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Impact of Treatment and Isolation on the Dynamics of HIV Transmission

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Abstract: *In this study, a dynamical system of ordinary differential equations has been formulated to describe to describe the dynamics of human population subjected to HIV disease. This study classifies human population into six compartments as susceptible class, primary class, asymptomatic class, symptomatic class, treatment class and AIDS (SPAJTV). A well-possedness of formulated dynamical system has been verified. Additionally, parametric expression of basic reproduction number has been constructed using next generation matrix method. The equilibrium points of formulated dynamical system are identified. Both Global stability and local stability of disease free equilibrium point has been analyzed. The local stability of endemic equilibrium point also analyzed using a reproduction number and Routh Hurwitz principle. A disease free equilibrium point is locally and globally stable for a reproduction number less than unity and unstable for greater than unity. Sensitivity analysis computation shows that death and recruitment rates are more sensitive in reproduction number. 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: *Global Stability, Local Stability, Basic Reproduction Number, Routh Hurwitz criterion, well-possedness, Simulation.*

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I. Introduction

HIV is an infectious disease that caused by a virus called Human Immunodeficiency Virus that leads to the advanced and most severity stage called AIDS which stands for Acquired Immunodeficiency Syndrome. Starting from the beginning to the end the virus HIV and its advanced AIDS focus in weaking the immune system of the body that fight against antihuman body as natural defense. Starting from its discovery HIV disease has brought a great impact in terms of socially, economically, culturally, morally, and physically in all over the world. As serious diseases they are sources of discrimination and stigma around the world [1]. Whenever a person becomes infectious of HIV virus then his or her immune system becomes weaker and weaker. It leads to the poor and at the end it becomes hard for the immune system in order to fight over diseases and infections [1, 2].

According to WHO notification along GHO (Global Health Observatory) data in 2018 it is observed that since the beginning of the epidemic the total number of population infected by Fata disease HIV is estimated to about 75 million people gets infected with the HIV virus. According to the given data in 2018 by GHO it is estimated about 32 million human population have been died of HIV. Worldwide census survey of HIV infected people indicates that about 37.9 million [32.7-44.0 million] human populations were living with HIV at the end of 2018. The age categorical statistics of HIV infected human population estimation described as 0.8% [0.6-0.9%] of adult aged human population in the range of 15-49 years are living with HIV infection worldwide. Although there is a difference in burden to carry out situation associated with the HIV epidemic considerably between regions and countries, African region remains as most fatal infected, with approximately 1 in every 25 adults which constitutes about (3.9%) living with HIV and this accounts for more than two thirds of the total human population living with HIV worldwide.

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].

In our previous work given in [5] we have described the dynamics of human population by dividing total human population into five compartments. In this study we extend the work done in [5] into six compartmental model. The procedures of the work are outlined as follows: 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

In this study the deterministic dynamical system of ordinary differential equations has been formulated to show the dynamics of human population in the presence of HIV (Human Immunodeficiency Virus) and ART as treatment. Here, human population under consideration is divided into six compartments. The descriptions of these compartments are as follows:

(i) Susceptible compartment. It is denoted by $S(t)$. These are humans who are free of HIV infection but are capable of becoming infected future in infectious environment (ii) Primary compartment. It is denoted by $P(t)$.This compartment includes all humans who infected with HIV for the first time and that do not know their HIV status but transmit the disease to others with effective contact (iii) Asymptomatic compartment. It is denoted by $A(t)$. This compartment includes all humans who know that they are infected with virus but no signs of infections is visible and abstain from transmitting virus to others. They also do not want to take any treatment because of cultural trends in the society (iv) Symptomatic compartment. It is denoted by $J(t)$. This compartment includes 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 join treatment compartment at some rate (v) Treatment compartment. It is denoted by $T(t)$. This compartment includes portion of symptomatic compartment that join it because of infection (vi) AIDS compartment. It is denoted by $V(t)$. This compartment includes who are at last stage or advanced stage of HIV.

Now, a mathematical model of Human Immunodeficiency virus (HIV) is formulated based on the stated assumptions on the human population as listed below:

(i) The total size of human's population under consideration is assumed to be constant.
(ii) The numbers of births and deaths of human population are assumed to be equal.
(iii) Deterministic dynamical system in the presence of Human Immunodeficiency virus (HIV) classifies human population under observation into six compartments as SPAJTV at any time.
(iv) Susceptible humans are recruited to the compartment $S(t)$ at some constant rate τ.
(v) Susceptible humans can be infected if they make effective contact with primary infected population whose status of HIV is not known yet and join primary infected compartment at a constant rate β.
(vi) Primary infected humans transfer into asymptomatic compartment at a constant rate κ.
(vii) Asymptomatic humans transfer into symptomatic humans at a rate θ.
(viii) The symptomatic humans transfer into treatment compartment at the rate ω.
(ix) All categories of human compartments face the same natural mortality with a rate μ.
(x) Some treated group leave treatment compartment and transfer to asymptomatic compartment at a constant rate of ϕ.
(xi) Some humans under treatment resist drugs and transfer to AIDS compartment at a constant rate γ.
(xii) All AIDS humans suffer disease induced death at a constant rate δ.
(xiii) All parameters used in the dynamical system 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
$\mathbf{T(t)}$	Population size of humans under treatment
$\mathbf{V(t)}$	Population size of AIDS humans

Table 2 Model parameters notations and description

Parameter	Description
τ	Recruitment rate of susceptible human population. With this constant rate new humans will born and enter into susceptible compartment
$\boldsymbol{\beta}$	Transmission rate of primary infected humans. With this rate primary infected humans transfer into P
$\boldsymbol{\kappa}$	Rate of humans transferring from compartment P to A
$\boldsymbol{\theta}$	Rate of humans transferring from compartment A to J
$\boldsymbol{\omega}$	Rate of humans transferring from compartment J to T
$\boldsymbol{\phi}$	Rate of humans transferring from compartment T to A
$\boldsymbol{\mu}$	Natural death rate. With this rate humans in all compartments die naturally
$\boldsymbol{\gamma}$	Rate of humans transferring from compartment T to V.

δ	Disease induced death rate of AIDS humans

Now considering basic assumptions and description of both model variables and parameters given the schematic diagram of the formulated deterministic dynamical system is described in the Figure 1.

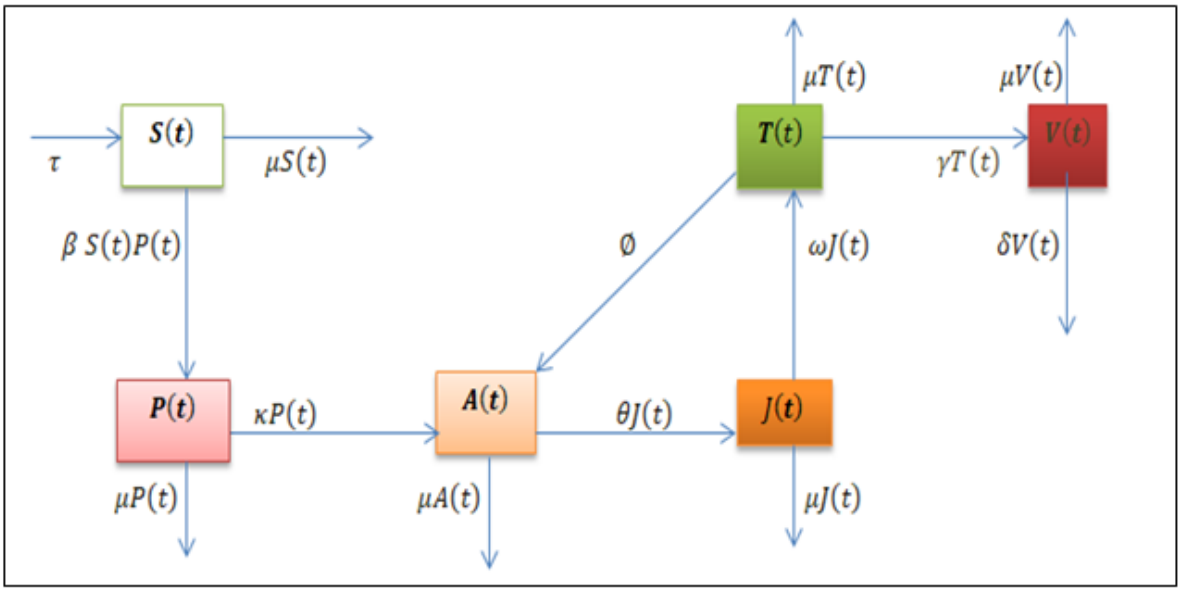


Figure 1 Schematic diagram of compartmental structure of the model

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:

$$dS/dt = \tau - \beta S(t)P(t) - \mu S(t) \quad (1)$$
$$dP/dt = \beta S(t)P(t) - (\kappa + \mu)P(t) \quad (2)$$
$$dA/dt = \kappa P(t) + \phi T(t) - (\theta + \mu)A(t) \quad (3)$$
$$dJ/dt = \theta A(t) - (\omega + \mu)J(t) \quad (4)$$
$$dT/dt = \omega J(t) - (\phi + \gamma + \mu)T(t) \quad (5)$$
$$dV/dt = \gamma T(t) - (\delta + \mu)V \quad (6)$$

The non-negative initial conditions of the model equations (1) – (6) are denoted by $S(0) \geq 0,\ P(0) \geq 0,\ A(0) \geq 0,\ J(0) \geq 0,\ T(0) \geq 0,\ V(0) \geq 0$. This system consists of six first order non-linear ordinary differential equations.

III. Mathematical analysis of the model

In this section we describe the mathematical analysis of the present improved and modified model. The analysis consists of the following points (i) existence, positivity and boundedness of solutions (ii) Equilibrium points (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.

1.1 Existence, Positivity and Boundedness of solution

In order to say that the formulated dynamical system is biologically valid and mathematically well-posed, it is required to show that the solutions of the system of differential equations (1) – (6) exist, non-negative and bounded for all time t . It is done starting with proving Lemma 1.

Lemma 1 (Existence) Solutions of the model equations (1) – (6) together with the initial conditions$S(0) \geq 0,\ P(0) \geq 0,\ A(0) \geq 0,\ J(0) \geq 0,\ T(t),\ V(0) \geq 0$ exist in$\mathbb{R}^6_+$i.e. the model variables $S(t),\ P(t),\ A(t),\ J(t),\ T(t)$ and $V(t)$ exist for all t and will remain in$\mathbb{R}^6_+$.

Proof: Let the right hand sides of the system of equations (1) – (6) are expressed as follows:

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

$$dT/dt = \omega J(t) - (\phi + \gamma + \mu)T(t) \equiv g_5(S,\ P,\ A,\ J,\ T,\ V)$$
$$dV/dt = \gamma T(t) - (\delta + \mu)V \equiv g_6(S,\ P,\ A,\ J,\ T,\ V)$$

According to Derrick and Groosman theorem, let R denote the region$R = \{(S,\ P,\ A,\ J,\ V) \in \mathbb{R}_+^6;\ N \leq \tau\delta+\mu$. Then equations (1) – (5) have a unique solution if $\partial gi\partial xj,\ \forall\ i,\ j=1,\ 2,\ 3,\ 4,\ 5,\ 6$ are continuous and bounded in$R$. Here, the notations$x_1 = S,\ x_2 = P,\ x_3 = A,\ x_4 = J,\ x_5 = T,\ x_6 = V,$ are employed. The Existence, continuity and the boundedness of$g_1,\ g_2,\ g_3,\ g_4,\ g_5$ and $g_6$are verified as here under:

Table 3 Verification of Continuity and Boundedness of the Function

Function	Existence and Continuity	Boundedness
g_1	$(\partial g_1)/(\partial S) = -[\beta P(t) + \mu]$ $(\partial g_1)/(\partial P) = -\beta S(t)$ $(\partial g_1)/(\partial A) = 0$ $(\partial g_1)/(\partial J) = 0$ $(\partial g_1)/(\partial T) = 0$ $(\partial g_1)/(\partial V) = 0$	$\lvert(\partial g_1)/(\partial S)\rvert = \lvert-[\beta P(t) + \mu]\rvert < \infty$ $\lvert(\partial g_1)/(\partial P)\rvert = \lvert-\beta S(t)\rvert < \infty$ $\lvert(\partial g_1)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_1)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_1)/(\partial T)\rvert = 0 < \infty$ $\lvert(\partial g_1)/(\partial V)\rvert = 0 < \infty$
g_2	$(\partial g_2)/(\partial S) = \beta P(t)$ $(\partial g_2)/(\partial P) = \beta S(t) - (\kappa + \mu)$ $(\partial g_2)/(\partial A) = 0$ $(\partial g_2)/(\partial J) = 0$ $(\partial g_2)/(\partial T) = 0$ $(\partial g_2)/(\partial V) = 0$	$\lvert(\partial g_2)/(\partial S)\rvert = \lvert\beta P(t)\rvert < \infty$ $\lvert(\partial g_2)/(\partial P)\rvert = \lvert\beta S(t) - (\kappa + \mu)\rvert < \infty$ $\lvert(\partial g_2)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_2)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_2)/(\partial T)\rvert = 0 < \infty$ $\lvert(\partial g_2)/(\partial V)\rvert = 0 < \infty$
g_3	$(\partial g_3)/(\partial S) = 0$ $(\partial g_3)/(\partial P) = \kappa$ $(\partial g_3)/(\partial A) = -(\theta + \mu)$ $(\partial g_3)/(\partial J) = 0$ $(\partial g_3)/(\partial T) = \emptyset$ $(\partial g_3)/(\partial V) = 0$	$\lvert(\partial g_3)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_3)/(\partial P)\rvert = \kappa < \infty$ $\lvert(\partial g_3)/(\partial A)\rvert = \theta + \mu < \infty$ $\lvert(\partial g_3)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_3)/(\partial T)\rvert = \emptyset < \infty$ $\lvert(\partial g_3)/(\partial V)\rvert = 0 < \infty$
g_4	$(\partial g_4)/(\partial S) = 0$ $(\partial g_4)/(\partial P) = 0$ $(\partial g_4)/(\partial A) = \theta$ $(\partial g_4)/(\partial J) = -(\omega + \mu)$ $(\partial g_4)/(\partial T) = 0$ $(\partial g_4)/(\partial V) = 0$	$\lvert(\partial g_4)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_4)/(\partial P)\rvert = 0 < \infty$ $\lvert(\partial g_4)/(\partial A)\rvert = \theta < \infty$ $\lvert(\partial g_4)/(\partial J)\rvert = \omega + \mu < \infty$ $\lvert(\partial g_4)/(\partial T)\rvert = 0 < \infty$ $\lvert(\partial g_4)/(\partial V)\rvert = 0 < \infty$
g_5	$(\partial g_5)/(\partial S) = 0$ $(\partial g_5)/(\partial P) = 0$ $(\partial g_5)/(\partial A) = 0$ $(\partial g_5)/(\partial J) = \omega$ $(\partial g_5)/(\partial T) = -(\phi + \gamma + \mu)$ $(\partial g_5)/(\partial V) = 0$	$\lvert(\partial g_5)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial P)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial J)\rvert = \omega < \infty$ $\lvert(\partial g_5)/(\partial T)\rvert = \phi + \gamma + \mu < \infty$ $\lvert(\partial g_5)/(\partial V)\rvert = 0 < \infty$
g_6	$(\partial g_5)/(\partial S) = 0$ $(\partial g_5)/(\partial P) = 0$ $(\partial g_5)/(\partial A) = 0$ $(\partial g_5)/(\partial J) = 0$ $(\partial g_5)/(\partial T) = \gamma$ $(\partial g_5)/(\partial V) = -(\delta + \mu)$	$\lvert(\partial g_5)/(\partial S)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial P)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial A)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial J)\rvert = 0 < \infty$ $\lvert(\partial g_5)/(\partial T)\rvert = \gamma < \infty$ $\lvert(\partial g_5)/(\partial V)\rvert = \delta + \mu < \infty$

Thus, all the partial derivatives $(\partial g_i)/(\partial x_j)$: $i,\ j = 1,\ 2,\ 3,\ 4$ exist, and are both continuous and bounded in$R$. Hence, by Derrick and Groosman theorem, a solution for the model (1) – (6) exists and is unique.

Lemma 2 (Positivity) Solutions of the model equations (1) – (6) 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$\mathbb{R}_+^6$.

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

Positivity of $S(t)$: The model equation (1) given by $dS/dt = \tau - \beta S(t)P(t) - \mu S(t)$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 $dP/dt = \beta S(t)P(t) - (\kappa + \mu)P(t)$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$dP/dt \geq -\big((\kappa + \mu)\big)P(t)$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $P(t) \geq P(0)e^{-((\kappa+\mu))t}$.

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

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

Positivity of $V(t)$*:* The model equation (6) given by $dV/dt = \gamma T(t) - (\delta + \mu)V$ can be expressed without loss of generality, after eliminating the positive term$\gamma T(t)$ which is appearing on the right hand side, as an inequality as $dV/dt \geq -(\delta + \mu)V$. Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $V(t) \geq V(0)e^{-(\delta+\mu)t}$. Recall that an exponential function is always non–negative irrespective of the sign of the exponent i.e. the exponential function$e^{-(\delta+\mu)t}$is a non-negative quantity. Hence, it can be concluded that $V(t) \geq 0$.

Thus, the model variables $S(t)$, $P(t)$, $A(t)$, $J(t)$, $T(t)$and $V(t)$ representing population sizes of various types of human population are positive quantities and will remain in $\mathbb{R}_+^6$for all$t$.

Lemma 2 (Boundedness) The non-negative solutions of the system of model equations (1) – (6) are bounded. That is the model variables$S(t)$, $P(t)$, $A(t)$, $J(t)$, $T(t)$ and $V(t)$ are all bounded for all t [4, 7, 9, 10].

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 $N(t) = S(t) + P(t) + A(t) + J(t) + T(t)\ V(t)$ is bounded for all t. It can be begun by showing that all feasible solutions are uniformly bounded in a proper subset$R \in \mathbb{R}_+^6$where the feasible region R is given by$R = \{(S,\ P,\ A,\ J,\ T,\ V) \in \mathbb{R}_+^6\ ;\ \mathrm{N} \leq \tau\delta+\mu.$

Now, summation of all the five equations (1) – (6) of the model gives $\mathrm{dN(t)/dt} = \tau - \mu\mathrm{N(t)} - \delta\mathrm{V}(t)$. Again considering total population N(t) and subpopulation V(t) further we can write the equation as inequality of the form$\mathrm{dN(t)/dt} \leq [\tau - (\mu + \delta)\mathrm{N(t)}]$. Equivalently this inequality can be expressed as a linear ordinary differential inequality as $[\mathrm{dN(t)/dt}] + [(\mu + \delta)\mathrm{N(t)}] \leq \tau$ giving general solution upon solving as$\mathrm{N(t)} \leq [\tau/(\mu + \delta)] + ce^{-(\mu+\delta)t}$. 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$\mathrm{N(t)} \leq [\tau/(\mu + \delta)] + [\mathrm{N(0)} - (\tau/(\mu + \delta))]e^{-(\mu+\delta)t}$. Further, it can be observed that $N(t) \to [\tau/(\mu + \delta)]$as $t \to \infty$. That is, total population size $N(t)$takes off from a value $\mathrm{N(0)}$ at the initial time $\mathrm{t} = 0$ and ends up with a bounded value$[\tau/(\mu + \delta)]$as the time t progresses to infinity. Thus, it can be concluded that $N(t)$ is bounded within a pair of values as$0 \leq N(t) \leq [\tau/(\mu + \delta)]$.

Therefore, $[\tau/(\mu + \delta)]$is an upper bound of $N(t)$. Hence, feasible solution of the system of model equations (1) – (6) remains in the region R which is a positively invariant set. Thus, the system is biologically meaningful in the domain$R$. Further, it is sufficient to consider the dynamics of the populations represented by the model system (1) – (6) 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".

Therefore, the formulated model is biologically meaningful and mathematically well-posed.

3.2 Equilibrium points

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) – (6) 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 $P(t) = A(t) = J(t) = T(t) = V(t) = 0$ and also setting the right hand sides of the model equations (1) – (6) 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) – (6) is given by

$$E_0 = (S^0, \quad 0, \quad 0, \quad 0, \quad 0, \quad 0) = ((\tau/\mu), \quad 0, \quad 0, \quad 0, \quad 0, \quad 0)$$

3.2.2 Endemic equilibrium point

The endemic equilibrium point $E_1 = \{S^1, \quad P^1, \quad A^1, \quad J^1, \quad T^1, \quad V^1\}$ 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) – (6) to zero. That is, setting $dS/dt = dA/dt = dJ/dt = dT/dt = dV/dt = 0$ the model equations take the form as

$$\tau - \beta S(t)P(t) - \mu S(t) = 0 \quad (7)$$

$$\beta S(t)P(t) - (\kappa + \mu)P(t) = 0 \quad (8)$$

$$\kappa P(t) + \phi T(t) - (\theta + \mu)A(t) = 0 \quad (9)$$

$$\theta A(t) - (\omega + \mu)J(t) = 0 \quad (10)$$

$$\omega J(t) - (\phi + \gamma + \mu)T(t) = 0 \quad (11)$$

$$\gamma T(t) - (\delta + \mu)V(t) = 0 \quad (12)$$

Here in (7) – (12), the quantities $a, \ b, \ c$ represent the parametric expressions as $a = \kappa + \mu, \ b = \theta + \mu, \ c = \omega + \mu, \ d = \phi + \gamma + \mu, \ e = \delta + \mu$. Clearly, solutions of (7) – (12) 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$\beta S - a = 0$ or equivalently $S = (a/\beta)$. That is, the S^1 component of$E_1$is given by

$$S^1 \equiv S = (a/\beta) = (\tau/\mu R_0) \quad (13)$$

(ii) Now the solution for P can be obtained by substituting equation (13) into equation (7) and rewriting the resulting equation as $\tau - \beta(\tau/\mu R_0)P - \mu(\tau/\mu R_0) = 0$ giving

$$P^1 \equiv P = (\mu/\beta)(R_0 - 1) \quad (14)$$

(iii) Again the solution for T can be obtained by substituting P value from (14), A from (10), and J from (11) into equation (9). Now, the reduced expression has the form

$$T^1 \equiv T = \{[(\kappa\mu)(R_0 - 1)]/[\beta((bcd/\theta\omega) - \emptyset)]\} \quad (15)$$

(iv) By substituting T value from (15) into equation (11) the solution for J is given as

$$J^1 \equiv J = \{[(d\kappa\mu)(R_0 - 1)]/[\beta\omega((bcd/\theta\omega) - \emptyset)]\} \quad (16)$$

(v) By substituting J value from (16) into equation (10) the solution for A is given as

$$A^1 \equiv A = \{[(cd\kappa\mu)(R_0 - 1)]/[\theta\beta\omega((bcd/\theta\omega) - \emptyset)]\} \quad (17)$$

(vi) Finally, using the value of T from (15) and solving for V from equation (12) we have

$$V' \equiv V = \{[(\gamma\kappa\mu)(R_0 - 1)]/[e\beta((bcd/\theta\omega) - \emptyset)]\} \quad (18)$$

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 [4, 5]. 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, 4, 5]. From the system (1) – (6) the five equations of infected individuals are considered and decomposed into two groups: F contains newly infected cases and v contains the remaining terms. Let $X = [S,\ P,\ A,\ J,\ T,\ V]^t$ be a column vector and the differential equations of the first four compartments are rewritten as $F(X) - T(X)$.

Now, let $F(X) = [F_1,\ F_2,\ F_3,\ F_4,\ F_5]^t$. 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, (iii) $F_4 = 0$ denotes newly infected case from susceptible compartment into Treatment compartment, and (iv) $F_5 = 0$ denotes newly infected case from susceptible compartment into AIDS compartment. Further, let $T(X) = [T_1,\ T_2,\ T_3,\ T_4,\ T_5]^t$. Here $T_1 = aP,\ T_2 = -\kappa P - \emptyset T + bA,\ T_3 = -\theta A + cJ, T_4 = -\omega J + dT,$ and $-\gamma T + eV$. Here, the values of $a,\ b,\ c,\ d,$ and e are as defined above.

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

$$F \equiv J_F(E_0) = \begin{bmatrix} \beta\tau/\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } T \equiv J_T(E_0) = \begin{bmatrix} a & 0 & 0 & 0 & 0 \\ -\kappa & b & 0 & -\emptyset & 0 \\ 0 & -\theta & c & 0 & 0 \\ 0 & 0 & -\omega & d & 0 \\ 0 & 0 & 0 & -\gamma & e \end{bmatrix} \quad (19)$$

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

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

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

$$\lambda_1 = (\beta\tau/\mu\, a),\quad \lambda_2 = 0,\quad \lambda_3 = 0,\quad \lambda_4 = 0\,,\quad \lambda_5 = 0$$

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 point

In absence of the infectious disease, the model populations have a unique disease free equilibrium pointE_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 $E_0$the mathematical procedures described in [10] is used. It is already shown that the DFE of model (1) – (6) is given by$E_0 = \{\tau/\mu,\ 0,\ 0,\ 0,\ 0,\ 0\}$. Now, following [10] 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) – (6) 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) – (6) as functions so as to find the Jacobian matrix as follows:

$$dS/dt = \tau - \beta S(t)P(t) - \mu S(t) \equiv g_1(S,\ P,\ A,\ J,\ T,\ V)$$
$$dP/dt = \beta S(t)P(t) - (\kappa + \mu)P(t) \equiv g_2(S,\ P,\ A,\ J,\ T,\ V)$$
$$dA/dt = \kappa P(t) + \phi T(t) - (\theta + \mu)A(t) \equiv g_3(S,\ P,\ A,\ J,\ T,\ V)$$
$$dJ/dt = \theta A(t) - (\omega + \mu)J(t) \equiv g_4(S,\ P,\ A,\ J,\ T,\ V)$$
$$dT/dt = \omega J(t) - (\phi + \gamma + \mu)T(t) \equiv g_5(S,\ P,\ A,\ J,\ T,\ V)$$
$$dV/dt = \gamma T(t) - (\delta + \mu)V \equiv g_6(S,\ P,\ A,\ J,\ T,\ V)$$

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

$$J(S,\ P,\ A,\ J,\ T,\ V) = \begin{bmatrix} -\beta P - \mu & -\beta S & 0 & 0 & 0 & 0 \\ \beta P & \beta S - (\kappa + \mu) & 0 & 0 & 0 & 0 \\ 0 & \kappa & -(\theta + \mu) & 0 & \emptyset & 0 \\ 0 & 0 & \theta & -(\omega + \mu) & 0 & 0 \\ 0 & 0 & 0 & \omega & -(\emptyset + \gamma + \mu) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -(\delta + \mu) \end{bmatrix} \quad (20)$$

Now, the Jacobian matrix of $g_1,\ g_2,\ g_3,\ g_4,\ g_5,\ g_6$ with respect to $S,\ P,\ A,\ J,\ T,\ V$at the disease free equilibrium E_0 is given by

$$J(E_0) = \begin{bmatrix} -\mu & -(\kappa + \mu)R_0 & 0 & 0 & 0 & 0 \\ 0 & (\kappa + \mu)(R_0 - 1) & 0 & 0 & 0 & 0 \\ 0 & \kappa & -(\theta + \mu) & 0 & \emptyset & 0 \\ 0 & 0 & \theta & -(\omega + \mu) & 0 & 0 \\ 0 & 0 & 0 & \omega & -(\emptyset + \gamma + \mu) & 0 \\ 0 & 0 & 0 & 0 & \gamma & -(\delta + \mu) \end{bmatrix}$$

Now, to determine the signs of eigenvalues we use the concept of trace and determinant of a given matrix as mentioned in the [5].

(1) Trace of $J(E_0) = (\kappa + \mu)(R_0 - 1) - \mu - \theta - \mu - \omega - \mu - \emptyset - \gamma - \mu - \delta - \mu < 0$, if $R_0 < 1$

(2) Determinant of $J(E_0) = a\mu e(\emptyset\theta\omega - bcd)(R_0 - 1) > 0$, Provided that either of the following two pairs of conditions are satisfied: (i) $\emptyset\theta\omega < bcd$ and $R_0 < 1$ or (ii) $\emptyset\theta\omega > bcd$ and $R_0 > 1$

Now, from trace and determinant obtained in (1) and (2) with the given conditions we conclude that all eigenvalues of a matrix $J(E_0)$ are negative provided the mentioned conditions are satisfied. Thus, from Hurwitz – Routh principle disease free equilibrium point is locally asymptotically stable if $R_0 < 1$ and unstable if$R_0 > 1$.

3.4.2 Global Stability of Disease Free Equilibrium Point

Here, we follow the procedure given in [11]. That is, let $x \in R^n$is disease compartment and $y \in R^m$be disease free compartment the disease transmission model (1)-(6) can be written in the form:

$$\dot{x} = -(T - F)x - h(x, y) \quad (21)$$
$$\dot{y} = g(x, y) \quad (22)$$

Here in (21), the notations $F = J_F(E_0)$ and $T = J_T(E_0)$ given is (19) 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) – (6) 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 nonsingular M-matrix. From the previous computations (19) we have

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

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

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

Now, $\det(T - F) = [\mathrm{e}(\emptyset\theta\omega - \mathrm{bcd})(\beta\tau - a\mu)]/\mu$ and $\rho(B) = max\{\beta(\tau/\mu), \ \sqrt[3]{\emptyset\theta\mu}\}$ and $T - F$ is nonsingular matrix provided that the conditions $\emptyset\theta\omega \neq \mathrm{bcd}$ and $\beta\tau \neq a\mu$ 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 $N(t) = S(t) + P(t) + A(t) + J(t) + T(t) + V(t)$ satisfies $N' = \Pi - \delta N - \delta V \leq \Pi - \mu N$, so that $N(t) \leq S_0 - \left(S_0 - N(0)\right)e^{-\mu t}$, with $S_0 = \tau/\mu$. 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) – (6) 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 $E_1 = \{S^1, \ P^1, \ A^1, \ J^1, \ T^1, \ V^1\}$ is the point where the disease persists or exists. To analyze the local stability of E_1, Jacobian matrix of the model that evaluated at this equilibrium point is used. Further, remember that the endemic equilibrium point $E_1 = \{S^1, \ P^1, \ A^1, \ J^1, \ V^1\}$ of the given model (1) – (6) 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 of E_0. Thus, the procedure starts with the construction of Jacobian matrix at E_1. Now, the Jacobian matrix of the model given in (20) at endemic equilibrium point E_1 takes the form as

$$J(S,\ P,\ A,\ J,\ T,\ V) = \begin{bmatrix} -\beta P - \mu & -\beta S & 0 & 0 & 0 & 0 \\ \beta P & \beta S - a & 0 & 0 & 0 & 0 \\ 0 & \kappa & -b & 0 & \emptyset & 0 \\ 0 & 0 & \theta & -c & 0 & 0 \\ 0 & 0 & 0 & \omega & -d & 0 \\ 0 & 0 & 0 & 0 & \gamma & -e \end{bmatrix}$$

Hence, $$J(E_1) = \begin{bmatrix} -\mu R_0 & -\beta(\tau/\mu R_0) & 0 & 0 & 0 & 0 \\ \mu(R_0 - 1) & 0 & 0 & 0 & 0 & 0 \\ 0 & \kappa & -b & 0 & \emptyset & 0 \\ 0 & 0 & \theta & -c & 0 & 0 \\ 0 & 0 & 0 & \omega & -d & 0 \\ 0 & 0 & 0 & 0 & \gamma & -e \end{bmatrix}$$

Now the trace of $J(E_1)$ is a negative quantity while determinant of $J(E_1)$ computed as $-\beta\tau\varepsilon\,(\emptyset\theta\omega - \mathrm{bcd})(R_0 - 1)/\mathrm{R}_0$ and is a positive quantity provided that,

(i) $\emptyset\theta\omega < bcd$ and $R_0 > 1$

(ii) $\emptyset\theta\omega > \mathrm{bcd}$ and $R_0 < 1$

Hence, the endemic equilibrium point E_1 is locally asymptotically unstable if $R_0 < 1$ and stable if $R_0 > 1$ provided that the afro mentioned conditions are satisfied.

2. Numerical Simulation

In this section, numerical simulations of model equations (1) – (6) have been carried out with the support of MATLAB software. 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.

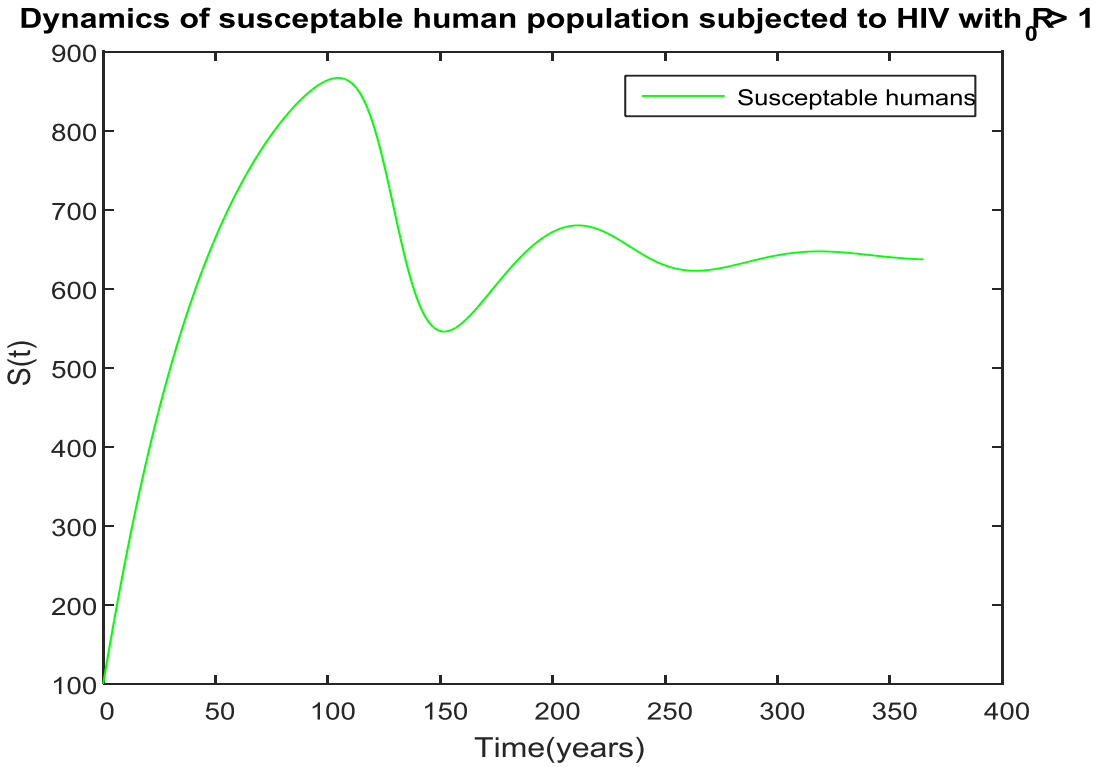


Figure 1 Dynamics of susceptible population with parametric values $\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

According to simulations given in Figure 1 above the followings can be observed. It can be observed that the susceptible populations are increased in the first hundred years as the numbers of primary infected patients are less or decreased for about hundred years.

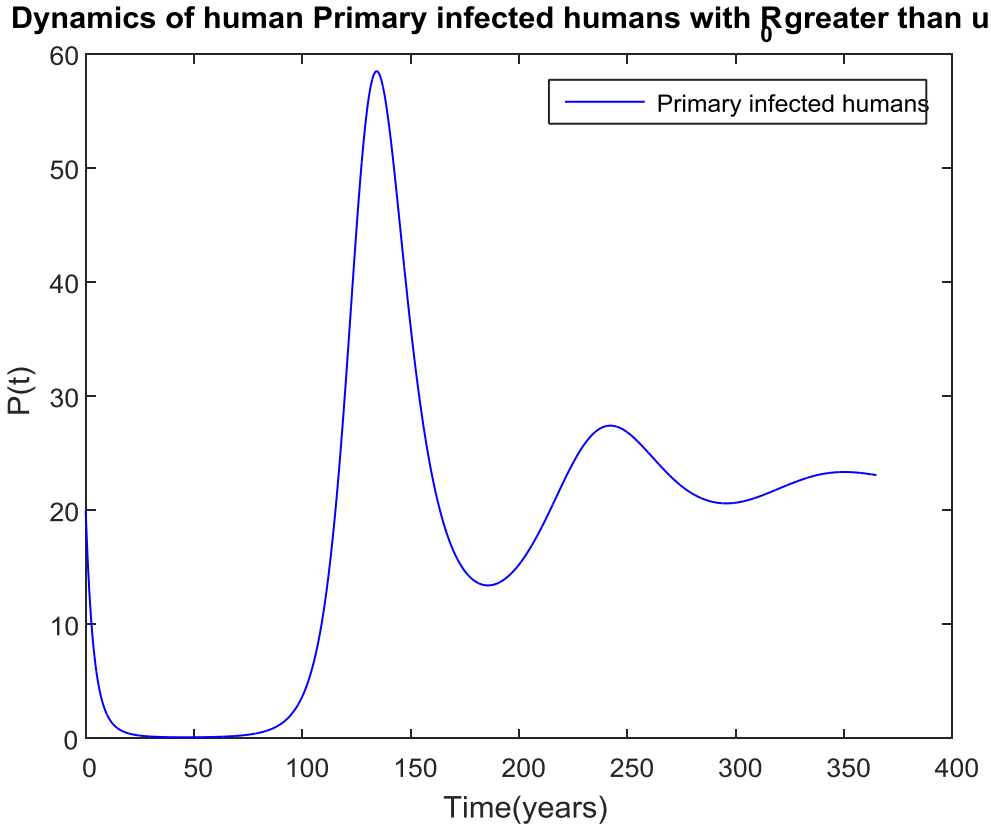


Figure 2 Dynamics of primary patients with parametric values $\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \ \kappa = 0.3,\ \ \omega = 0.08,\ \ \phi = 0.01,\ \ \theta = 0.06,\ \ \gamma = 0.01,\ \ \delta = 0.05$

The simulation in Figure 2 shows that starting from the beginning the number of primary infected patient's decreases as less number of susceptible population infected at the first hundred years.

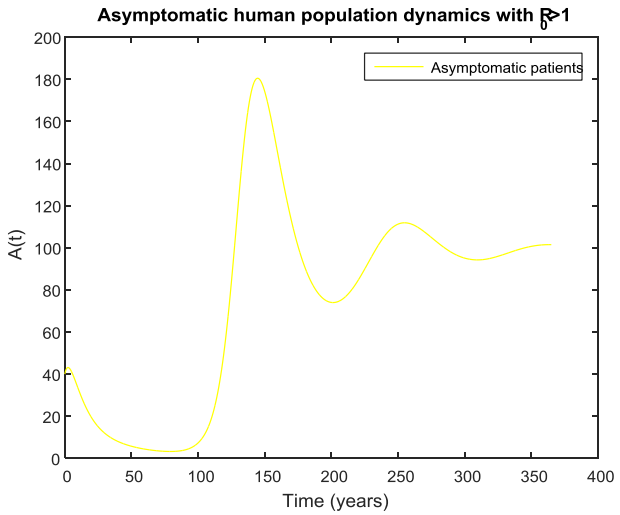
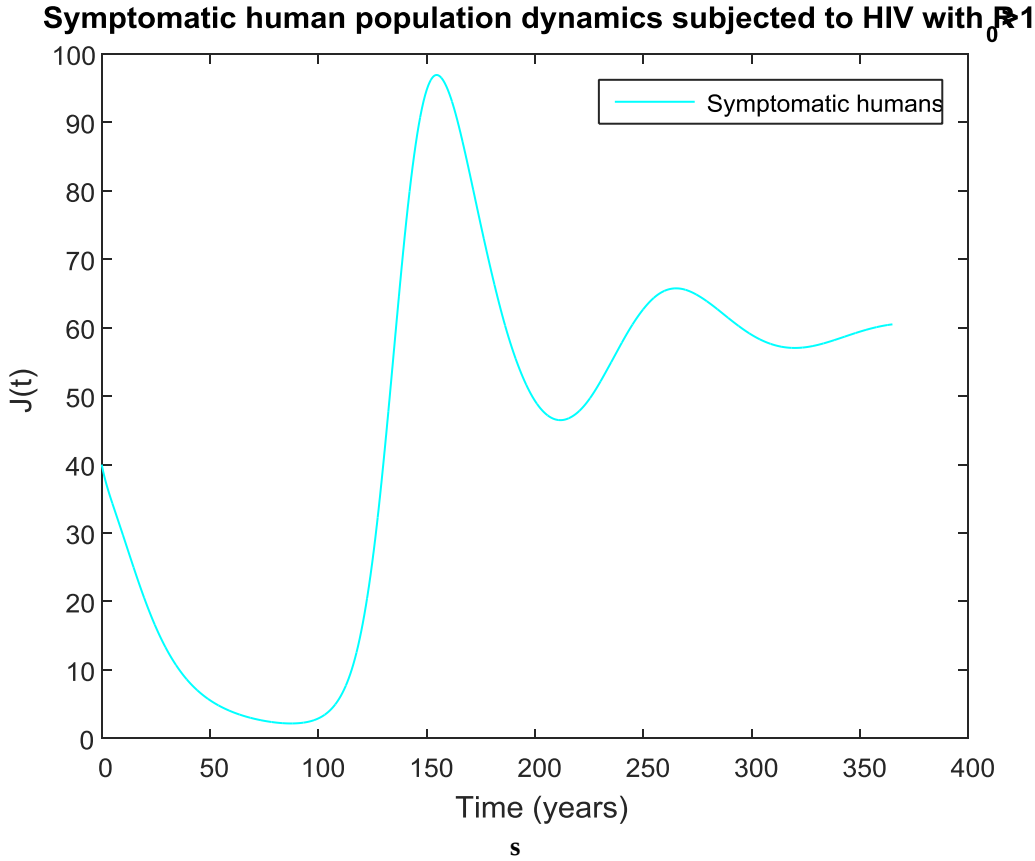


Figure 3 Dynamics of susceptible population with parametric values $\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \ \kappa = 0.3,\ \ \omega = 0.08,\ \ \phi = 0.01,\ \ \theta = 0.06,\ \ \gamma = 0.01,\ \ \delta = 0.05$

From Figure 3 we observe that the asymptomatic patients decrease for about hundred years as there less number of primary infected patients and increases as the number of primary infected and treated symptomatic patients who use herbal medicine increase. Finally, the populations continue with small changes.



s

Figure 4 Dynamics of AIDS patients with parametric values $\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

Figure 4 illustrates the simulation of symptomatic patients which decreases initially as the resultof less infected patients for about a century and increases for the next fifty years. Finally, it continues with small alternative changes in the population.

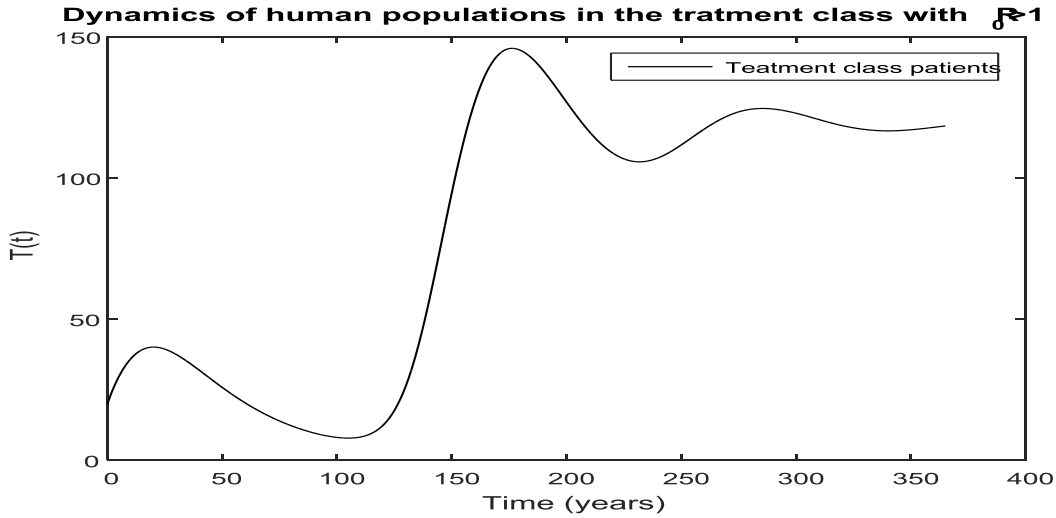


Figure 5 Dynamics of treatment class patients with parametric values $\boldsymbol{\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05}$

Figure 5 illustrates the simulation of treated patients that increase initially as syptomatic patients come in treatment class and decreases initially as the result of less infected patients withdraw treatment class because of their healthy condition safe or because of the resistance of drugs which leads to AIDS class.

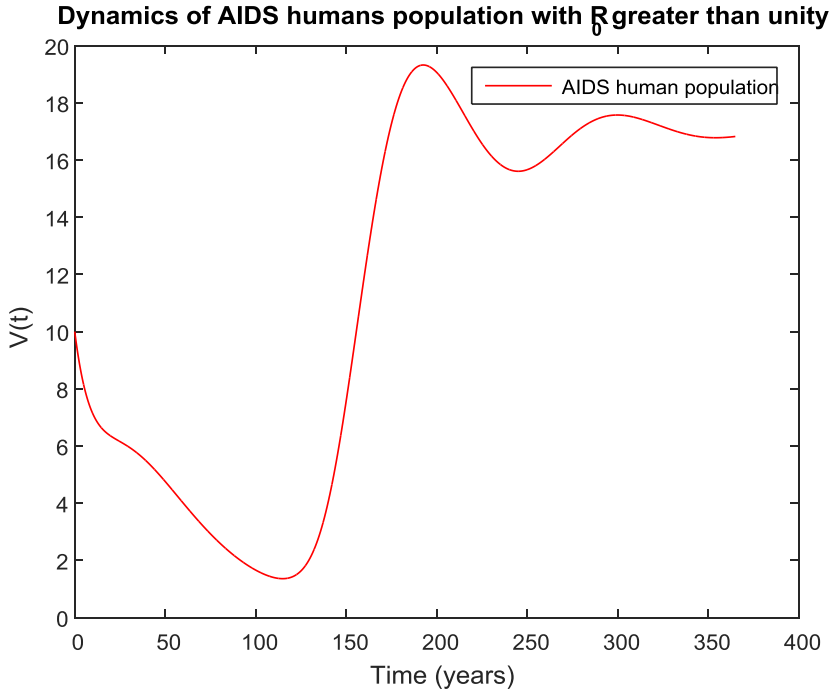


Figure 6 Dynamics of AIDS patients with parametric values $\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

Figure 6 illustrates the simulation of AIDS patients that decreases initially as less number of infected patients join it. Then increases because of patients under treatement class resist drugs and continue alternaatively with small changes.

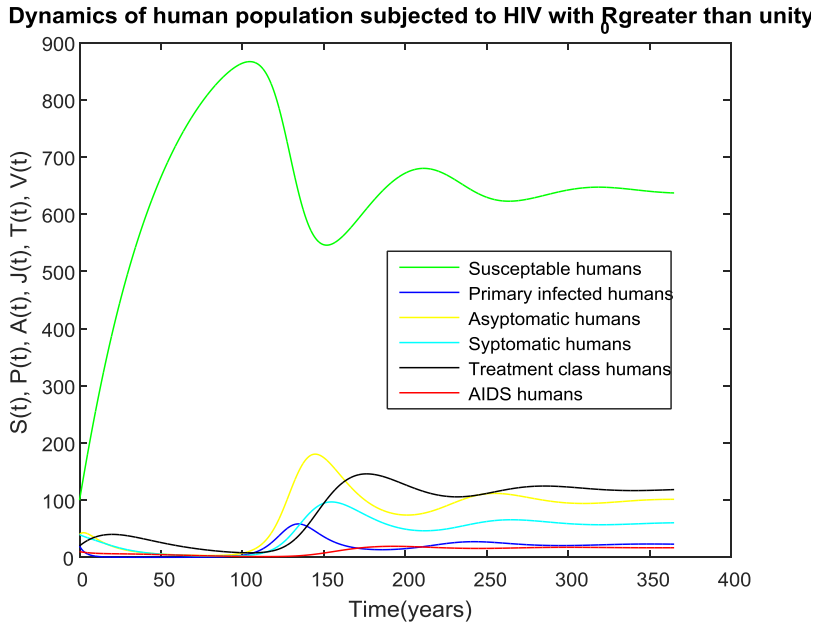


Figure 7 Dynamics of susceptible population with parametric values$\tau = 20,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

In Figure 7 the following can be observed: (i) initially the population size of susceptible compartment S isincrease because of 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 decrease because of less number of infected susceptible populations. Then increase followed by alternative small changes that lead to constant in number as there is a balance in population dynamics (iii) the asymptomatic patient's compartment decrease as initially as there are less number of patients that show no symptoms (iv) symptomatic compartment decreases initially as patients transfer to treatment class and. Then with small changes alternatively increases and decrease followed with constant as the result of balances in the transfer of patients(v) Treatment compartment increase initially as patients transfer to treatment class and. Then changes with small number that make alternatively increases and decrease followed with constant as the result of balances in the transfer of patients(vi) The AIDS compartment decrease initially because of fewer patients from treatment class joins it. Then increase followed with alternative changes with small amount that lead to constant number of patients as the result of balance of transfer in patients.

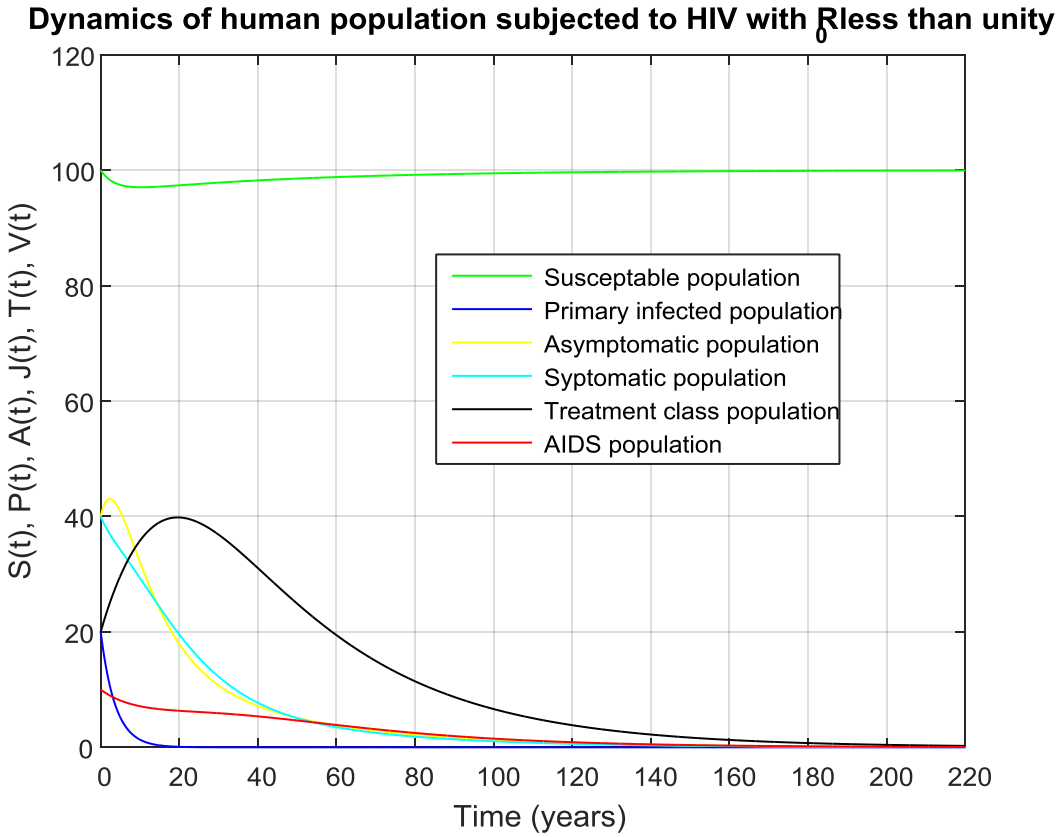


Figure 8 Population dynamics of $SPAJTV$ compartments with the parametric values $\tau = 2,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

In figure 8, 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 decreases as the number of patients enters symptomatic compartment (iv) the symptomatic compartment increases as asymptomatic patients shows symptoms of the disease and gets decrease as patients join treatment class from it (v) The treatment compartment individuals gets decrease as patients leaves the compartment willingly or because of severity of the disease that lead to AIDS stage (vi) AIDS compartment decrease as patients from treatment compartment decrease and disease induced death rate.

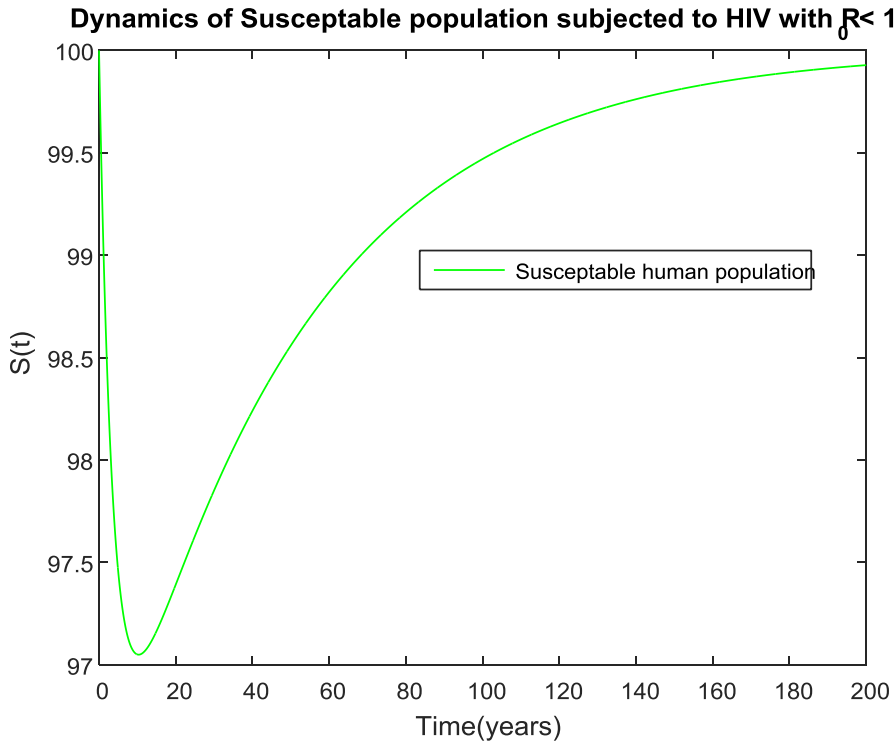


Figure 9 Population dynamics of susceptible compartment with the parametric values $\tau = 2,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

Figure 9 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.

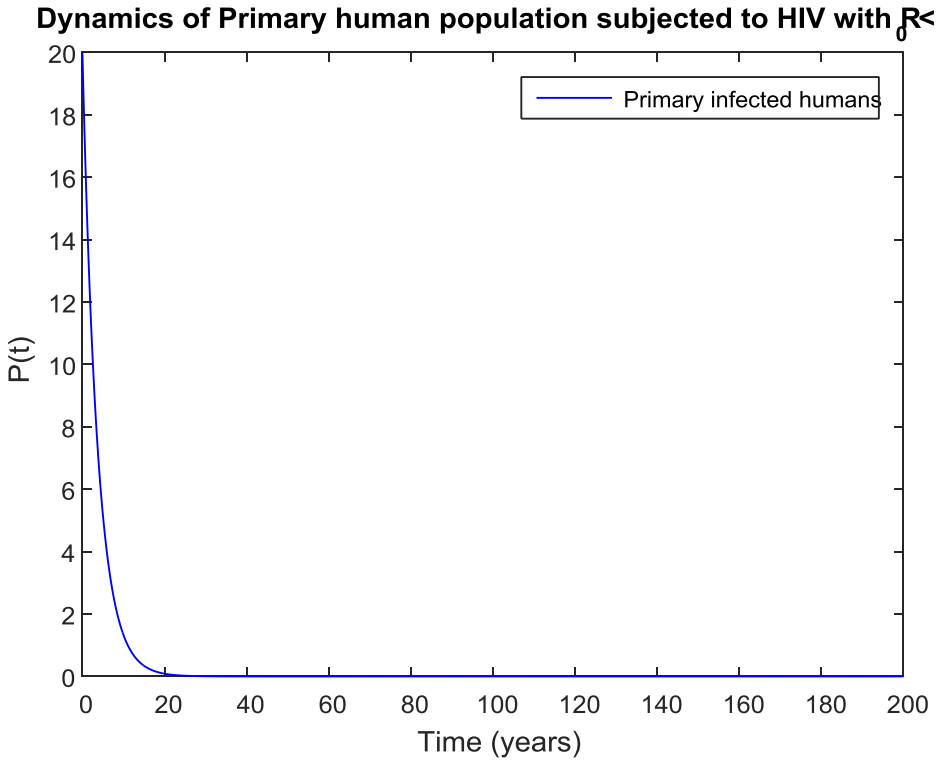


Figure 10 Dynamics of susceptible compartment with the parametric values $\tau = 2,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

From Figure 10 we observe that the number of primary patients decrease as less number of susceptible humans is infected.

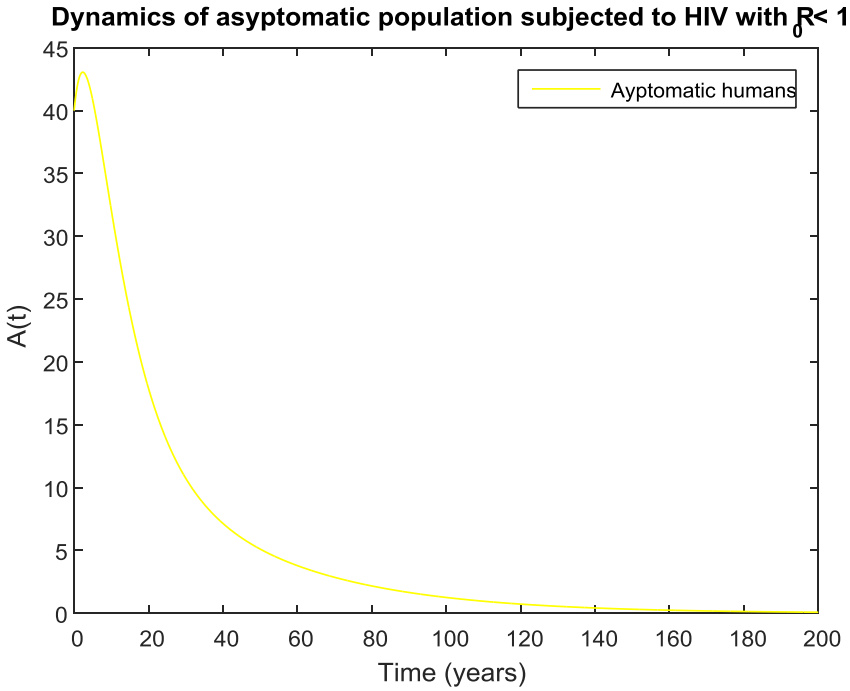


Figure 11 Population dynamics of asymptomatic compartment with the parametric values $\tau = 2,\ \mu = 0.02,$ $\beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

From figure 11above we observe that asymptomatic compartment gets increase initially as primary patients enters this compartment and others willingly come from treatment compartment.

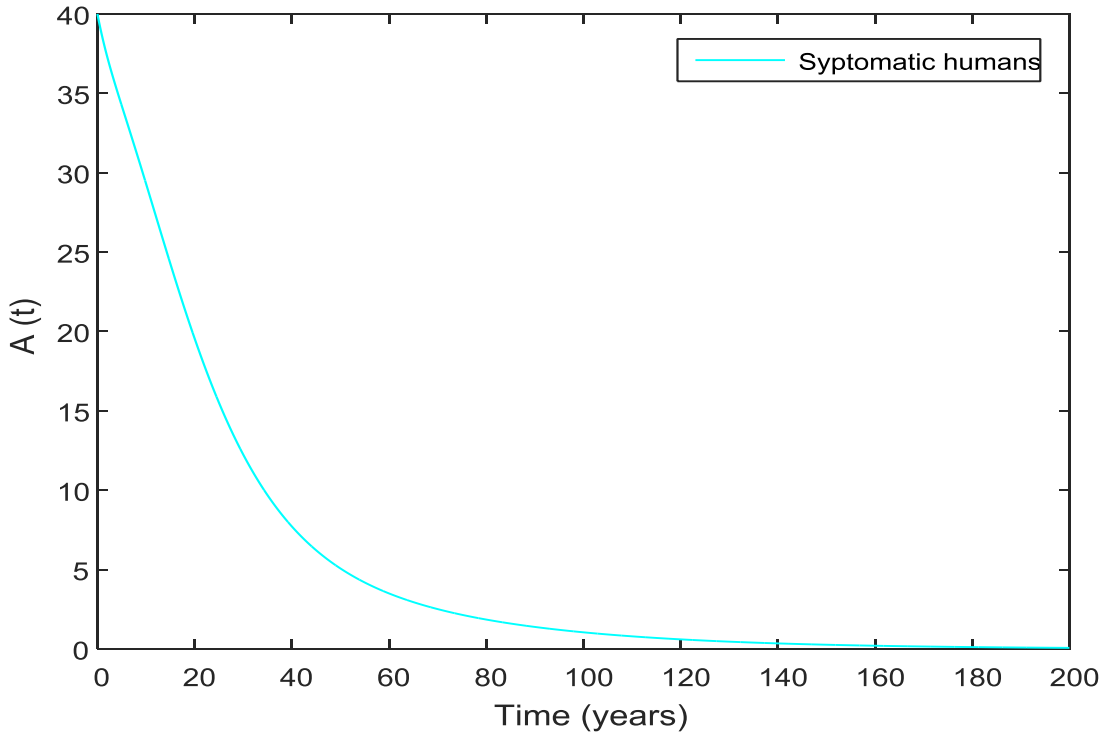


Figure 12 Population dynamics of symptomatic compartment with the parametric values $\tau = 2,\ \mu = 0.02,$ $\beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

Figure 12 Illustrate the fact that the number of symptomatic patients gets decrease as patients in asymptomatic patients decrease that lead to zero for a long interval of time.

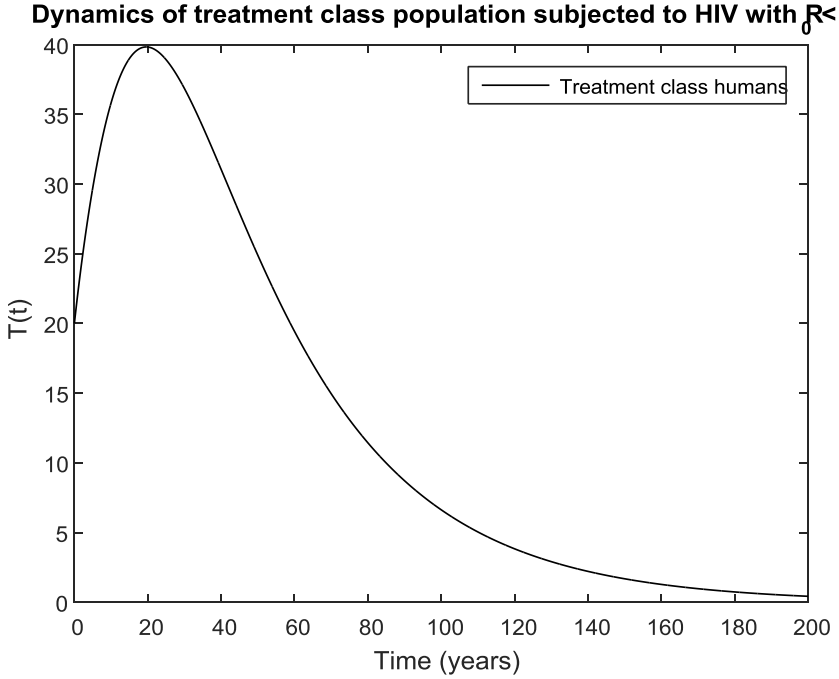


Figure 13 Population dynamics of treatment compartment with the parametric values $\tau = 2,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

From Figure 13 above it can be observed that initially the treatment class population increase as more symptomatic class patients joins the compartment. It gets decreasing as some patients leave the compartment willingly to join the asymptomatic compartment and some patients cells resist the medications and transferred to AIDS compartment class.

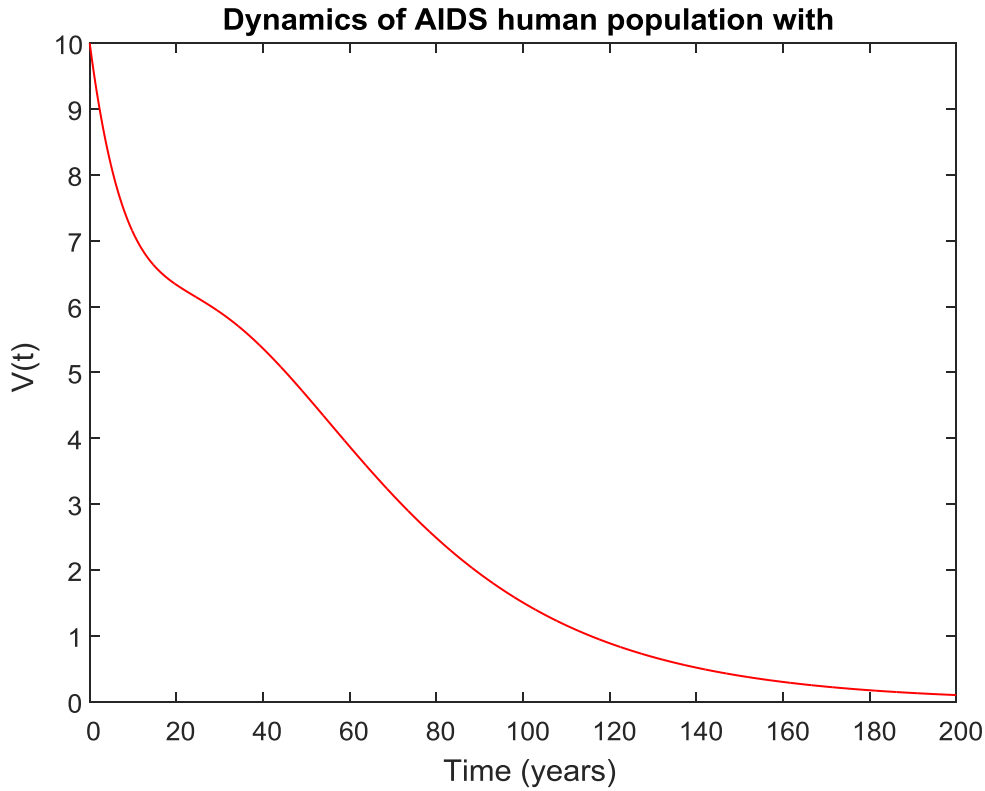


Figure 14 Population dynamics of AIDS compartment with the parametric values $\tau = 2,\ \mu = 0.02,\ \beta = 0.0005,\ \kappa = 0.3,\ \omega = 0.08,\ \phi = 0.01,\ \theta = 0.06,\ \gamma = 0.01,\ \delta = 0.05$

Figure 14 shows that AIDS patients get decreasing 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 that leads to zero patients in long time.

The differences and similarities between the existing and modified model are given respectively in the tables 4 and 5.

Table 4 Differences of existing and modified model

Differences		
SN	**Existing model [5]**	**Modified (Present) model**
1	Has five compartments	Has six compartments
2	Has susceptible class, primary class, asymptomatic class, symptomatic class, AIDS class	Has susceptible class, primary class, asymptomatic class, symptomatic class , treatment class, AIDS class
3	Herbal medicine considered for separate class	Herbal medicine and ART used in treatment class
4	Asymptomatic class used treatment	Asymptomatic class used no treatment

Table 5 Similarities of existing and modified model

Similarities
Both existing **[**5**]** and modified (Present) model have the following similarities (i) Disease induced death rate in AIDS patients (ii) Transmission rate (iii) Natural mortality (iv) Transfer of patients from asymptomatic to asymptomatic

IV. 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 [4]. 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.

Definition The normalized forward sensitivity index of a variable R_0 that depend differentially on a parameter p is defined by [12, 13, 14, 4]

$$\Upsilon_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}$$

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$R_0 = [(\beta\tau)/\mu(\kappa + \mu)]$. 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 follows:

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

Table 6 Sensitivity of R_0 evaluated for the parametric values given under Figure 1

Parameter	Sensitivity index
$\boldsymbol{\mu}$	-1.3125
$\boldsymbol{\beta}$	+1
$\boldsymbol{\kappa}$	-0.9375
$\boldsymbol{\tau}$	+1

From Table 6, it can be observed that the values of two parameters $\boldsymbol{\tau}$ and $\boldsymbol{\beta}$ are positive sensitivity indices and values of the remaining two parameters μ and κ get negative sensitivity indices.

As it is observed from the table the parameter with large magnitude are μ, τ and β. 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.

V. Result and Discussion

In this study, a deterministic dynamical system consists of six equations has been formulated to describe the dynamics of human population subjected to HIV Human Immunodeficiency Virus (HIV) epidemics. A formulated dynamical system is biologically meaningful and mathematically well-posed. The computation of the sensitivity indices shows that increasing recruitment rate and transmission rate increases the transmission of the disease. It is also observed from the simulation that increasing recruitment rate make the disease to persist in the population and this concept is supported with performed Figures (1) – (7).On the other hand decreasing this parameter values decrease the transmission of the disease and this concept is supported by the simulation done in Figures (8) – (14). Additionally, the negatively computed values of indices show that increasing these parametric values decrease the transmission of the disease in the population. Further from sensitivity indices computation we observed that the three most sensitive parameters in the formulated dynamical system for human population subjected to the HIV epidemic are τ, κ, and μ. 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 8-14. 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 (1-7).

VI. Conclusions

The formulated deterministic dynamical system of human population subjected to HIV epidemics is biologically meaningful and mathematically well-posed which is supported with the work done in showing existence, positivity and boundedness of the formulated dynamical system (see Table 3). In formulated deterministic dynamical system it is important to significantly assume or carefully take the values of recruitment rate, transmission rate, and natural death rate. The endemic equilibrium point is locally unstable for reproduction number less than unity. The stability analyses of the formulated model were investigated using the tools known as basic reproduction number and Routh Hurwitz criterion. The disease free equilibrium point is globally stable for reproduction number is less than unity and unstable for reproduction number is greater than unity. Also, the solution of the formulated model equations is numerically described with simulation and

sensitivity analysis of the formulated model is conducted. Furthermore, results of the performed research work in this paper reveal that the formulated model effectively supports treatment for HIV disease.

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