(\tau/\mu R_0) & 0 & 0 & 0 \\ \mu(R_0 - 1) & 0 & 0 & 0 & 0 \\ 0 & \kappa & -(\theta + \mu) & \omega & 0 \\ 0 & \eta & \theta & -(\omega + \gamma + \mu) & 0 \\ 0 & 0 & 0 & \gamma & -(\rho + \mu) \end{bmatrix}$$

Now the trace of $J(E_1)$is a negative quantity while determinant of $J(E_1)$ computed as $-\beta\tau(R_0 - 1\rho\mu 2 + \theta\mu 2 + \mu 2\omega + \mu 2\gamma + \mu 3 + \rho\theta\mu + \rho\mu\omega + \rho\theta\gamma + \rho\mu\gamma + \theta\mu\gamma R0$ and is a positive quantity provided that $R0<1$.

Hence, the endemic equilibrium point $E_1$is locally asymptotically unstable if $R_0 < 1$.

IV. Numerical Simulation

In this section, numerical simulation study of model equations (1) – (5) is carried out using the software MATLAB. To conduct the study, a set of physically meaningful values are assigned to the model parameters. These values are either taken from literature or assumed on the basis of reality. These sets of parametric values are given under figures.

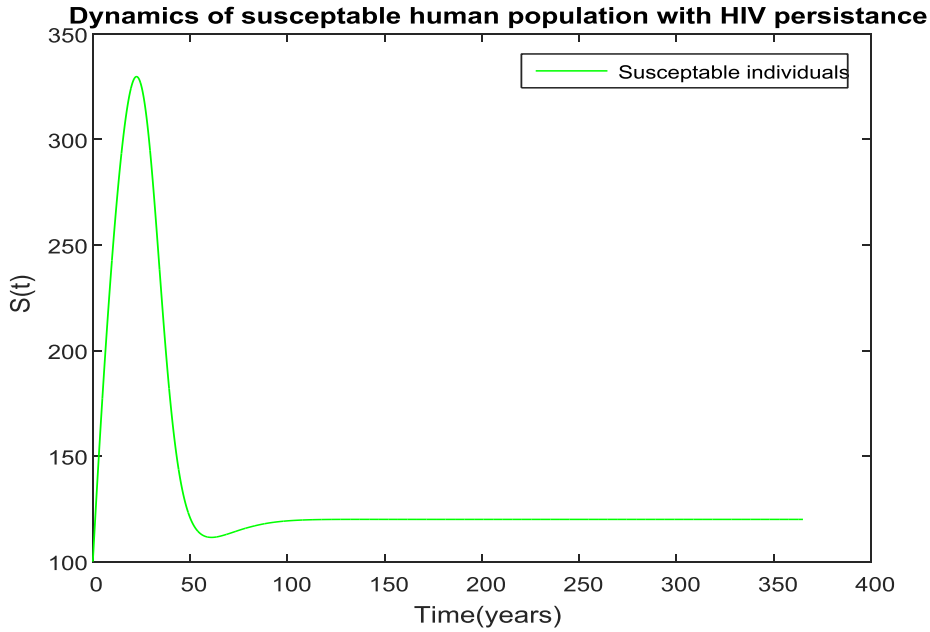

Figure 1Dynamics of susceptible population with parametric values $\boldsymbol{\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06}$

From the simulated graph given in Figure 1, it can be observed that the susceptible population (i) increases initially because of less number of primary patients but (ii) decreases at a later time because of more number of primary patients.

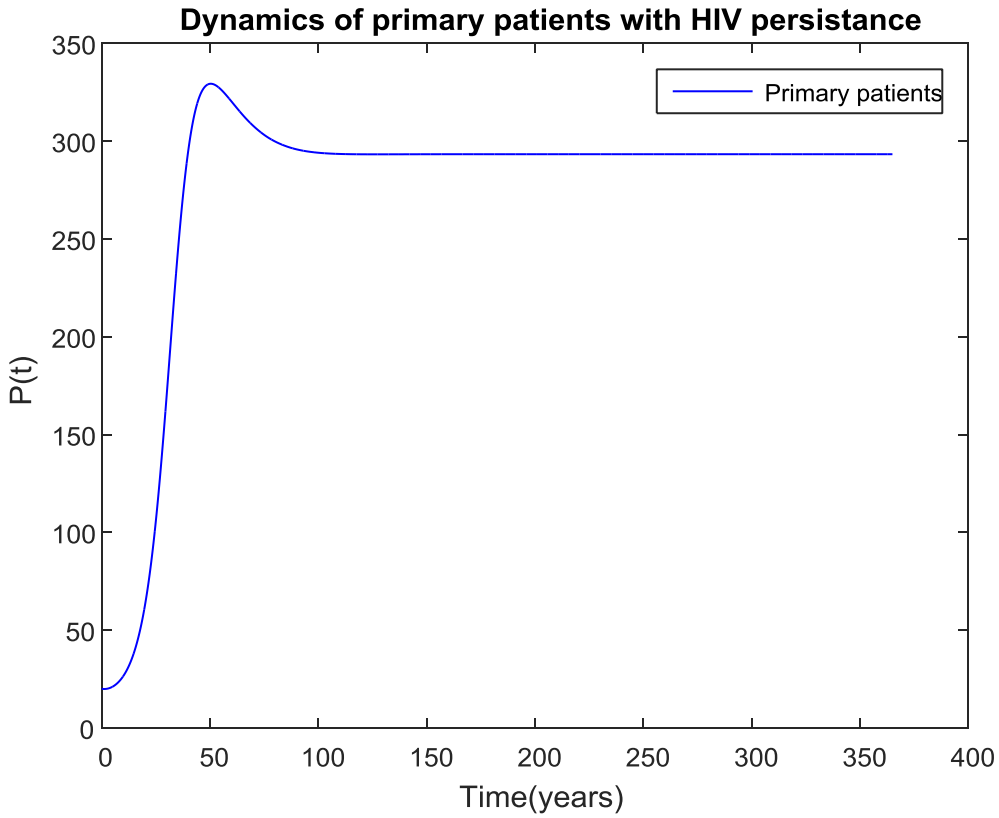

Figure 2 Dynamics of primary patients with parametric values
$\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\quad \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06$

Figure 2 shows that starting from the beginning the number of primary patient's increases as the result of delay in antibody of a patients or the patient do not want to know the result.

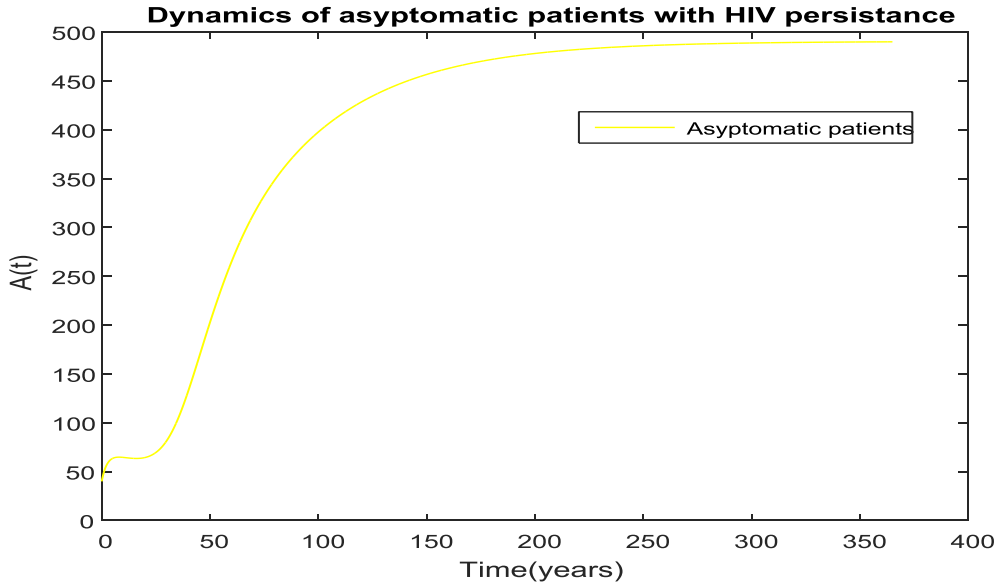

Figure 3 Dynamics of susceptible population with parametric values
$\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\quad \theta = 0.0007,\ \eta = 0.03,\quad \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\quad \gamma = 0.06$

From Figure 3 it can be observed that the asymptomatic patients increase as all patients want treatments and continue using it.

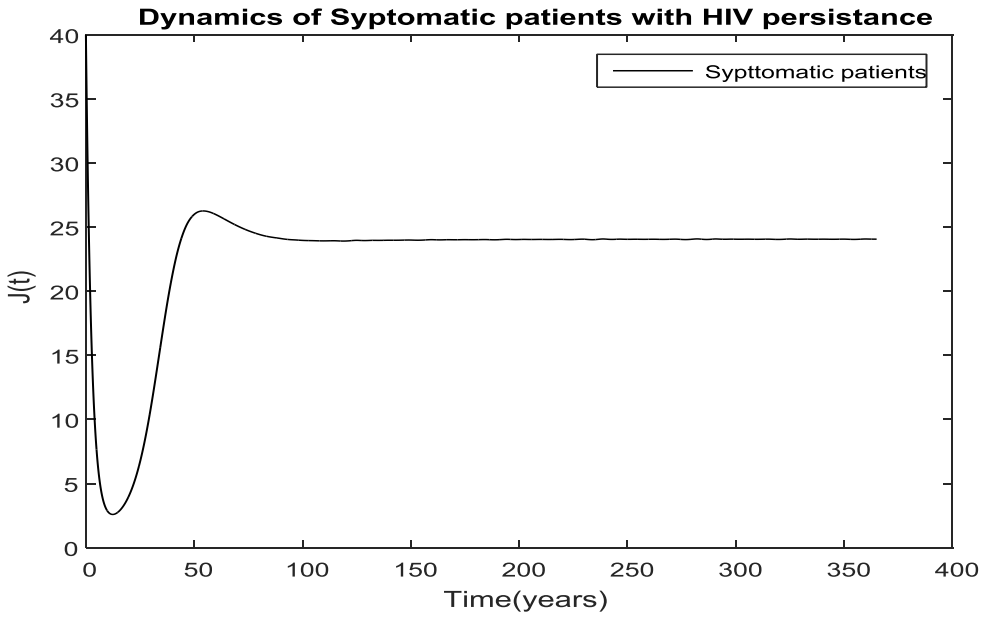

Figure 4 Dynamics of symptomatic patients with parametric values
$\boldsymbol{\tau = 20,\ \mu = 0.02,\quad \beta = 0.0005,\quad \theta = 0.0007,\quad \eta = 0.03,}$
$\boldsymbol{\omega = 0.3,\ \rho = 0.05,\ k = 0.01,\quad \gamma = 0.06}$

Figure 4 illustrates the dynamics of syptomatic patients that decrease initially as the result of less primary patients and syptomatic patients enters the compartement. The compartement also increases from some later point of time as some primary and asyptomatic patients want to use only herbal medicine as treatment.

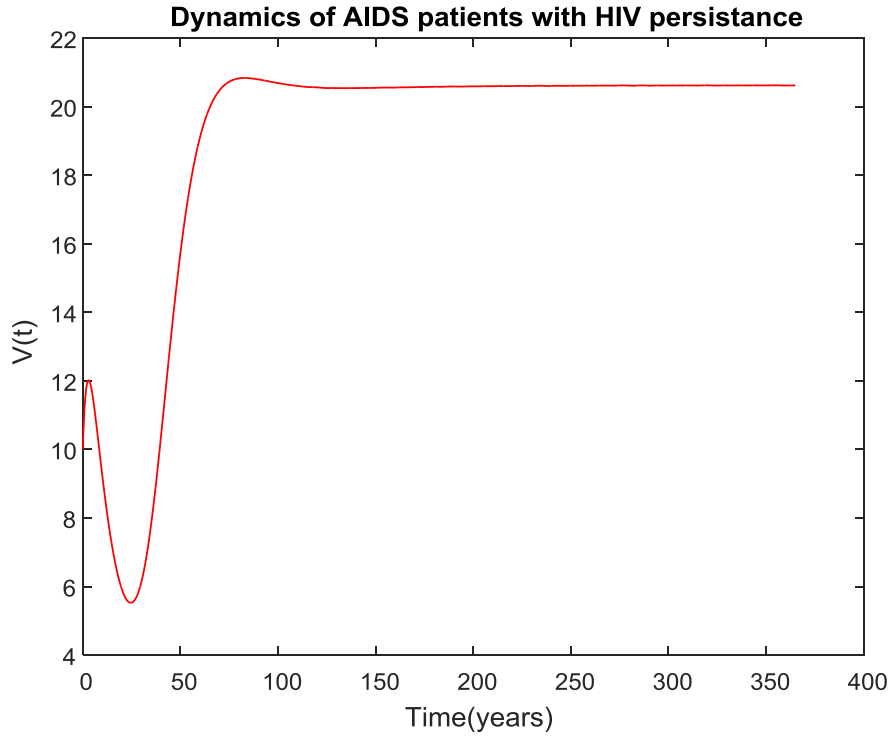

Figure 5 Dynamics of AIDS patients with parametric values
$\boldsymbol{\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\quad \theta = 0.0007,\ \eta = 0.03,\quad \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\quad \gamma = 0.06}$

Figure 5 illustrates the simulation of AIDS patients which increases initially as the resultof patients from syptomatic compartement enters it and decrease from there as result of death. Then increases as syptomatic patients increase.

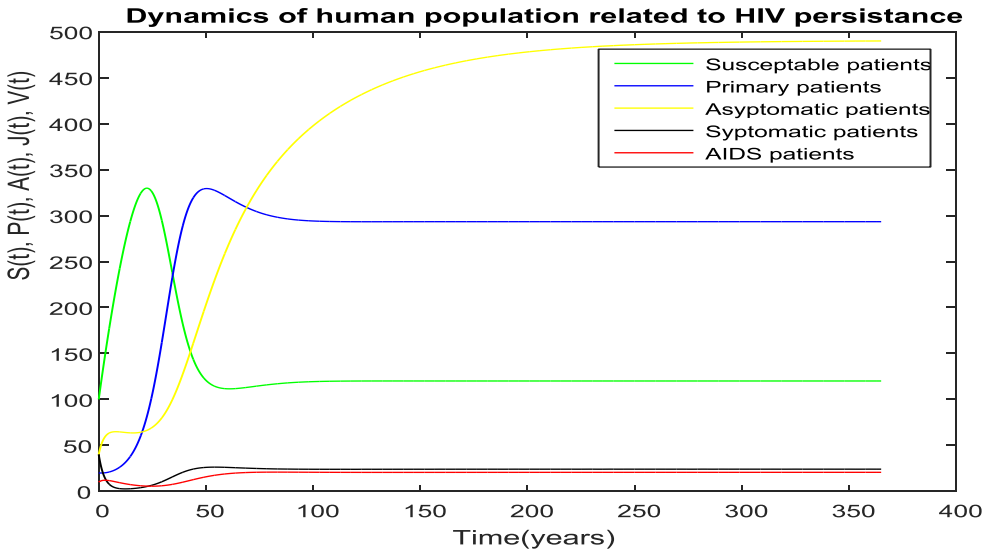

Figure 6 Dynamics of susceptible population with parametric values
$\boldsymbol{\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06}$

In Figure 6 the following can be observed: (i) initially the population size of susceptible compartment S is increasing because of recruitment rate and less number of primary patients. Then decreases followed by averagely constant in number as there are more primary patients followed with constant in number(ii) Initially the population size of primary patient's compartment P increase because of delay of instrument result or unwillingness of people to know their status. Then decrease followed by constant in number as there is a balance in population dynamics (iii) the asymptomatic patient's compartment increases as patients show no symptoms (iv) symptomatic compartment decreases initially as patients transfer to asymptomatic and AIDS compartment. Then increases followed with constant as the result of balances in the transfer of patients (v) The AIDS compartment increase initially because of patients from symptomatic compartment. Then decrease followed with constant number of patients as the result of balance of transfer in patients.

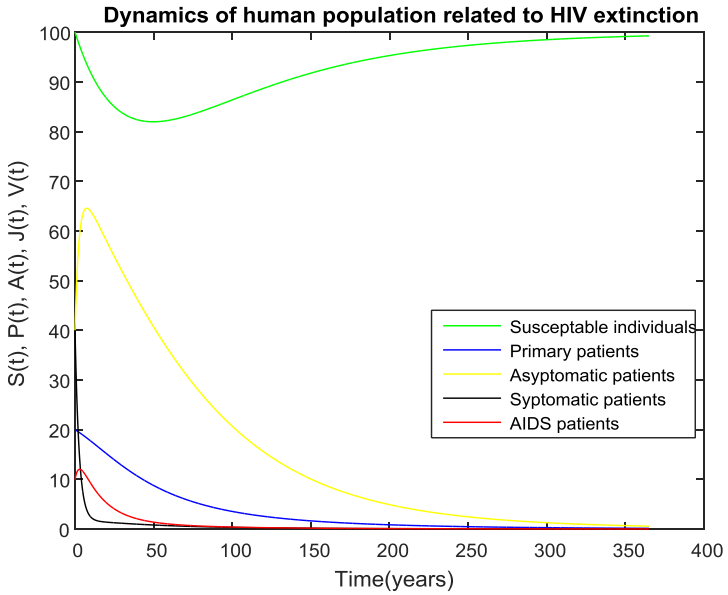

Figure 7 Population dynamics of $SPAJV$ compartments with the parametric values
$\boldsymbol{\tau = 2,\ \mu = 0.02,\ \beta = 0.00005,\ \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06}$

In figure 7, it can be observed that (i) initially the susceptible compartment decrease because of infection enter the compartment and finally increases as the patients come out of the compartment. (ii) The Primary compartment decreases as people tested and know their result. (iii)The asymptomatic compartment increases initially as people enters it from primary patients and symptomatic compartment. And decreases as the number of patients decreases (iv) the symptomatic compartment decreases starting from the beginning of less number of patients come into it from primary and asymptomatic compartments. (v) The AIDS compartment initially as patients from symptomatic compartment enters it and decrease continuously in the decrement of patients in the symptomatic compartment in addition to natural death disease induced death rate.

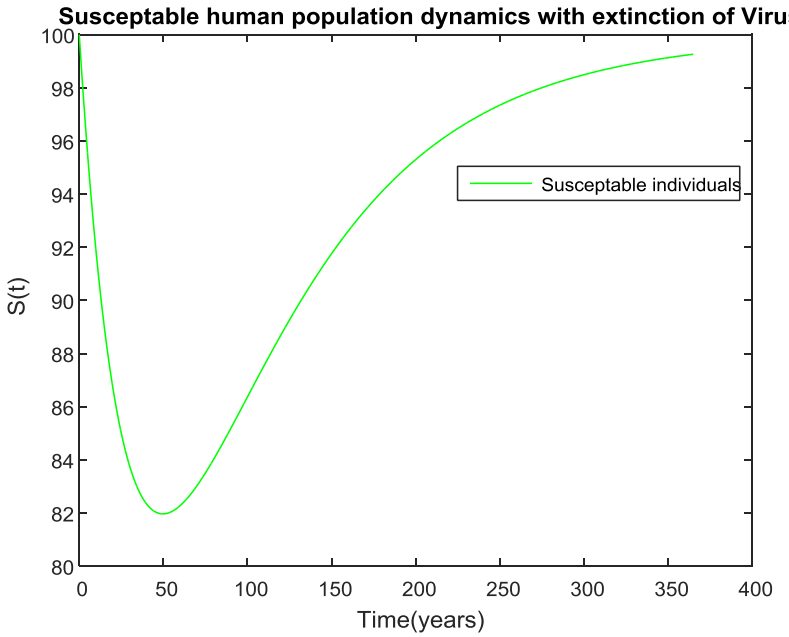

Figure 8 Population dynamics of susceptible compartment with the parametric values $\boldsymbol{\tau = 2,\ \mu = 0.02,\ \beta = 0.00005,\ \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06}$

Figure 8 illustrates for about fifty years the susceptible population are decreasing as the result of contact with primary. Finally, they increased as the number of primary patients decreased.

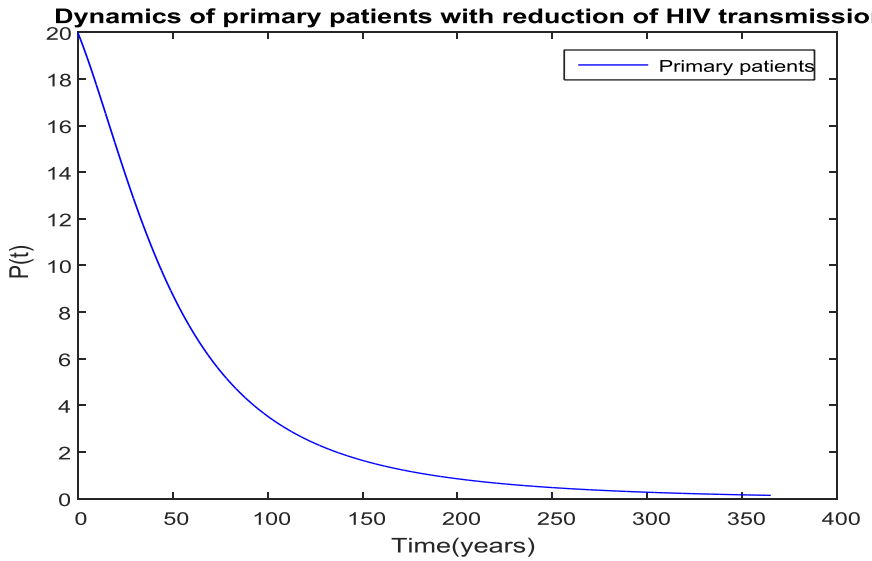

Figure 9 Population dynamics of primary compartment with the parametric values $\boldsymbol{\tau = 2,\ \mu = 0.02,\ \beta = 0.00005,\ \theta = 0.0007,\ \eta = 0.03,\ \omega = 0.3,\ \rho = 0.05,\ k = 0.01,\ \gamma = 0.06}$

Figure 9 illustrates the primary patients are decreasing as the result of contact with susceptible decreases.

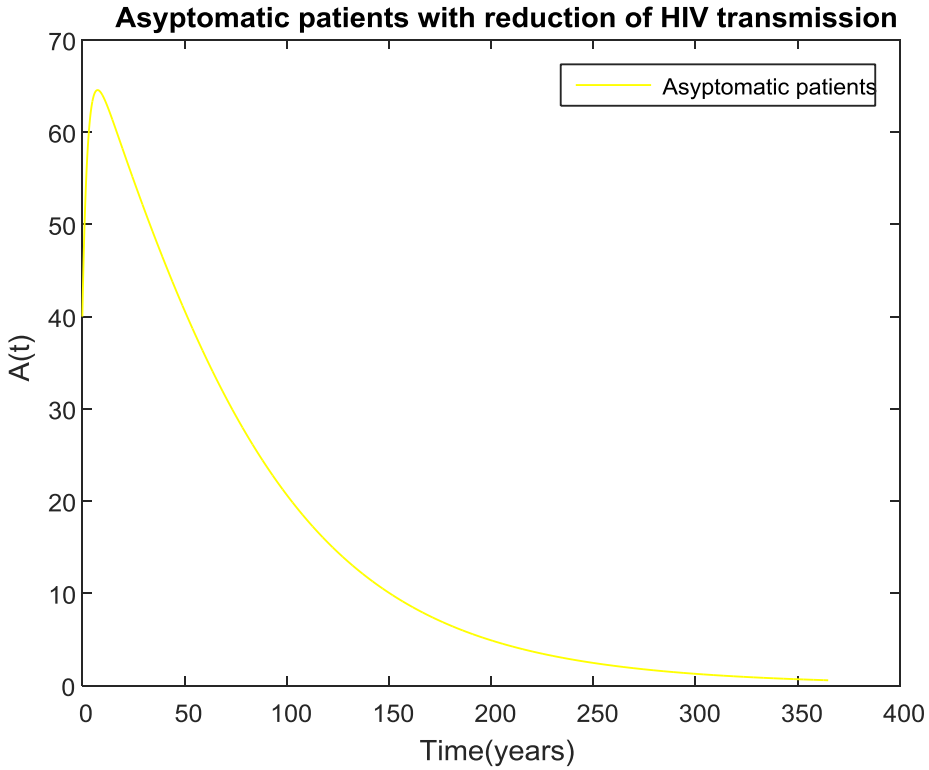

Figure 10 Population dynamics of asymptomatic compartment with the parametric values $\tau = 2$, $\mu = 0.02$, $\beta = 0.00005$, $\theta = 0.0007$, $\eta = 0.03$, $\omega = 0.3$, $\rho = 0.05$, $k = 0.01$, $\gamma = 0.06$

Figure 10 shows that initially more patients join asymptomatic compartment and increased it. Finally, as the number of primary patients decreased the asymptomatic compartment also decreasing to zero at the end.

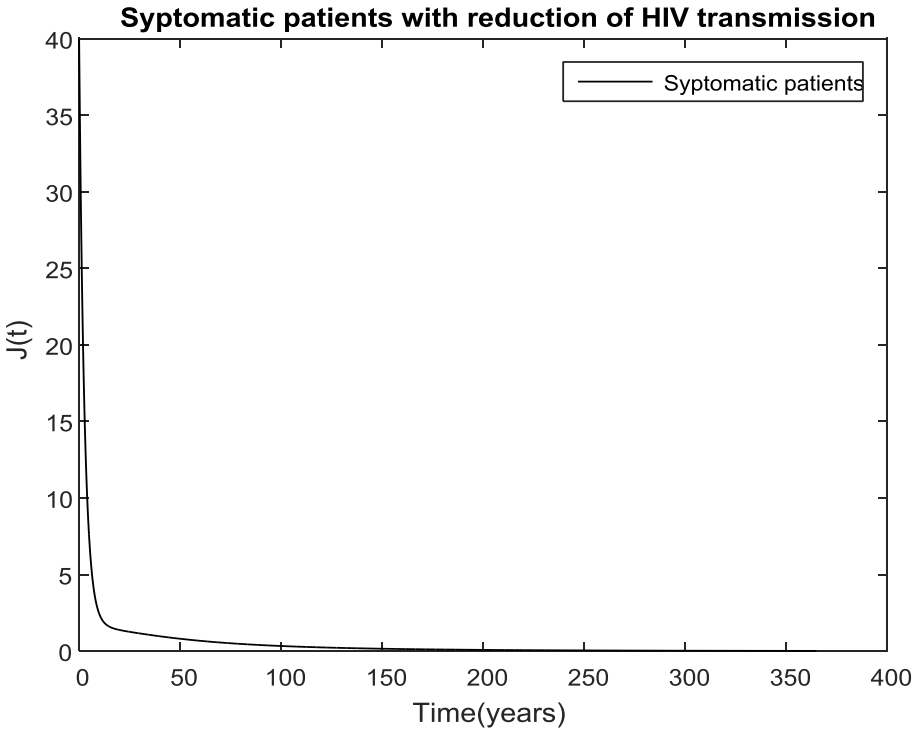

Figure 11 Population dynamics of symptomatic compartment with the parametric values $\tau = 2$, $\mu = 0.02$, $\beta = 0.00005$, $\theta = 0.0007$, $\eta = 0.03$, $\omega = 0.3$, $\rho = 0.05$, $k = 0.01$, $\gamma = 0.06$

Figure 11 Illustrates the fact that as the number of primary patients gets decreasing the number of patients in symptomatic patients also decreases to zero at the end of the time.

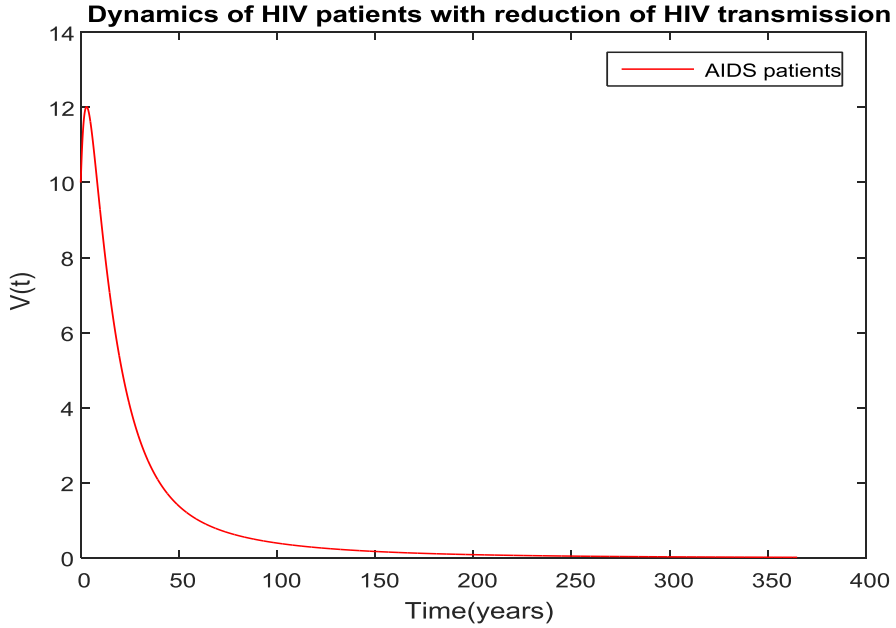

Figure 12 Population dynamics of AIDS compartment with the parametric values $\tau = 2$, $\mu = 0.02$, $\beta = 0.00005$, $\theta = 0.0007$, $\eta = 0.03$, $\omega = 0.3$, $\rho = 0.05$, $k = 0.01$, $\gamma = 0.06$

Figure 12 shows that initially AIDS patients get increasing as there be patients who did not fit with the treatment. Finally, as the number of patients decreased in all compartments the AIDS compartment patients also get decreasing leads to zero.

The differences and similarities between the existing and modified model are given respectively in the tables 1 and 2.

Table 1 Differences of existing and modified model.

Differences		
SN	**Existing model [2]**	**Modified (Present) model**
1	Has four compartments	Has five compartments
2	Has susceptible, symptomatic, asymptomatic, AIDS human population compartments	Has susceptible, primary, asymptomatic, symptomatic , AIDS human population's compartments
3	Assumed education and treatment	Assumed treatment

Table 2 Similarities of existing and modified model

Similarities	
Both existing **[2]** and modified (Present) model have the following similarities	
i)	Disease induced death rate in AIDS patients
i)	Transmission rate
i)	Natural mortality
v)	Transfer of patients from symptomatic to asymptomatic

V. Sensitivity Analysis

Sensitivity analysis is used to determine the sensitivity of the model with respect to the parameters involved in it. That is, how changes in the value of the parameters of the model result in changing the dynamics of the infection. It is used to discover parameters that have a high impact on $R_0$and should be targeted by intervention strategies. More precisely, sensitivity indices allow measuring the relative change in a variable when parameter changes [10]. If the result is negative, then the relationship between the parameters and $R_0$is inversely proportional. In this case, the modulus of the sensitivity index will be taken so that the size of the effect of changing that parameter can be deduced.

On the other hand, a positive sensitivity index means that both the function and the parameter are proportional to each other i.e. both of them grow or decay together.

It is already shown that the explicit expression of $R_0$is given by$R_0 = [(\beta\tau)/\mu(\eta + \kappa + \mu)]$. Since,R_0depends only on four parameters, an analytical expression will be derived for its sensitivity to each of the parameters using the normalized forward sensitivity index as given by Chitnis [10].

$$\Upsilon_{\beta}^{R_0} = [\partial R_0/\partial\beta] \times [\beta/R_0]$$
$$\Upsilon_{\mu}^{R_0} = [\partial R_0/\partial\mu] \times [\mu/R_0]$$
$$\Upsilon_{\eta}^{R_0} = [\partial R_0/\partial\eta] \times [\eta/R_0]$$
$$\Upsilon_{\tau}^{R_0} = [\partial R_0/\partial\tau] \times [\tau/R_0]$$
$$\Upsilon_{\kappa}^{R_0} = [\partial R_0/\partial\kappa] \times [\kappa/R_0]$$

Table 4 Sensitivity of R_0 evaluated for the parametric values given under figure 1

Parameter	Sensitivity index
$\boldsymbol{\mu}$	-1
$\boldsymbol{\eta}$	-0.5
$\boldsymbol{\beta}$	+1
$\boldsymbol{\kappa}$	-0.116
$\boldsymbol{\tau}$	+1

From Table 3, it can be observed that the values of two parameters $\boldsymbol{\tau}$, $\boldsymbol{\beta}$ are positive sensitivity indices and values of the remaining three parameters $\boldsymbol{\eta}$, $\boldsymbol{\kappa}$, $\boldsymbol{\mu}$ get negative sensitivity indices.

As it is observed from the table the parameter with large magnitude are $\boldsymbol{\mu}$, $\boldsymbol{\tau}$, and $\boldsymbol{\beta}$. Hence, they are most sensitive parameter in the model equations. On the other hand an increase in these positive parameter values will cause an increasingR_0 this implies that disease transmission in human population. Similarly, a decrease in negative parameter values will cause a decrease inR_0 which means the disease transmission decreases in human population.

VI. Result and Discussion

In this study, a mathematical model describing the dynamics of five compartmental human population related to Human Immunodeficiency Virus (HIV) with treatment is formulated and analyzed. The model is developed based on biologically reasonable assumptions made about Human Immunodeficiency Virus (HIV) and its treatment. The mathematical analysis has shown that if the reproduction number $R_0 < 1$ then the disease free equilibrium point is locally and globally asymptotically stable implying that the disease transmission decreases with decreased recruitment rate value which is supported by the simulation results given in Figure 7-12. Also, if $R_0 > 1$ then the disease free equilibrium point is unstable implying that the transmission of disease increases. These theoretical results have been supported by the simulation study as it is shown in Figure 1-6.

VII. Conclusions

In this study, a mathematical model of five compartments related to Human Immunodeficiency Virus HIV with treatment has been formulated. Moreover, existence, positivity and boundedness of the formulated model are verified to illustrate that the model is biologically meaningful and mathematically well posed. In particular, the stability analyses of the model were investigated using the basic reproduction number and Routh Hurwitz criterion. Also, the solution of the model equations is numerically simulated and sensitivity analysis of the model is conducted. Furthermore, results of the research work presented in this paper reveal that the model formulated here effectively supports treatment for HIV disease.

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