Analysis of HIV/AIDS Model with Nonlinear Incidence Function

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ABSTRACT

Human Immunodeficiency Virus – Acquired Immune Deficiency Syndrome HIV/AIDS stands as one of the most prevalent sexually transmitted disease globally and is regarded as one of the deadliest epidemic in human history. This study presents a mathematical model for understanding the dynamics of HIV/AIDS transmission, incorporating a saturated incidence rate. The model employs a system of ordinary differential equations, comprising various group of individuals including susceptible $S(t)$, asymptomatic infective $I_1(t)$, symptomatic infective $I_2(t)$, treated $T(t)$ and AIDS $A(t)$ class. The validity of the solution states affirms that the model is well-defined and holds epidemiological significance. The disease-free and endemic equilibrium states are identified, and their stability is analyzed using Routh Hurwitz criteria. Sensitivity analysis was carried out using normalized forward sensitivity index and result showed that the contact rate β_1 is the most sensitive parameter. However, it is observed from the numerical simulation that screening and treatment of the infective play a significant role in reducing the transmission of the disease. The outcome of the stability analysis for both disease-free and endemics equilibrium states indicates the potential for HIV/AIDS control.

Keywords: *HIV/AIDS, Saturated Incidence, Screening, Treatment, Basic Reproduction Number, local Stability, global stability, Infective, Sensitivity Analysis.*

INTRODUCTION

HIV/AIDS is seen as a chronic disease nowadays, because HIV-positive people can live with infection for many years provided their immune systems are checked. It gradually destroys the immune system until it is unable to fight infections that would normally have been prevented. With deterioration of the immune system, the body develops opportunistic diseases that lead to AIDS [1]. As the immune system becomes compromised, the HIV opportunistic diseases such as meningitis, Cancers and Tuberculosis do easily attack the body [2].

In 2022, approximately 39million people died from AIDS worldwide, about 740 children became infected with HIV and approximately 274 children died from AIDS related causes every day [3]. More so, in the countries hardest hit, AIDS has sapped the population of young men and women who form the foundation of the labor force. Most die while in the peak of their reproductive years. Moreover, the epidemic has

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overwhelmed health care systems, increases the number of orphans, and caused life expectancy rates to plummet. It therefore constitutes a serious threat to future development in Africa.

At the end of 2022, Approximately 39.0 million (33.1million-45.7million) people were living with HIV globally. 1.3million [1million-1.7million] people became newly infected with HIV while 630000 [480000-880000] people died from AIDS-related illnesses. 1.5 million (1.2-2.1million) children were living with HIV (0-14 years old) [4]. More so, according to HIV/AIDS prevalence estimates between 2023-2024, African countries with HIV/AIDS adult prevalence rates among adults in various countries according to CIA world fact book are Eswatini with 28.30%, Lesotho 24.10%, Botswana 22.60% and Zimbabwe 22.10% and outside Africa, Bahamas has the highest prevalence rate [5].

Transmission of HIV/AIDS causing virus occurs most commonly through sexual intercourse. HIV can also be transmitted through transfusions of HIV-contaminated blood or by using a contaminated needle or syringe to inject drugs into the blood stream. Infection with HIV does not necessarily mean that a person has AIDS. Some people who have HIV infection may not develop any of the clinical illnesses that define the full-blown disease of AIDS for ten years or more. Physicians prefer to use the term AIDS for cases where a person has reached the final, life-threatening stage of HIV infection [6]. In a person infected with HIV, the virus steadily destroys $CD4^{+}T$ cells over a period of years, diminishing the cells protective ability and weakening the immune system. HIV infects some other cells and wrecks the largest part of the $CD4^{+}T$ cells and this causes the $CD4^{+}T$ cells destruction and decline, hence reducing the confrontation of the susceptible system [7], [8].

Screening is the process of performing the HIV-antibody test to all individuals within a defined population. Routine screening of unaware infective has now become an integral part of programs in low and middle income countries. People can get HIV tests done at a health clinic, at special HIV voluntary counseling and testing (VCT) sites [9]. Antiretroviral medication has made it possible for many individuals who have been very sick with HIV/AIDS to become fully functioning again, with a low or even undetectable viral load. Without antiretroviral therapy, someone who has AIDS typically dies within a year [10]. Transmission of the disease and influence of the infection can be curbed by taking adequate treatment and administering suitable vaccine to infected class [11-14,27]. New drugs are available that can prolong the life span and improve the quality of life of infected people [12,22,37].

In the context of mathematical modeling of HIV/AIDS dynamics, Saturation incidence refers to a situation where the rate of rate of new infections reaches a plateau, indicating that the epidemic has stable and under control and therefore no longer growing exponentially. This simply means that the number of new infections is roughly balanced by the number of individuals recovering or dying from the infection.

Various mathematical models were formulated for developing strategies to control the outbreak of disease and thereby making a trade-offs in choosing a best possible treatment for curbing and controlling the infection. Ross Ronald derived a threshold quantity called basic reproduction number. This helps the planners such as medical and health practitioners to determine and conclude when infection will fade out in a system. Incidence rate play a vital and significance role in discussing nature of a disease in epidemiology. In early, bilinear incidence rate in the form βxy and standard incidence rate in the form $\frac{\beta xy}{N}$ are usually used in epidemic model where β is the transmission of the disease per contact. x and y are the number of susceptible population and infected population respectively where N is the total number of population [15]. Many authors studied nonlinear types of incidence rate. Few among them are [15-18, 20]. Many authors studied nonlinear types of incidence rate. To avoid unboundedeness of infection [39-40] introduced saturated types of infection rate in the form $\frac{\beta xy}{(1+\alpha y)}$ where βy denotes force of infection and $\frac{\beta xy}{(1+\alpha y)}$ measures inhibition factors due to crowding effect of infected class and also an appropriate social awareness

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given by the population. α is the coefficient of inhibition. Later, a non-monotone type of infection rate in the form $\frac{\beta xy}{(1+\alpha y^2)}$ was also introduced to include the population psychological effect of population [11, 20]. [21] also introduced Saturated incidence rate $\frac{\beta SI}{(1+\alpha I)}$, tends to a saturation level when it gets large, where βI measures the infection force when the disease is entering a fully susceptible population and $\frac{1}{(1+\alpha I)}$ measures the inhibition effect from the behavioral change of susceptible. The saturated incidence rate of the form $\frac{1}{(1+\alpha_1 S)}$ where α_1 is a positive constant. Here, the effect of saturation factor α_1 stems from epidemic control taking appropriate precautionary measures. The saturated incidence function of the form $\frac{1}{(1+\alpha_2 I)}$, where α_2 is a positive constant. Here, the number of effective contacts between infective individuals or due to the precautionary measures taken by the susceptible individuals.

In recent years, a number of authors have studied epidemiological models with non-linear rates. The most common non-linear incidence rates take the form $\beta I^P S^q$ where I and S are respectively, the number of infective and susceptible individuals, and β, P and q are positive constants. Epidemiological models with this incidence rate have been studied by [28] and later by [29]. Other authors such as [30-32] also incorporated this incidence into their models. [33] independently introduced nonlinear incidence rate known as Bedding-ton and De-Angelis type incidence rate $\left(\frac{\alpha}{(1+\beta S+\gamma I)}\right)$. Later some other researchers also used this incidence rate to describe epidemiological models.

From an epidemiological standpoint, saturation incidence suggests that the epidemic has reached a steady state, with the disease prevalence remaining relatively constant over time. This could simply be due to various factors such as behavioral changes, implementation of prevention strategies, availability of treatment, or natural progression of the epidemic within a population. Understanding saturation incidence therefore helps public health officials and policy makers assess the effectiveness of interventions and plan future strategies for controlling the spread of the menace.

Therefore, in this work, our aim was to investigate the effects of screening and treatment on the transmission of HIV/AIDS epidemic model with saturated incidence function. The paper is organized as follows. In section 2, we formulate and explain the model and show the existence and uniqueness of the model. In section 3, we explore existence of disease free equilibrium point, the endemic equilibrium point and both local and global stabilities of their equilibria were analyzed and the computation of sensitivity analysis and its interpretations were done. Also, we identify the most sensitive parameter which have most effect on basic reproduction number. In section 4, the paper ends with some numerical simulations to support and compliment the theoretical finding.

MODEL FORMULATION

In this research, the susceptible and infectious epidemic model (SI) is considered as presented by [19, 23]. A population size of $N(t)$ was partitioned into 5 subclasses of individuals which are; susceptible, asymptomatic infective, symptomatic infective, treated infective, and AIDS with sizes denoted by $S(t), I_1(t), I_2(t), T(t),$ and $A(t)$, respectively such that $N = S + I_1 + I_2 + T + A,$ as shown in figure 1.

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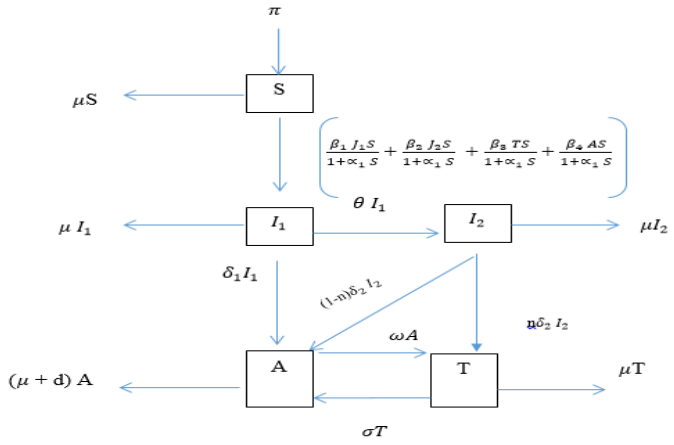


Figure 1: *Transmission diagram for susceptible, infected model with the saturation term to the susceptible*

$(1+\alpha_1 S)$

Following the transmission diagram, in Figure 1, which incorporates the saturation term to the susceptible, asymptomatic infective, symptomatic infective, treated infective and AIDS individual. The following assumptions are taken into consideration in formulating our proposed model: People are recruited into the Susceptible population by birth . All parameters are positive. Asymptomatic infective class (I_1) can move to symptomatic class (I_2) and full blown AIDS and asymptomatic infective population can be screened at a rate θ and progress to symptomatic class. Asymptomatic infective, Symptomatic infective, Treated class (T) and full blown AIDS can infect Susceptible class at different rates $\beta_1, \beta_2, \beta_3,$ and β_4 respectively.

Asymptomatic infective, Symptomatic infective, Treated class (T) will move to full blown AIDS at different rates $\delta_1, \delta_2,$ and σ respectively . Infective can be treated with ARV therapy (i.e can move to treated class) at rate ω.

Considering all the above assumptions made, the following system of ordinary differential equation of the proposed model is therefore considered.:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_3 TS}{(1+\alpha_1 S)} - \frac{\beta_4 AS}{(1+\alpha_1 S)} - \mu S \\
\frac{dI_1}{dt} &= \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 TS}{(1+\alpha_1 S)} + \frac{\beta_4 AS}{(1+\alpha_1 S)} - (\theta + \mu + \delta_1) I_1 \\
\frac{dI_2}{dt} &= \theta I_1 - (\mu + \delta_2) I_2 \\
\frac{dT}{dt} &= n\delta_2 I_2 - (\sigma + \mu) T + \omega A \\
\frac{dA}{dt} &= (1-n)\delta_2 I_2 + \delta_1 I_1 - (\mu + d + \omega) A + \sigma T
\end{aligned}\right\} \qquad (1)$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

As initial condition, we choose

$$S(0)=S_0, I_1(0)=I_{1_0}, I_2(0)=I_{2_0}, T(0)=T_0, A(0)=A_0$$

The model parameters are defined as follows

$\pi>0$ is the constant number of recruitment of susceptible, $\beta_1,\beta_2,\beta_3,\beta_4$ are the transmission rates for susceptible individuals with asymptomatic infective, susceptible individuals with symptomatic infective, susceptible individuals with treated infective, and susceptible individuals with AIDS respectively, α is the Saturation term to the susceptible individual. μ is the natural mortality rate unrelated to AIDS, σ is rate at which treated individual develops to full blown AIDS, ω is the rate at which AIDS patients get treatment, δ_1 is rate of movement from asymptomatic class I_1(t) to full blown AIDS A(t). δ_2 is rate of movement from symptomatic class I_2(t) to treated and AIDS class respectively, *n* is the fraction of symptomatic infective that moved to treated class, *d* is the AIDS related death rate/Disease related death rate and θ is the screening rate.

2.1 Existence and uniqueness of solution

The model is analyzed by proving the existence and uniqueness of solution

Theorem 2.1: (Derrick and Grossman 1976)

Let

$$\frac{dS}{dt}=\pi-\frac{\beta_1 I_1 S}{(1+\alpha_1 S)}-\frac{\beta_2 I_2 S}{(1+\alpha_1 S)}-\frac{\beta_3 TS}{(1+\alpha_1 S)}-\frac{\beta_4 AS}{(1+\alpha_1 S)}-\mu S \qquad ;S(t_0)=S_0$$

$$\frac{dI_1}{dt}=\frac{\beta_1 I_1 S}{(1+\alpha_1 S)}+\frac{\beta_2 I_2 S}{(1+\alpha_1 S)}+\frac{\beta_3 TS}{(1+\alpha_1 S)}+\frac{\beta_4 AS}{(1+\alpha_1 S)}-(\theta+\mu+\delta_1)I_1 \qquad ; I_1(t_0)=I_0$$

$$\frac{dI_2}{dt}=\theta I_1-(\mu+\delta_2)I_2 \qquad ; I_2(t_0)=I_0 \qquad (2)$$

$$\frac{dT}{dt}=n\delta_2 I_2-(\sigma+\mu)T+\omega A \qquad ; T(t_0)=I_0$$

$$\frac{dA}{dt}=(1-n)\delta_2 I_2+\delta_1 I_1-(\mu+d+\omega)A+\sigma T \qquad ; A(t_0)=A_0$$

Let

$$D=\left\{(S,I_1,I_2,T,A):|S-S_0|\le a,|I-I_{1_0}|\le b,|I-I_{2_0}|\le c,|T-T_0|\le d,|A-A_0|\le e\right\}$$

Then equation (1) has a unique solution in D with initial values

$$S(0)=S_0,\ I_1(0)=I_{1_0},\ I_2(0)=I_{20},\ T(0)=T_0,\ A(0)=A_0$$

Proof:

Let

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$\left.\begin{aligned}
\frac{dS}{dt} &= f_1(S,I_1,I_2,T,A) = \pi - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_3 TS}{(1+\alpha_1 S)} - \frac{\beta_4 AS}{(1+\alpha_1 S)} - \mu S \\
\frac{dI_1}{dt} &= f_2(S,I_1,I_2,T,A) = \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 TS}{(1+\alpha_1 S)} + \frac{\beta_4 AS}{(1+\alpha_1 S)} - (\theta+\mu+\delta_1) I_1 \\
\frac{dI_2}{dt} &= f_3(S,I_1,I_2,T,A) = \theta I_1 - (\mu+\delta_2) I_2 \\
\frac{dT}{dt} &= f_4(S,I_1,I_2,T,A) = n\delta_2 I_2 - (\sigma+\mu) T + \omega A \\
\frac{dA}{dt} &= f_5(S,I_1,I_2,T,A) = (1-n)\delta_2 I_2 + \delta_1 I_1 - (\mu+d+\omega) A + \sigma T
\end{aligned}\right\} \quad (3)$$

$$\left.\frac{\partial f_1}{\partial S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{-\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_1 I_1 S\alpha_1}{(1+\alpha_1 S)^2} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S\alpha_1}{(1+\alpha_1 S)^2} - \frac{\beta_3 T}{(1+\alpha_1 S)^2} + \frac{\beta_3 T\alpha_1 S}{(1+\alpha_1 S)} - \frac{\beta_4 A}{(1+\alpha_1 S)} + \frac{\beta_4 AS\alpha_1}{(1+\alpha_1 S)^2} - \mu$$

$$\left|\frac{\partial f_1}{\partial S}\right|_{S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left| - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_1 I_1 S\alpha_1}{(1+\alpha_1 S)^2} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S\alpha_1}{(1+\alpha_1 S)^2} - \frac{\beta_3 T}{(1+\alpha_1 S)^2} + \frac{\beta_3 T\alpha_1 S}{(1+\alpha_1 S)} - \frac{\beta_4 A}{(1+\alpha_1 S)} + \frac{\beta_4 AS\alpha_1}{(1+\alpha_1 S)^2} - \mu \right|$$

$$\left.\frac{\partial f_1}{\partial I_1}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_1 S}{1+\alpha_1 S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_1}{\partial I_1}\right|_{(S_0,I_{1_0},I_{2_0},A_0)} = \frac{\beta_1 S_0}{1+\alpha_1 S_0}$$

$$\left.\frac{\partial f_1}{\partial I_2}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_1 S}{1+\alpha_1 S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_1}{\partial I_2}\right|_{(S_0,I_{1_0},I_{2_0},A_0)} = \frac{\beta_1 S_0}{1+\alpha_1 S_0}$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$\left.\frac{\partial f_1}{\partial T}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{-\beta_3 T}{1+\alpha_1 S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_1}{\partial T}\right|_{(S_0,I_{1_0},I_{2_0},A_0)} = \frac{\beta_3 T_0}{1+\alpha_1 S_0}$$

$$\left.\frac{\partial f_1}{\partial A}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{-\beta_4 S}{1+\alpha_1 S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_1}{\partial A}\right|_{(S_0,I_{1_0},I_{2_0},A_0)} = \frac{\beta_4 S_0}{1+\alpha_1 S_0}$$

$$\left.\frac{\partial f_2}{\partial S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_1 I_1 S\alpha_1}{(1+\alpha_1 S)^2} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S\alpha_1}{(1+\alpha_1 S)^2} + \frac{\beta_3 T}{(1+\alpha_1 S)^2} - \frac{\beta_3 T\alpha_1 S}{(1+\alpha_1 S)} + \frac{\beta_4 A}{(1+\alpha_1 S)} - \frac{\beta_4 AS\alpha_1}{(1+\alpha_1 S)^2}$$

$$\left|\frac{\partial f_2}{\partial S}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left|\frac{\beta_1 I_{10} S_0}{(1+\alpha_1 S_0)} - \frac{\beta_1 I_{10} S_0\alpha_1}{(1+\alpha_1 S_0)^2} + \frac{\beta_2 I_{20} S_0}{(1+\alpha_1 S_0)} - \frac{\beta_2 I_{20} S_0\alpha_1}{(1+\alpha_1 S_0)^2} + \frac{\beta_3 T_0}{(1+\alpha_1 S_0)^2} - \frac{\beta_3 T_0\alpha_1 S_0}{(1+\alpha_1 S_0)} + \frac{\beta_4 A_0}{(1+\alpha_1 S_0)} - \frac{\beta_4 A_0 S_0\alpha_1}{(1+\alpha_1 S_0)^2}\right|$$

$$\left.\frac{\partial f_2}{\partial I_1}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_1 S}{(1+\alpha_1 S)} - (\theta+\mu+\delta_1)\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_2}{\partial I_1}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{\beta_1 S_0}{(1+\alpha_1 S_0)} - (\theta+\mu+\delta_1)$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$\left.\frac{\partial f_2}{\partial I_2}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_1 S}{(1+\alpha_1 S)}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_2}{\partial I_2}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{\beta_1 S_0}{(1+\alpha_1 S_0)}$$

$$\left.\frac{\partial f_2}{\partial T}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_3 S}{(1+\alpha_1 S)}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_2}{\partial T}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{\beta_3 S_0}{(1+\alpha_1 S_0)}$$

$$\left.\frac{\partial f_2}{\partial A}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \left.\frac{\beta_4 S}{(1+\alpha_1 S)}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)}$$

$$\left|\frac{\partial f_2}{\partial A}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \frac{\beta_4 S_0}{(1+\alpha_1 S_0)}$$

$$\frac{\partial f_3}{\partial S} = 0$$

$$\left.\frac{\partial f_3}{\partial I_1}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = \theta$$

$$\left.\frac{\partial f_3}{\partial I_2}\right|_{(S_0,I_{1_0},I_{2_0},T_0,A_0)} = (\mu+\delta_2)$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$\left.\frac{\partial f_3}{\partial T}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = 0$$

$$\left.\frac{\partial f_3}{\partial A}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = 0$$

$$\left.\frac{\partial f_4}{\partial S}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = 0$$

$$\left.\frac{\partial f_4}{\partial I_1}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = 0$$

$$\left.\frac{\partial f_4}{\partial I_2}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = n\delta_2$$

$$\left.\frac{\partial f_4}{\partial T}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = -(\sigma + \mu)$$

$$\left.\frac{\partial f_4}{\partial A}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = \omega$$

$$\left.\frac{\partial f_5}{\partial S}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = 0$$

$$\left.\frac{\partial f_5}{\partial I_1}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = \delta_1$$

$$\left.\frac{\partial f_5}{\partial I_2}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = (1-n)\delta_2$$

$$\left.\frac{\partial f_5}{\partial T}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = \sigma$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$\left.\frac{\partial f_5}{\partial A}\right|_{(S_0, I_{1_0}, I_{2_0}, T_0, A_0)} = (\mu + d + \omega)$$

Hence, by the condition of the theorem $\left|\frac{\partial f_i}{\partial S}\right|, \left|\frac{\partial f_i}{\partial I_1}\right|, \left|\frac{\partial f_i}{\partial I_2}\right|, \left|\frac{\partial f_i}{\partial T}\right|, \left|\frac{\partial f_i}{\partial A}\right|, i = 1...5$ are continuous and bounded. Therefore, the problem (3) has a unique solution.

MATHEMATICAL ANALYSIS OF THE MODEL

3.1 Disease free equilibrium point

This is the equilibrium point at which population remains in the absence of disease. In this case, no strain of the disease is present in the entire population.

At the equilibrium,

$$\frac{dS}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dT}{dt} = \frac{dA}{dt} = 0$$

Equation (1) becomes

$$\pi - \frac{\beta_1 I_1 S}{(1+\alpha_1 S)} - \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} - \frac{\beta_3 T S}{(1+\alpha_1 S)} - \frac{\beta_4 A S}{(1+\alpha_1 S)} - \mu S = 0$$

$$\frac{\beta_1 I_1 S}{(1+\alpha_1 S)} + \frac{\beta_2 I_2 S}{(1+\alpha_1 S)} + \frac{\beta_3 T S}{(1+\alpha_1 S)} + \frac{\beta_4 A S}{(1+\alpha_1 S)} - (\theta + \mu + \delta_1) I_1 = 0$$

$$\theta I_1 - (\mu + \delta_2) I_2 = 0 \qquad (4)$$

$$n\delta_2 I_2 - (\sigma + \mu) T + \omega A = 0$$

$$(1-n)\delta_2 I_2 + \delta_1 I_1 + \sigma T - (\mu + d + \omega) A = 0$$

At disease free,

$S \neq 0, I_1 = I_2 = T = A = 0$ Substituting these to equation (4) and solving gives the infection – free equilibrium as

$$E^0 = \left(S^0, I_1^0, I_2^0, T^0, A^0\right) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$$

3.2 Endemic equilibrium point

The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the endemic equilibrium, $S \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$

Solving equations (4) simultaneously when $S \neq 0, I_1 \neq 0, I_2 \neq 0, T \neq 0, A \neq 0$, we have the endemic equilibrium points respectively as;

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$E^* = \left(S^*, I_1^*, I_2^*, T^*, A^*\right) = \left\{ \begin{array}{l} \dfrac{\pi}{R_0 I_1 + \mu} \\ \dfrac{\pi R_0 - \mu K_1}{K_1 R_0}, \\ \dfrac{\theta I_1}{K_2}, \\ \dfrac{\left(\delta_2\theta\left(\eta\left(\mu K_4 + \sigma(\mu+d)\right) + \omega\left((1-n)+\sigma\right)\right) + \omega K_2 K_3 \delta_1\right) I_1}{K_2\left(\mu K_4 + \sigma(\mu+d)\right)}, \\ \dfrac{\left(\delta_2\theta\left((1-n)+\sigma\right) + K_2 K_3 \delta_1\right) I_1}{K_2\left(\mu G_4 + \sigma(\mu+d)\right)} \end{array} \right\} \quad (5)$$

respectively, where,

$$K_1 = (\theta+\mu+\delta), K_2 = (\mu+\delta_2), K_3 = (\sigma+\mu), K_4 = (\mu+d+\omega)$$

3.3 Derivation of Basic Reproduction Number, R_0

The computation of the basic reproduction number is essential. The basic reproduction number R_0 is defined as the effective number of secondary infections caused by infected individual during his entire period of infectiousness [24]. To determine the next generation matrix for the model, the following are considered:

1. The number of ways that new infections can arise or be created;
2. The number of ways that infections can be transferred between compartments. Thus, the I_1, I_2, T, A compartment of system (1).

Then F_i and V_i are computed as follows using the approach of [34]:

$$F_i = \begin{pmatrix} \dfrac{\beta_1 S I_1}{(1+\alpha_1 S)} + \dfrac{\beta_2 S I_2}{(1+\alpha_1 S)} + \dfrac{\beta_3 S T}{(1+\alpha_1 S)} + \dfrac{\beta_4 S A}{(1+\alpha_1 S)} \\ 0 \\ 0 \\ 0 \end{pmatrix}, \; V_i = \begin{pmatrix} (\theta+\mu+\delta_1) I_1 \\ -\theta I_1 + (\mu+\delta_2) I_2 \\ -n\delta_2 I_2 + (\sigma+\mu) T - \omega A \\ -(1-n)\delta_2 I_2 - \delta_1 I_1 - \sigma T + (\mu+d+\omega) A \end{pmatrix} \quad (6)$$

Let G be the next generation matrix. It comprises of two parts F and V^{-1} where

$$F = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{pmatrix} \dfrac{\beta_1\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_2\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_3\pi}{(\mu+\alpha_1\pi)} & \dfrac{\beta_4\pi}{(\mu+\alpha_1\pi)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad (7)$$

$$V = \left[\frac{\partial V_1(x_0)}{\partial x_j}\right] = \begin{pmatrix} \theta+\mu+\delta_1 & 0 & 0 & 0 \\ -\theta & (\mu+\delta_2) & 0 & 0 \\ 0 & -n\delta_2 & \sigma+\mu & -\omega \\ -\delta_1 & -(1-n)\delta_2 & -\sigma & \mu+d+\omega \end{pmatrix} \quad (8)$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

The basic reproduction number, which is the dominant eigenvalue of the product FV^{-1}, is therefore obtained as:

$$R_0 = \frac{\pi\left(\left(\beta_1 K_2 + \theta\beta_2\right)\left(\mu K_4 + \sigma\left(\mu + d\right)\right) + \beta_3\left(\delta_2\theta\left(\omega + n\left(\mu + d\right)\right) + \omega\delta_1 K_2\right) + \beta_4\left(\delta_2\theta\left(\sigma + \mu\left(1-n\right)\right) + \delta_1 K_2 K_3\right)\right)}{K_1 K_2\left(\mu K_4 + \sigma\left(\mu + d\right)\right)} \quad (9)$$

3.4 Stability analysis of the Disease Free Equilibrium

3.4.1 Local Stability

Theorem 2:

Let $(K_1 + K_2 + K_3 + K_4) > \frac{\beta_1\pi}{\mu}$,

$$\left(K_3(K_1 + K_2 + K_4) + K_4(K_1 + K_2) + K_2K_1\right) > \left(\frac{\beta_1(K_2 + K_3 + K_4) + \beta_2\theta}{\mu}\right),$$

$$\left(K_4(K_1 + K_2) + K_1K_2)K_3 + K_4K_2K_1\right)\mu > \frac{\pi}{\mu}\left[\begin{matrix}\beta_1\left((K_2 + K_4)K_3 + K_2K_4\right) + \beta_2\theta(K_3 + K_4) + \\ \delta_2\theta\left(\beta_4(1-n) + \beta_3 n\right)\end{matrix}\right],$$

$$K_4K_3K_2K_1\left(1 - R_0\right) > 0$$

Then the disease free equilibrium is locally asymptotically stable.

Proof:

The linearized Jacobian matrix at the disease free equilibrium $\left(\frac{\pi}{\mu},0,0,0,0\right)$ is

$$E^0 = \begin{pmatrix} -\mu & -\beta_1 q & -\beta_2 q & -\beta_3 q & -\beta_4 q \\ 0 & \beta_1 q - K_1 & \beta_2 q & \beta_3 q & \beta_4 q \\ 0 & \theta & -K_2 & 0 & 0 \\ 0 & 0 & n\delta_2 & -K_3 & 0 \\ 0 & 0 & (1-n)\delta_2 & \sigma & -K_4 \end{pmatrix} \quad (10)$$

Where,

$$K_1 = \left(\theta + \mu + \delta\right), K_2 = (\mu + \delta_2), K_3 = \left(\sigma + \mu\right), K_4 = \left(\mu + d + \omega\right)$$

The characteristic equation is obtained as

$$\left(\lambda + \mu\right)\left(P_0\lambda^4 + P_1\lambda^3 + P_2\lambda^2 + P_3\lambda + P_4\right) = 0 \quad (11)$$

Clearly, $\lambda = -\mu$

and,

$P_0 = 1,$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$P_1 = (K_1 + K_2 + K_3 + K_4) - \frac{\beta_1 \pi}{\mu},$$

$$P_2 = \left(K_3(K_1 + K_2 + K_4) + K_4(K_1 + K_2) + K_2 K_1\right) - \left(\frac{\beta_1(K_2 + K_3 + K_4) + \beta_2 \theta}{\mu}\right),$$

$$P_3 = \left(K_4(K_1 + K_2) + K_1 K_2)K_3 + K_4 K_2 K_1\right) - \frac{\pi}{\mu}\begin{bmatrix} \beta_1\left((K_2 + K_4)K_3 + K_2 K_4\right) + \\ \beta_2\theta(K_3 + K_4) + \delta_2\theta\left(\beta_4(1-n) + \beta_3 n\right) \end{bmatrix}$$

$$P_4 = K_4 K_3 K_2 K_1 (1 - R_0) > 0$$

$$P_0 > 0, P_1 > 0, P_2 > 0, P_3 > 0, P_4 > 0 \Rightarrow R_0 > 1,$$

then by Routh Hurwitz criteria, the remaining four eigenvalues are negative. Hence, the disease free equilibrium is locally asymptotically stable.

3.4.2 Stability of the Endemic equilibrium

Theorem 3

Let

$$K_1 + K_2 + K_3 + K_4 + A > \beta_1 S,$$

$$(K_1 + K_2)(A + K_3 + K_4) + A(K_4 + K_3)K_1 K_2 + \mu K_4 + \sigma(\mu + d) + \beta SB > \beta S(A + K_4 + K_3 + K_2)$$

$$\begin{pmatrix} B\beta_2\theta + (K_4 + K_3)((K_1 + K_2)A + K_1 K_2 + \beta_1 B) + (\mu K_4 + \sigma(\mu + d))(A + (K_1 + K_2)) \\ + AK_1 K_2 \end{pmatrix} >$$
$$S\begin{pmatrix} \beta_1(\mu K_4 + \sigma(\mu + d)) + BK_2 + (\beta_1 A + \beta_2\theta + K_2)(K_3 + K_4) + (\beta_2\theta + K_2)A + \\ \delta_2\theta(\beta_3 n + \beta_4(1-n)) \end{pmatrix}$$

$$\begin{pmatrix} (\mu K_4 + \sigma(\mu + d))((K_1 + K_2)A + K_1 K_2)AK_1 K_2(K_3 + K_4) + \\ BS((\beta_1 + \beta_2\theta)K_2(K_3 + K_4) + \beta_1(\mu K_4 + \sigma(\mu + d)) + +(\beta_3 n + \beta_4(1-n))\delta_2\theta) \end{pmatrix} >$$
$$S\begin{Bmatrix} \beta_1[(\mu K_4 + \sigma(\mu + d))(K_2 + A) + K_2(K_3 + K_4)A] \\ \beta_2\theta((K_3 + K_4)(A) + (\mu K_4 + \sigma(\mu + d))) + \beta_3(n(A + K_4) + (1-n)\omega)\theta\delta_2 + \\ \beta_4((1-n)A + \delta_2\theta[(1-n)K_3 + n\theta]) \end{Bmatrix}$$

And

$$` K_1 K_2(\mu K_4 + \sigma(\mu + d))(A + R_0 B)S + \beta_3 B(1-n)\omega\theta S > (K_1 K_2(\mu K_4 + \sigma(\mu + d))R_0 + \beta_3(1-n)\omega\theta)AS$$

Then the endemic equilibrium point $(S^*, I_1^*, I_2^*, T^*, A^*)$ is locally asymptotically stable.

Proof:

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

Equations (1) can be expressed in matrix form as the Jacobian matrix of the equation (1), at endemic equilibrium point $(S^*, I_1^*, I_2^*, T^*, A^*)$

Let $$C^* = \begin{pmatrix} A & -\beta_1 S^* & (-\beta_2 S^*) & -\beta_3 S^* & -\beta_4 S^* \\ B & (\beta_1 S^* - (\theta+\mu+\delta_1)) & \beta_2 S^* & \beta_3 S^* & \beta_4 S^* \\ 0 & \theta & -(\mu+\delta_2) & 0 & 0 \\ 0 & 0 & n\delta_2 & -(\sigma+\mu) & \omega \\ 0 & \delta_1 & ((1-n)\delta_2) & \sigma & -(\mu+d+\omega) \end{pmatrix} + \textit{nonlinear terms} \quad (12)$$

where,

$$A = \begin{pmatrix} -\beta_1 I_1^* + \beta_1 I_1^* \alpha_1 S^* + \beta_1 I_1^* \alpha_2 I_1^{**} - \beta_2 I_2^* + \beta_2 I_2^* \alpha_1 S^* + \beta_2 I_2^* \alpha_2 I_1^{**} + \beta_2 I_2^* S^* \alpha_1 - \beta_3 T^* \\ + \beta_3 T^* \alpha_1 S^* + \beta_3 T^* \alpha_2 I_1^* + \beta_3 T^* s^* \alpha_1 - \beta_4 A^* + \beta_4 A^* \alpha_1 S^* + \beta_4 A^* \alpha_2 I_1^{**} + \beta_4 A^* s^* \alpha_1 - \mu \end{pmatrix}$$

$$B = \left(-\beta_1 S^* + \beta_1 S^* \alpha_2 I_2^* + \beta_1 I_1^* S^* \alpha_2 + \beta_2 I_2^* S^* \alpha_2 + \beta_3 T^* s^* \alpha_2 + \beta_4 A^* s^* \alpha_2\right)$$

Therefore the characteristics equation

$$|C^* - \lambda I| = 0 \Rightarrow \begin{vmatrix} A-\lambda & -\beta_1 S^* & -\beta_2 S^* & -\beta_3 S^* & -\beta_4 S^* \\ B & (\beta_1 S^* - (\theta+\mu+\delta_1)) - \lambda & \beta_2 S^* & \beta_3 S^* & \beta_4 S^* \\ 0 & \theta & -(\mu+\delta_2) - \lambda & 0 & 0 \\ 0 & 0 & n\delta_2 & -(\sigma+\mu) - \lambda & \omega \\ 0 & \delta_1 & (1-n)\delta_2 & \sigma & -(\mu+d+\omega) - \lambda \end{vmatrix} = 0 \quad (13)$$

Evaluating for λ in (13) yields the characteristic equation

$$\lambda^3 + Z_1\lambda^4 + Z_2\lambda^3 + Z_3\lambda^2 + Z_4\lambda + Z_5 = 0 \quad (14)$$

Where.

$$Z_0 = 1$$

$$Z_1 = K_1 + K_2 + K_3 + K_4 + A - \beta_1 S$$

$$Z_2 = (K_1 + K_2)(A + K_3 + K_4) + A^*(K_4 + K_3)K_1K_2 + \mu K_4 + \sigma(\mu + d) - \beta S(A + K_4 + K_3 + K_2 - B)$$

$$Z_3 = \beta_1\left[\{-(K_3 + K_4 + K_2)A - (K_4 - B + K_2)K_3 + (K_2 - B)K_4 - BK_2 - \sigma\omega\}\right]S$$
$$- \beta_2\{\theta[(A - B) + K_3 + K_4]\} - \beta_3 n\delta_2\theta S - \beta_4\delta\theta(1-n)S + (K_4 + K_3)[(K_1 + K_2)A + K_1K_2]$$
$$+ (\mu K_4 + \mu(\mu + d))[A + (K_1 + K_2)]K_1K_2A$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$$Z_4 = (\mu K_4 + \sigma(\mu+d))((K_1+K_2)A + K_1K_2)AK_1K_2(K_3+K_4) + BS\begin{pmatrix}(\beta_1+\beta_2\theta)K_2(K_3+K_4)+\\ \beta_1(\mu K_4+\sigma(\mu+d))+\\ +(\beta_3 n+\beta_4(1-n))\delta_2\theta\end{pmatrix}$$

$$-S\begin{Bmatrix}\beta_1[(\mu K_4+\sigma(\mu+d))(K_2+A)+K_2(K_3+K_4)A]\\ \beta_2\theta((K_3+K_4)(A)+(\mu K_4+\sigma(\mu+d)))+\beta_3(n(A+K_4)+(1-n)\omega)\theta\delta_2+\\ \beta_4((1-n)A+\delta_2\theta[(1-n)K_3+n\theta])\end{Bmatrix}$$

$$Z_5 = K_1K_2(\mu K_4+\sigma(\mu+d))(A+R_0B)S+\beta_3B(1-n)\omega\theta S -$$
$$(K_1K_2(\mu K_4+\sigma(\mu+d))R_0+\beta_3(1-n)\omega\theta)AS$$

Using Descartes rule of sign change, there is no sign change in the roots of the equation (14)

If the following assumptions holds, i.e

$Z_1 > 0$, provided $(K_1+K_2+K_3+K_4+A) > \beta_1 S$

$Z_2 > 0$, provided
$(K_1+K_2)(A+K_3+K_4)+A(K_4+K_3)K_1K_2+\mu K_4+\sigma(\mu+d) > \beta S(A+K_4+K_3+K_2-B)$

Then the endemic equilibrium point $(S^*, I_1^*, I_2^*, T^*, A^*)$ is locally asymptotically stable, it follows from Descartes rule of signs, that equation (14) have no change in sign meaning there are no positive roots.

Therefore, since all eigenvalues are negative then the endemic or disease equilibrium is locally asymptotically stable

3.4.3 Global stability for disease free equilibrium

Theorem 3: The disease free equilibrium of system (4) is globally asymptomatically stable

If $R_1 > 0, R_2 > 0, R_3 > 0,$ and $R_4 > 0$

Proof:

Using Comparison theorem as implemented in [35-36] that the rate of change of the infected compartment of system (1) can be written as

$$\begin{bmatrix}\frac{dx}{dt}\\ \frac{dy}{dt}\\ \frac{dz}{dt}\\ \frac{dp}{dt}\end{bmatrix} = (F-V)\begin{bmatrix}x\\ y\\ z\\ p\end{bmatrix} - F_i\begin{bmatrix}x\\ y\\ z\\ p\end{bmatrix} \qquad (15)$$

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

Where

$$F=\begin{pmatrix}\beta_1 q & \beta_2 q & \beta_3 q & \beta_4 q\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0\end{pmatrix}, V=\begin{pmatrix}K_1 & 0 & 0 & 0\\ -\theta & K_2 & 0 & 0\\ 0 & -n\delta_2 & K_3 & -\omega\\ -\delta_1 & -(1-n)\delta_2 & -\sigma & K_4\end{pmatrix}$$

$$\Rightarrow F-V=\begin{pmatrix}\beta_1 q-K_1 & \beta_2 q & \beta_3 q & \beta_4 q\\ \theta & -K_2 & 0 & 0\\ 0 & n\delta_2 & -K_3 & \omega\\ \delta_1 & (1-n)\delta_2 & \sigma & -K_4\end{pmatrix}=0 \qquad (16)$$

Where,

$$K_1=(\theta+\mu+\delta_{1,}), K_2=(\mu+\delta_2), K_3=(\sigma+\mu), K_4=(\mu+d+\omega)$$

$$q=\frac{\pi}{(\mu+\alpha_1\pi)}$$

then all eigenvalues of (F-V) are all negative i.e,

Simplifies to give

$$a_0\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0 \qquad (17)$$

Where,

$$a_0=1$$

$$a_1=K_1+K_2+K_3+K_4-\beta q_1$$

$$a_2=(K_4+K_1+K_3)K_2+K_1(K_3+K_4)+\mu K_4+\sigma(\mu+d)-q(\beta_1(K_2+K_3+K_4)+\beta_2\theta+\beta_3\delta_1)$$

$$a_3=K_1(K_3+K_4)+\mu K_4+\sigma(\mu+d)-q\begin{pmatrix}\beta_1(K_2(K_4+K_3)+\mu K_4+\sigma(\mu+d))+\beta_2\theta(K_4+K_3)+\\ \beta_3(\delta_2 n\theta+\delta_1\omega)+\beta_4(\delta_1(K_3+K_2)+\delta_2(1-n)\theta)\end{pmatrix}$$

$$a_4=K_3K_2(\mu K_4+\sigma(\mu+d))(1-R_0)$$

Then, equation (17) will have no positive root if $R_0<1$ and by Descarte's rule of signs, all eigenvalues are negative, Therefore the disease free equilibrium is globally asymptotically stable (GAS).

3.4.5 GLOBAL STABILITY OF ENDEMIC EQUILIBRIUM

Theorem 4: If $R_0>1$, then the endemic equilibrium point of the model equation (1) is globally asymptotically stable in Ω, provided $S\geq S^*, I_1\geq I_1^*, I_2\geq I_2^*, T>T^*$, and $A\geq A^*$.

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

Proof: To establish the global stability of the endemic equilibrium E^*, following the approach of [26], we analyzed by constructing the following quadratic Lyapunov function L, such that

$$L = \frac{1}{2}\left[\left(S - S^*\right) + \left(I_1 - I_1^*\right) + \left(I_2 - I_2^*\right) + \left(A - A^*\right) + \left(T - T^*\right)\right]^2$$

By direct calculation of the time derivatives $L(t)$ along the solutions of the system (1) is obtained as

$$\frac{dL}{dt} = \left[\left(S - S^*\right) + \left(I_1 - I_1^*\right) + \left(I_2 - I_2^*\right) + \left(T - T^*\right) + \left(A - A^*\right)\right]\left(\frac{dS}{dt} + \frac{dI_1}{dt} + \frac{I_2}{dt} + \frac{dT}{dt} + \frac{dA}{dt}\right)$$

Substituting the appropriate solutions of the system (1) into the derivative of $L(t)$ gives

$$\frac{dL}{dt} \le \left[\left(S - S^*\right) + \left(I_1 - I_1^*\right) + \left(I_2 - I_2^*\right) + \left(T - T^*\right) + \left(A - A^*\right)\right]\frac{dN}{dt}$$

$$\frac{dL}{dt} \le \left[\left(S - S^*\right) + \left(I_1 - I_1^*\right) + \left(I_2 - I_2^*\right) + \left(T - T^*\right) + \left(A - A^*\right)\right]\left(\Lambda - \mu N\right)$$

$$\le \left(N - \frac{\Lambda}{\mu}\right)\left(\Lambda - \mu N\right) \tag{18}$$

We obtain the result by rearranging and simplifying (18)

$$\le -\left(\frac{\Lambda}{\mu} - N\right)\left(\Lambda - \mu N\right)$$

$$\le -\frac{1}{\mu}\left(\Lambda - \mu N\right)\left(\Lambda - \mu N\right)$$

$$\le -\frac{1}{\mu}\left(\Lambda - \mu N\right)^2$$

Let

$$\xi = \Lambda - \mu N$$

$$\Rightarrow \frac{dL}{dt} \le -\frac{1}{\mu}\xi^2 \tag{19}$$

Hence,

$\left(\frac{dL}{dt}\right)(S, I_1, I_2, T, A) \le 0$ and $\frac{dL}{dt} = 0$, if and only if $S = S^*, I_1 = I_1^*, I_2 = I_1^*, T = T^*, A = A^*$ Therefore, If $X < Y$, then, $\frac{dL}{dt}$ will be negative definite, implying that $\frac{dL}{dt} = 0$, if and only if

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

$S = S^*, I_1 = I_1^*, I_2 = I_2^*, T = T^*,$ and $A = A^*$. Therefore, the largest positive invariant set in $\left\{\left(S^*, I_1^*, I_2^*, T^*, A^*\right) \in \Omega : \frac{dL}{dt} = 0\right\}$ is a singleton $\{E_1\}$, where E_1 is globally asymptotically stable in the set Ω in accordance to LaSalle's invariant principle [37], it then implies that E_1 is globally asymptotically stable in Ω .

3.5 Computing numerical sensitivity

Using [25], approach the normalized forward sensitivity index of a variable "P" that depends differentiable on a parameter "q" is defined as

$$X_q^p := \frac{\partial p}{\partial q} * \frac{q}{p} \tag{20}$$

As we have an explicit formula for R_0 in equation (9), we derive an analytical expression for the sensitivity of R_0, as $X_q^p := \frac{\partial p}{\partial q} * \frac{q}{p}$ with respect to each of the parameters involved in R_0 as follows:

Table 3.1: Numerical values of sensitivity indices of R_0

Parameter	Sensitivity indices	Values	Source
β_1	0.86	+ 0.847905341	[19]
θ	0.1	+ 0.0102753420	[23]
μ	0.02	- 0.4949636613	Assumed Value
ω	0.01	+0.0768207413	[23]
α_1	0.1	- 0.4949636613	Assumed value
δ_1	0.2	-0.7712241608	[19]
σ	0.001	-0.0037923097	[19]
d	1.0	-0.0893691487	[39]
β_2	0.15	+0.049296821	[19]
β_3	0.10	+0.0884620457	[19]
β_4	+ 0.08	+ 0.0143357909	Assumed value

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

π	0.2	+0.2857142856	Assumed value
δ_2	0.01	-0.2692217346	[19]

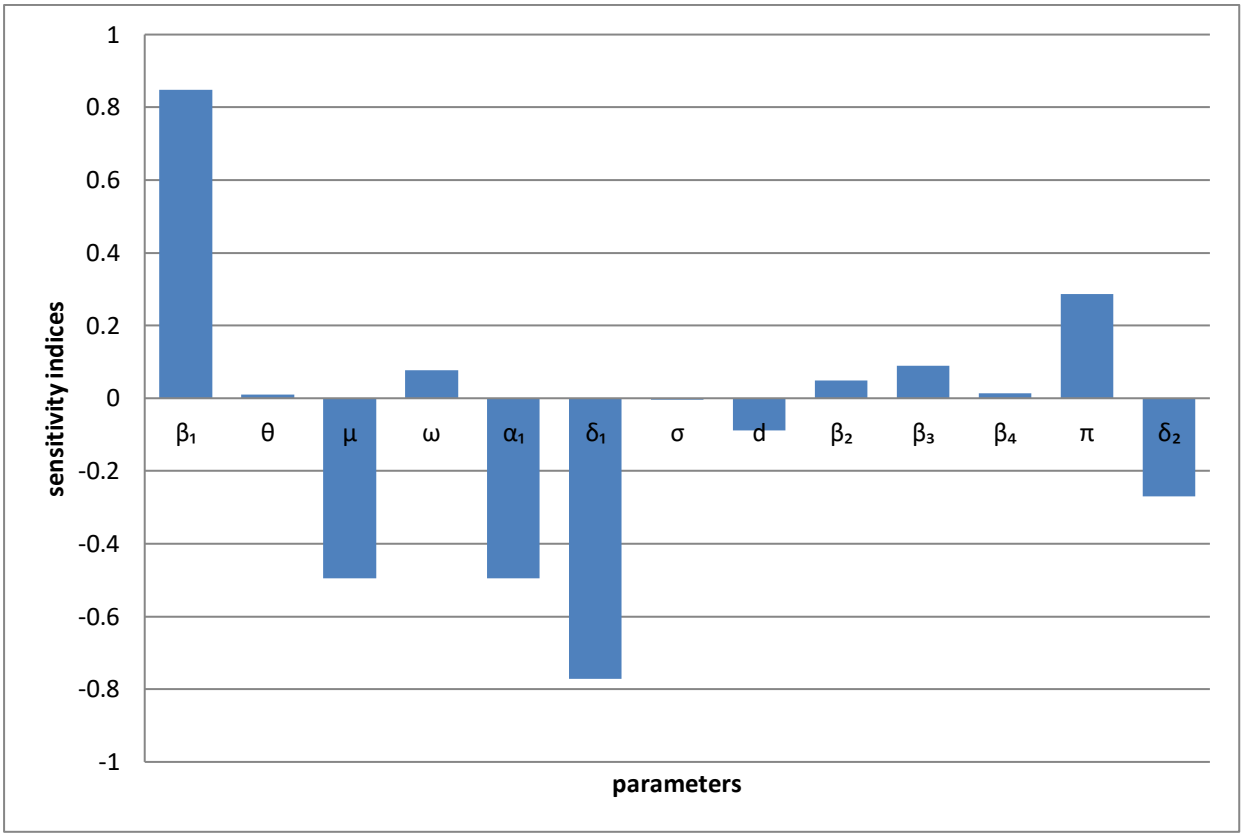


Figure 2: Graphical Representation of the Sensitivity indices of R_0

3.5.1 Interpretation of Sensitivity Indices

The Table 3.1 represents the sensitivity index for the base line parameter values and it shows that when the parameters $\beta_1, \pi, \omega, \beta_4, \theta, \beta_3$ and β_2 increase while the other parameters remain constant, the value of R_0 will also increase implying that they increase the endemicity of the disease as they have positive indices and should be targeted by intervention strategies.

More so, when the parameters $\delta_1, \alpha_1, \mu, \delta_2, d, n$ and σ increase while keeping other parameters constant, the value of R_0 will decrease, implying that they decrease the endemicity of the disease as they have negative indices and should equally be targeted by intervention strategies. From the table, it was revealed that the most sensitive parameter is the transmission rates for the susceptible individuals with asymptomatic infective (β_1) followed by rate of movement from asymptomatic class to AIDS class (δ_1) when we take absolute values of the sensitivity index. For instance, $X_{\beta_1}^{R_0}$ = +0.8479053410 means that increasing or decreasing β_1 by 10% increases or (decreases) R_0 by 8.479053410% while $X_{\mu}^{R_0}$ =- 0.4949636613 means that increasing or (decreasing) μ by 10% decreases (or increases) R_0 by 4.949636613%. we can easily calculate for every other parameters following similar trend.

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

NUMERICAL SIMULATIONS AND DISCUSSION

This section presents the numerical simulation results for the HIV/AIDS model.

The parameters in the model (1) shown in table 4.1 were obtained from literatures published by different researchers and referenced accordingly.

Table 4.1: **parameters, values and source used in the model**

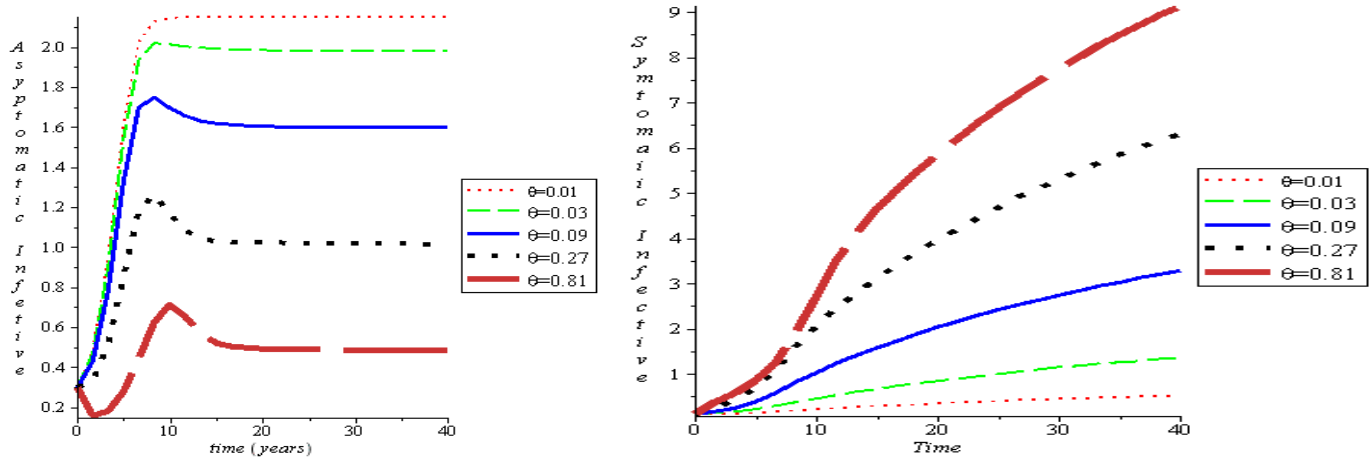
Parameter	Value	Source
d	1.0	[39]
θ	0.01	[23]
μ	0.02	Assumed
ω	0.1	[23]
n	0.2	Assumed
α_1	0.1	Assumed
δ_1	0.2	[19]
δ_2	0.01	[19]
σ	0.001	[19]
β_1	0.86	[19]
β_2	0.15	[19]
β_3	0.10	[19]
β_4	0.08	Assumed

4.1 Numerical Simulation

Simulation of the model was performed for better understanding of dynamical spread of transmission of HIV/AIDS infection using Maple 18 software.

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

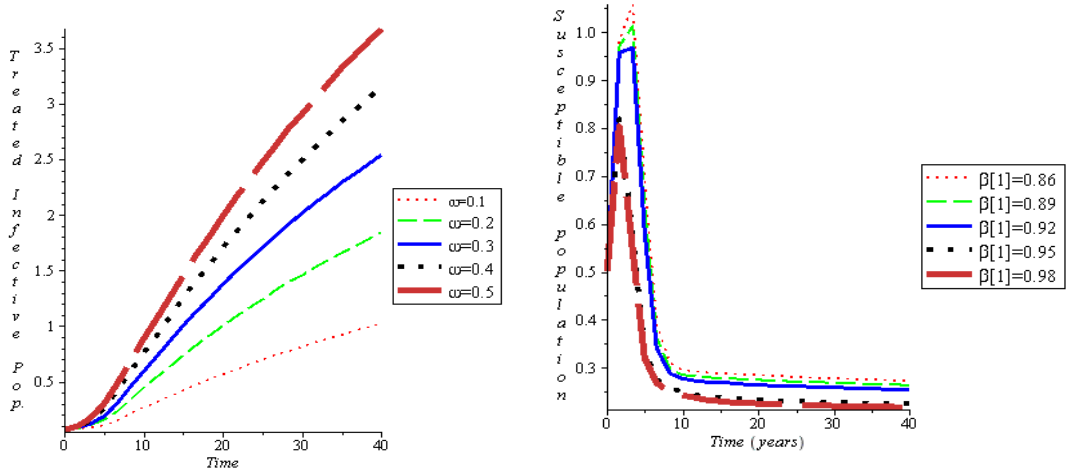
The result of the model equations are presented below in form of graphs and are discussed in the figure below to illustrate the changes in the compartments. The screening rate, treatment rate and the saturation terms were checked in order to observe their impact on the numerical spread of the disease using a set of reasonable parameters.



(a) (b)

Figure 1(a) depicts the effect of screening rates (θ) on asymptomatic infective population $I_1(t)$ against time (t) and symptomatic infective $I_2(t)$ population.

As the asymptomatic HIV infective population become aware of their infection, there is a decrease in the population of asymptomatic infective population which ultimately bring about an increase in the population of symptomatic infective population as seen in **Figure 1(b)**. It was observed here that there is an increase in the population of symptomatic infective as the screening rate increases. This is simply due to the population of asymptomatic infective that migrated to the symptomatic infective compartment as a result of awareness gotten.



(a) (b)

Figure 2(a) depicts the graph of Treated infective population $T(t)$ against Time (t) for different values of treatment rate and **Figure 2(b)** depicts the graph of susceptible Population against time (t) for different values of transmission rate for susceptible individuals with asymptomatic infective (β_1). The symptomatic infective undergo treatment with antiretroviral therapy which as a result leads to an increase in the

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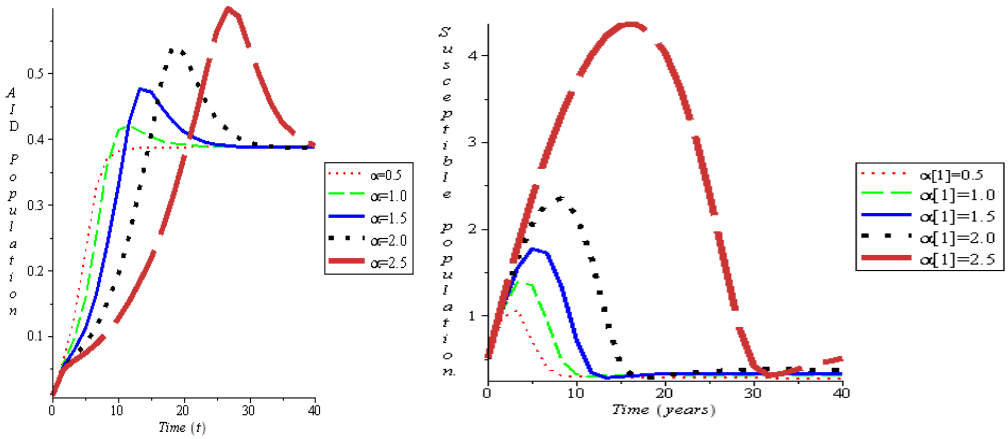
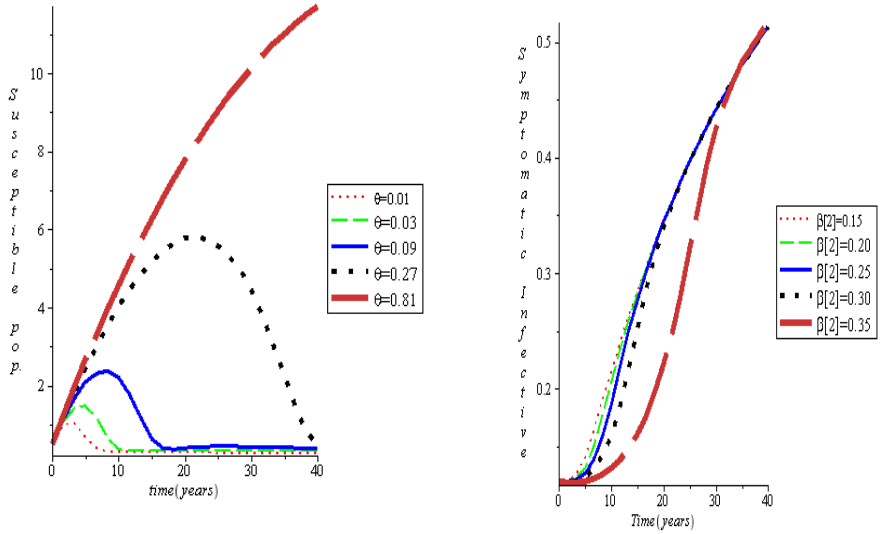
proportion of symptomatic infective population which ultimately leads to an increase in the proportion of treated infective populations as shown in **Figure (2a)** while **Figure (2b)** shows that as transmission rate increase, the susceptible population decrease significantly. It is clearly seen that as the transmission parameter values increase in the population, the number of susceptible population also reduce. As long as susceptible population continue to interact with the infective without taking any preventive measures at any of the infective stages, there is going to be a continuous reduction in the susceptible population.



(a) (b)

Figure (3a) depicts the graph of Aids population A(t) against time (t) for different values of saturation term α_1 while **Figure (3b)** depicts the graph of susceptible population against time for various values of saturation term.

This shows that as the saturation term α_1 increases, the AIDS population decreases. This is simply due to the crowding effects of AIDS individual and other infective as seen in **Figure (3a)** while also, as the saturation term α_1 increases in **Figure (3b)**, the susceptible population increases. This is simply due to the precautionary measures taken by susceptible individual.



(a) (b)

Figure (4a) depicts the graph of Susceptible population S(t) against time (t) for different rates of (θ) while **Figure (4b)** depicts the graph of symptomatic population against time for various values of screening rate.

ISSN No.2454-6194| DOI: 10.51584/IJRIAS| Volume IX Issue IV April 2024

This graph of **(4a)** shows that as the screening rate increases, the susceptible population increases also. When susceptible population go for screening, they become more careful and aware of their health status. This is why the susceptible population increases because they refuse to interact with other effective as seen in **Figure (4a).** The susceptible population must therefore take to and adhere with all precautionary measures to prevent the contact while also in **Figure (4b)**, the susceptible population decreases as the contact rate (β_2) increases.

CONCLUSION

In this paper, we formulated and presented a mathematical model for HIV/AIDS transmission, which incorporated saturated incidence term. It was shown that the system of equation represents a useful mathematical model of a physical system by carrying out a classical qualitative proof of the positivity of solution of the governing system of model equations. More so, the existence of both the disease-free and endemic equilibria were established and analyzed for stability, and it was shown that both equilibria was locally asymptotically stable. This implies that whenever the said conditions are satisfied, the HIV/AIDS infection can be controlled. The result of the sensitivity analysis showed the transmission rates for susceptible individuals with asymptomatic infective β_1, as the most sensitive parameter.

However, screening and treatment of the infective have a significant effect in reducing the transmission of the disease. Susceptible population also reduce when infective carelessly interact with other infective and also susceptible population increase as saturation increase due to the precautionary measures taken. Susceptible population should therefore follow preventive procedures and healthy regulations about the AIDS disease.

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