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# MATHEMATICAL MODEL OF THE SOCIAL PATHOGEN OF HIV/AIDS STIGMA

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**Abstract.** This study incorporated HIV/AIDS stigma as a social pathogen to understand its impact on transmission and treatment. Integrating stigma into epidemiological models, we seek to quantify its effects on the spread of HIV, the progression to AIDS, and the success of treatment interventions. The results show that higher stigma leads to more infections, faster progression to AIDS and fewer treated individuals. The model highlights the need for stigma reduction interventions like education and supportive policies to improve health outcomes and control HIV/AIDS.

**Keywords:** HIV/AIDS; stigma; transmission; intervention.

**2020 AMS Subject Classification:** 34A05, 92D30, 92D25.

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## 1. INTRODUCTION

In 2019 a young woman called Maria in a small town in Kenya was diagnosed with HIV. Despite the advances in HIV treatment, Maria was shunned by her community [31]. Friends stopped visiting, family members avoided her and she was fired from her job . Maria's experience is not alone. According to UNAIDS, 50% of people living with HIV globally have reported some form of discrimination because of their status [8][27]. This stigma not only affects their mental and physical health but also spreads HIV.

HIV/AIDS continues to be a global public health challenge, affecting millions of individuals worldwide. While biomedical advancements have significantly improved the management and treatment of HIV, the social aspects of the disease remain a considerable obstacle. One of the most pervasive and detrimental social factors is the stigma associated with HIV/AIDS. Stigma can manifest in various forms, including social ostracism, discrimination, and internalized shame among those living with the disease. This stigma not only hampers efforts to prevent and treat HIV but also exacerbates the psychological and physical health outcomes for affected individuals.

HIV/AIDS stigma is a multifaceted social pathogen that influences individual behaviors and societal norms. It can deter individuals from seeking testing and treatment, hinder adherence to antiretroviral therapy (ART), and reduce the overall effectiveness of public health interventions. The stigma is often fueled by misconceptions about the transmission and nature of HIV, as well as by pre-existing prejudices related to sexuality, drug use, and marginalized populations.

Stigma means devaluing and discriminating against people because of certain attributes or conditions and can lead to social exclusion and psychological harm. In the context of HIV/AIDS stigma shows up as fear, prejudice and misinformation and affects those living with the virus [7][1][3][4]. This paper argues that stigma works like a pathogen. Just as biological pathogens spread disease through a population, social pathogens like stigma spread bad attitudes and behaviours and makes life harder for those living with HIV/AIDS. Biological pathogens infect the body, causing physical illness and potentially leading to widespread health issues [39]. The body responds with its immune system, attempting to fight off the infection[22]. Some pathogens mutate and develop resistance to treatments, making them persistent and harder to eradicate [40] thereby leading to mortality. It can be managed through public health measures such as vaccinations, hygiene practices, and quarantine [41]. Patients require medical treatment and rehabilitation. On the other hand, stigma as a social pathogen infects the social body (communities, societies) causing psychological distress, marginalization, and social exclusion [45]. It can lead to reduced self-esteem, mental health issues, and decreased opportunities for the stigmatized individuals or groups [44] [43][42]. Societies respond with awareness campaigns, education, and legislation to combat stigma. Individuals develop coping mechanisms and resilience[47][48]. Stigma can persist over time, evolving and adapting to new contexts. Efforts to reduce stigma can face resistance due to deeply ingrained beliefs and cultural norms[49] thereby leading to social morbidity, such as increased rates of mental health disorders, discrimination, and social inequalities[50]. It burdens social services and impacts economic productivity.

This stigma comes from misconceptions about the disease, moral judgments about how it is contracted and fear of contagion[5] [6] [2]. Historically HIV/AIDS has been linked to marginalized groups such as LGBTQ+ individuals, sex workers and drug users and that has added to the stigma [9][16]. Stigma shows up in many forms including social exclusion, discrimination in healthcare and employment and internalized stigma where individuals feel ashamed of their condition[24].

The concept of a social pathogen helps us understand how stigma spreads and perpetuates within communities [26]. A social pathogen, like a biological pathogen, can be transmitted from person to person through social interactions, media and institutional practices[12] [11]. It infects the social fabric and leads to widespread prejudice and discrimination [14] [13]. Misconceptions about HIV transmission (e.g. you can get it from casual contact) fuel stigma. Education and correct information is key to fighting these myths. In many cultures HIV is seen as a moral failure, stigmatizing those who are infected. These norms are perpetuated through religious teachings and traditional beliefs[22][21]. Media plays a dual role in perpetuating and combating stigma. Negative portrayals of people living with HIV/AIDS (PLWHA) [10][15] can reinforce stereotypes and prejudices, while positive and accurate representations can reduce stigma. Policies and practices within institutions like healthcare, law enforcement and the workplace can either combat or contribute to stigma. Discriminatory practices and lack of supportive policies can exacerbate stigmatization[13].

Stigma has big impact on individuals and communities. Fear of stigma means delayed testing and treatment, not disclosing HIV status and poor treatment adherence which worsens health outcomes and increases transmission[5] [6]. People facing stigma experience social isolation, loss of social support and damaged relationships[14][44]. This isolation can lead to mental health issues like depression and anxiety. Stigma means loss of employment, reduced productivity and increased healthcare costs due to late treatment[13]. We must address this stigma for the health and well-being of PLWHA and for public health[24]. By treating stigma as a social pathogen we can understand how it works and develop interventions to combat it.

Mathematical models have been developed by academics to better understand HIV/AIDS dynamics. Epidemiological models for HIV infection and spread in Africa have been studied [3], [17][30], and [18],[19], and [20]. Some models incorporate characteristics that impact the behavior of people living in high-HIV-prevalence societies [34][23] . Only a few infectious illness models explicitly integrate stigma. One system of four ODEs[32] has been used to show dynamics and game theoretical results for stigmatization and disease prevalence in a general infectious disease context [29]. Two recent studies used structural equation modeling and cohort scenario analysis to look at stigma on African women with HIV [25] [28]. A data-driven approach to create a time-dependent stigma function that captures both the level of internalized and enacted stigma in the population has been studied. The model explores a range of scenarios in which either internalized or enacted stigma levels vary from those predicted by the data [24].

In this work, we propose a mathematical model that incorporate HIV/AIDS stigma as a social pathogen to understand its impact on transmission and treatment. By including stigma in an epidemiological model with compartments for susceptible $S$, HIV infected $H$, AIDS $A$, people receiving antiretroviral therapy $P$ and virally suppressed individuals $V$, we show how stigma increases transmission rates and decreases treatment seeking. The model highlights the need for stigma reduction interventions like education and supportive policies to improve health outcomes and control HIV/AIDS.

## 2. MATHEMATICAL MODELING APPROACH

The number of people at any time $t$, shown as $N(t)$, is split into five groups: healthy people(susceptible) $S(t)$, people with HIV $H(t)$, people with AIDS $A(t)$, individual receiving antiretroviral therapy $P(t)$ and people whose virus is under control(virally suppressed group) $V(t)$ In some diseases, people who get better might stay immune forever or might get sick again, but for HIV/AIDS, there is no "Recovered" group since there's no cure. Instead, we have the virally suppressed group $V$, where people have very low virus levels and don't spread the disease much. Both the HIV group and the AIDS group can spread HIV/AIDS to healthy people(susceptible) directly or indirectly. New people join the susceptible at a rate $\Lambda$, and people get infected by others based on the number of contacts they have. The natural mortality rate is represented by $\mu$. How often people have contact that can spread the infection represent $\beta$, and the fraction of people with HIV/AIDS represents $\frac{H+A+P}{N}$. The stigma level is $\sigma$. The term $(1+m\sigma)$ shows how stigma affects the risk of spreading the infection, where $m$ is a factor that changes how stigma impacts this risk. The rate at which new infections happen because of stigma is

$$\lambda = \beta\left(\frac{H+A+P}{N}\right)(1+m\sigma)$$

After some time, $\psi^{-1}$, an infected person will develop full-blown AIDS. Individual with HIV-$H(t)$ and AIDS-$A(t)$ receiving antiretroviral therapy $P(t)$ at the rate $\phi_1$ and $\phi_2$ respectively. The term $\left(\dfrac{1}{1+k\sigma}\right)$ is the stigma impact on treatment seeking, where $k$ is modification factor of stigma on treatment-seeking behavior. Therefore $\phi_1 H\left(\frac{1}{1+k\sigma}\right)$ and $\phi_2 A\left(\frac{1}{1+k\sigma}\right)$ represent the rate of infectious individuals $H$ and $A$ seeking treatment due to stigma. However, if someone with an undetectable viral load $V$ stops treatment, the viral load might go up again, making the person move back to group $H(t)$ (infectious) or even to group $A$ with rates $\varepsilon_1$ and $\varepsilon_2$ respectively. We also consider that people in group $A$ can die from HIV-related causes at rate $\delta_1$ and people in group $V$ can die at rate $\delta_2$. When people receive antiretroviral therapy and it is effective, individual becomes virally suppressed $(V)$ at the rate $\chi$. To integrate the education aspect, a new compartment labeled ( $R-$ removed) can be introduced which indicates persons who via educational efforts change their sexual behavior to minimize their vulnerability to HIV transmission [46]. The parameter $\eta_1$ indicates the pace at which individuals adopt this protective action. However, these people may revert to their prior habits, making them susceptible to infection at a rate $\eta_2$.

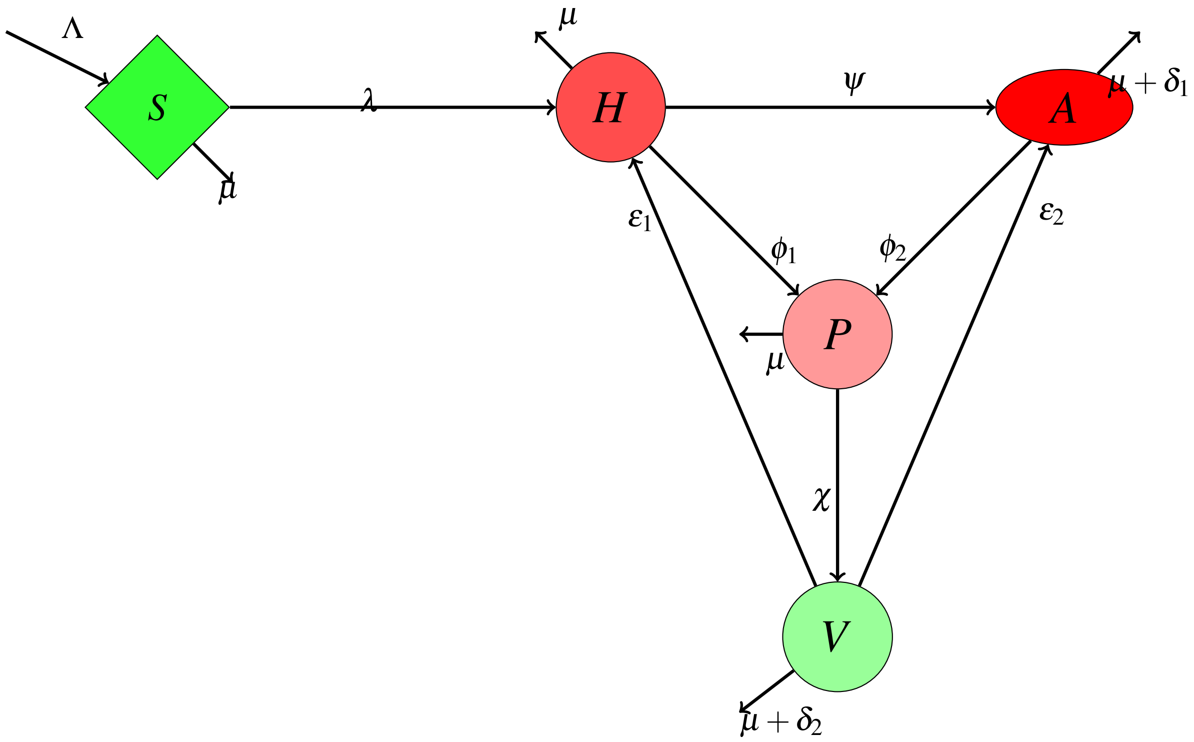


FIGURE 1. Flowchart of the HIV/AIDS Stigma Model

Therefore, the mathematical model equations are:

$$
\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta S\left(\frac{H+A+P}{N}\right)(1+m\sigma) - \mu S - \eta_1 S + \eta_2 R,\\
\frac{dH}{dt} &= \beta S\left(\frac{H+A+P}{N}\right)(1+m\sigma) - \phi_1 H\left(\frac{1}{1+k\sigma}\right) - \psi H + \varepsilon_1 V - \mu H,\\
\frac{dA}{dt} &= \psi H + \varepsilon_2 V - \phi_2 A\left(\frac{1}{1+k\sigma}\right) - (\mu+\delta_1)A,\\
\frac{dP}{dt} &= (\phi_1 H + \phi_2 A)\left(\frac{1}{1+k\sigma}\right) - \mu P - \chi P,\\
\frac{dV}{dt} &= \chi P - \varepsilon_1 V - \varepsilon_2 V - (\mu+\delta_2)V,\\
\frac{dR}{dt} &= \eta_1 S - \eta_2 R - \mu R.
\end{aligned}
\tag{1}
$$

$S(0) > 0, H(0) \geq 0, A(0) \geq 0, P(0) \geq 0, V(0) \geq 0, R(0) \geq 0$ and

$$N(t) = S(t) + H(t) + A(t) + P(t) + V(t) + R(t).$$

## 3. ANALYSIS OF THE MODEL

**3.1. Positivity of Solutions.** To demonstrate the mathematical and epidemiological significance of our model, the state variables in the model equations (1) remain non-negative for any time $t > 0$ [35][33]. This means that if the initial conditions are positive, the solution space will stay positive indefinitely.

**Theorem 3.1.** *The solution space* $(S,H,A,P,V,R)$ *of the model (1) will remain positive if initially* $S(0) > 0, H(0) \geq 0, A(0) \geq 0, \geq 0, P(0) \geq 0, V(0) \geq 0, R(0) \geq 0$.

*Proof.* From the initial conditions,

$$\begin{aligned}
\frac{dS}{dt}|_{S=0} &= \Lambda > 0 \\
\frac{dH}{dt}|_{H=0} &= \beta S\left(\frac{A}{N}\right)(1+m\sigma)+\varepsilon_1 V+\gamma A > 0 \\
\frac{dA}{dt}|_{A=0} &= \psi H+\varepsilon_2 V > 0 \\
\frac{dV}{dt}|_{V=0} &= (\phi_1 H+\phi_2 A)\left(\frac{1}{1+k\sigma}\right) > 0 \\
\frac{dP}{dt}|_{P=0} &= (\phi_1 H+\phi_2 A)\left(\frac{1}{1+k\sigma}\right) > 0, \\
\frac{dV}{dt}|_{V=0} &= \chi P > 0, \\
\frac{dR}{dt}|_{R=0} &= \eta_1 S > 0.
\end{aligned} \tag{2}$$

Therefore the solutions $(S,H,A,P,V,R)$ remain positive □

**3.2. Invariant Regions.** There is a need to show that the solutions of the model system (1) with the initial conditions in the feasible region remain in the region for $t > 0$.

**Theorem 3.2.** *The region* $\mathscr{D}$ *given by*

$$\left\{(S,H,A,P,V,R) \in R^6_+ : N \leq \frac{\Lambda}{\mu}, S(0) > 0, H(0) \geq 0, A(0) \geq 0, P(0) \geq 0, V(0) \geq 0, R(0) \geq 0\right\}$$

*with the initial conditions is positive invariant for the system (1) where* $N = S+H+A+P+V+R$

*Proof.* Without loss of generality,

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

It follows that $0 \leq N \leq \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu})e^{-\mu t}$ where $N(0)$ is initial population. Therefore, as $t \longrightarrow \infty$, $N(t) \leq \frac{\Lambda}{\mu}$ . Thus, the region is positive invariant for the system (1).

□

### 3.3. Disease Free Equilibrium $D_0$.

The disease free equilibrium is a state where there are no diseases in the population. At this point, $H = 0, \quad A = 0, \quad P = 0$ and $V = 0$. Therefore

$$D_0 = (\Lambda/\mu, 0, 0, 0, 0, 0)$$

### 3.4. Basic Reproduction number $R_0$.

The reproduction number $R_0$ can be achieved using the next generation matrix technique [36] [36] [33]. We examine the frequency of new infection emergence in compartments $H$, $A$ and $P$, from system (1) and use the next generation matrix approach to obtain

$$\mathscr{F} = \begin{bmatrix} \beta S\left(\frac{H+A+P}{N}\right)(1+m\sigma) \\ 0 \\ 0 \end{bmatrix}, \quad F = \begin{bmatrix} \beta(m\sigma+1) & \beta(m\sigma+1) & \beta(m\sigma+1) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

The Jacobian matrix of $\mathscr{F}$ at disease free equilibrium, $D_0$, is $F$

and

$$\mathscr{V} = \begin{bmatrix} \phi_1 H(1/(1+k\sigma)) + \psi H - \varepsilon_1 V + \mu H \\ -\psi H - \varepsilon_2 V + \phi_2 A(1/(1+k\sigma)) + (\mu+\delta_1)A \\ -(\phi_1 H + \phi_2 A)(1/(1+k\sigma)) + \mu P + \chi P \end{bmatrix}, \quad V = \begin{bmatrix} \mu + \frac{\phi_1}{k\sigma+1} + \psi & 0 & 0 \\ -\psi & \delta_1 + \mu + \frac{\phi_2}{k\sigma+1} & 0 \\ -\frac{\phi_1}{k\sigma+1} & -\frac{\phi_2}{k\sigma+1} & \chi+\mu \end{bmatrix}.$$

The Jacobian matrix of $\mathscr{V}$ at disease free equilibrium, $D_0$, is $V$. The eigenvalues [33] of $FV^{-1}$ are

$$\left\{ 0:2, \frac{\beta(m\sigma+1)(a_1a_2 + a_1\delta_1 + a_1\mu + a_2\chi + a_2\mu + a_2\psi + \chi\delta_1 + \chi\mu + \chi\psi + \delta_1\mu + \mu^2 + \mu\psi)}{(\chi+\mu)(a_1+\mu+\psi)(a_2+\delta_1+\mu)} : 1 \right\}$$

such that

$$R_0 = \frac{\beta(m\sigma+1)(a_1a_2 + a_1\delta_1 + a_1\mu + a_2\chi + a_2\mu + a_2\psi + \chi\delta_1 + \chi\mu + \chi\psi + \delta_1\mu + \mu^2 + \mu\psi)}{(\chi+\mu)(a_1+\mu+\psi)(a_2+\delta_1+\mu)} \tag{3}$$

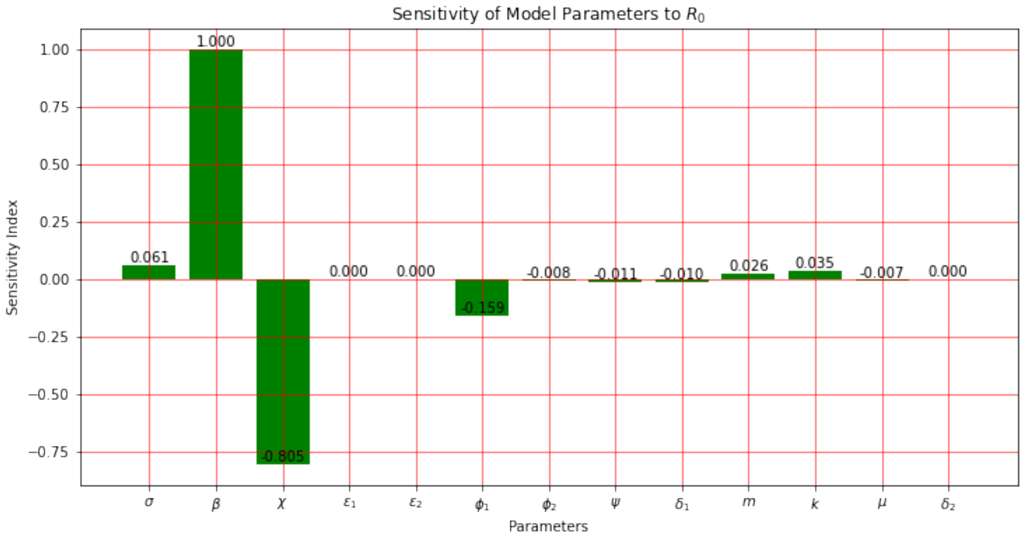
where $a_1 = \frac{\phi_1}{k\sigma+1}$ and $a_2 = \frac{\phi_2}{k\sigma+1}$.

The basic reproduction number $R_0$, indicates how readily HIV/AIDS can transmit within a population. When $R_0$ is greater than 1, it suggests that each infected individual is spreading the disease to more than one person leading to a likely increase in infections. Conversely, if $R_0$ is less than 1, it means each infected person transmits the disease to fewer than one person on average, implying that the disease will likely diminish over time. When $R_0$ equals 1, the rate of new infections remains constant, meaning the number of people contracting the disease is stable, neither increasing nor decreasing in the population.

**3.5. Sensitivity Analysis.** The concept of sensitivity may be used in infectious disease models to determine which variable or parameter is sensitive to a certain condition. There are three ways for calculating model sensitivity: full-normalization, half-normalization, and non-normalization. Based on the explicit form of $R_0$ in equation (3), the study used full-normalization for the sensitivity defined as

$$\mathbb{S}_x^{R_0} = \frac{x}{R_0} \times \frac{\partial R_0}{x} \tag{4}$$

where $x$ is the parameter.



(A) The effect of each parameter on $R_0$

FIGURE 2. Sensitivity bar chart

The sensitivity index in Figure (2) sequentially displays the parameters from the highest to the lowest sensitivity. Overall, four parameters significantly impact changes in the basic reproductive number. These parameters include the probability of transmission during contact $\beta$, the virally suppressed rate $\chi$, the rate at which HIV-positive individuals receive antiretroviral therapy $\phi_1$, and the stigma level $\sigma$.

### 3.6. Endemic Equilibrium $D_1$.

An endemic equilibrium of the model equation (1) is a stable state in which the illness remains in the population at a constant level across time. The endemic equilibrium point defined by the following:

$$D_1 = (S, H, A, P, V, R)$$

$$\begin{aligned}
S &= \frac{(\eta_2+\mu)\eta_1\Lambda}{((\eta_2+\mu)(\lambda+\mu+\eta_1)-\eta_1\eta_2)\eta_1} \\
H &= \frac{b_3e_2+\varepsilon_1\chi e_1b_4}{b_2b_4c_1e_2} \\
A &= \frac{e_2b_3\psi+\psi\varepsilon_1\chi e_1b_4+\varepsilon_2\chi b_4e_1c_1}{b_2b_4c_1c_2} \\
P &= \frac{c_2a_1b_3+a_2\psi b_3+\varepsilon_1a_2b_4c_1}{b_2b_4c_1c_2(\chi+\mu)-c_2\varepsilon_1b_4\chi-\psi\varepsilon_1\chi b_4a_2} \\
V &= \frac{\chi\,(c_2a_1b_3+a_2\psi b_3+\varepsilon_1a_2b_4c_1)}{b_2\,(b_2b_4c_1c_2(\chi+\mu)-c_2\varepsilon_1b_4\chi-\psi\varepsilon_1\chi b_4a_2)} \\
R &= \frac{\eta_1\Lambda}{(\eta_2+\mu)(\lambda+\mu+\eta_1)-\eta_1\eta_2}
\end{aligned} \tag{5}$$

where $e_1 = c_2a_1b_3+a_2\psi b_3+\varepsilon_1a_2b_4c_1$, $e_2 = b_2b_4c_1c_2(\chi+\mu)-c_2\varepsilon_1b_4\chi-\psi\varepsilon_1\chi b_4a_2$
$c_1 = a_1+\psi+\mu$, $c_2 = a_2+\mu+\delta_1$, $b_2 = \varepsilon_1+\varepsilon_2+\mu+\delta_2$, $b_3 = b_2(\eta_2+\mu)\eta_1\Lambda$
$b_4 = (b_1-\eta_1\eta_2)\eta_1$

### 3.7. Stability.

**Theorem 3.3.** *The disease free equilibrium point $D_0$ is locally asymptotically stable if $\beta(m\sigma+1) < \psi+\mu+a_1$.*

*Proof.* Evaluate the Jacobian matrix of the model equation at point $D_0$.

(6)

$$\begin{bmatrix} -\eta_1-\mu & -\beta(m\sigma+1) & -\beta(m\sigma+1) & -\beta(m\sigma+1) & 0 & \eta_2 \\ 0 & -a_1+\beta(m\sigma+1)-\mu-\psi & \beta(m\sigma+1) & \beta(m\sigma+1) & \varepsilon_1 & 0 \\ 0 & \psi & -a_2-\delta_1-\mu & 0 & \varepsilon_2 & 0 \\ 0 & a_1 & a_2 & -\chi-\mu & 0 & 0 \\ 0 & 0 & 0 & \chi & -\delta_2-\varepsilon_1-\varepsilon_2-\mu & 0 \\ \eta_1 & 0 & 0 & 0 & 0 & -\eta_2-\mu \end{bmatrix}$$

$D_0$ is locally asymptotically stable if $\beta(m\sigma+1) < \psi+\mu+a_1$ □

## 3.8. Global Stability of Equilibrium States.

**Theorem 3.4.** *The system is globally asymptotically stable at endemic equilibrium and globally stable at disease free equilibrium $E_0$.*

*Proof.* We employ the quadratic lyapunov function to prove the above theorem.

Let

$$Q = S+H+A+P+V+R$$

Consider the Lyapunov function [32],

$$2L = Q^2 \tag{7}$$

$$\begin{aligned} \frac{dL}{dt} &= Q\times\dot{Q} \\ &= (S+H+A+P+V+R)\times(\dot{S}+\dot{H}+\dot{A}+\dot{A}+\dot{P}+\dot{V}+\dot{R}) \\ &= (S+H+A+P+V+R)\times(k-\mu N-\delta_1 A-\delta_2 V). \end{aligned}$$

Clearly $0 \le N(t) \le \dfrac{k}{\mu}$ which implies $\mu N \le k$ , using $\mu N = k$

$$\begin{aligned} \frac{dL}{dt} &= Q\times\dot{Q} \\ &= (S+H+A+P+V+R)\times(\dot{S}+\dot{H}+\dot{A}+\dot{A}+\dot{P}+\dot{V}+\dot{R}) \\ &= (S+H+A+P+V+R)\times(k-\mu N-\delta_1 A-\delta_2 V) \end{aligned}$$

$$\leq (S+H+A+P+V+R) \times (-\delta_1 A - \delta_2 V)$$

$$= (-\delta_1 A - \delta_2 V)(S+H+A+P+V+R). \tag{8}$$

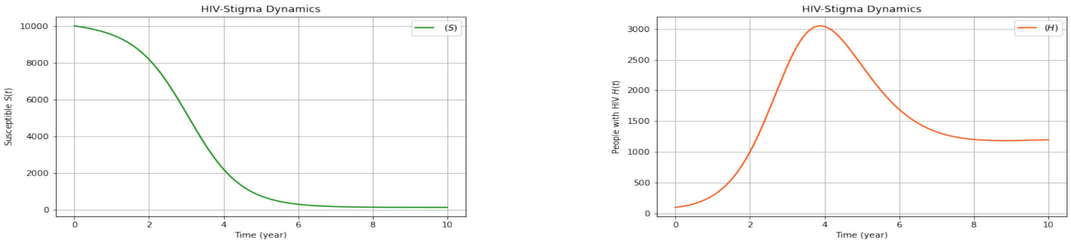
In equation (8) $\frac{dL}{dt} < 0$, the system is globally asymptotically stable at the endemic equilibrium . At the disease free equilibrium $\frac{dL}{dt} \leq 0$, the system is globally stable. $\square$

## 4. NUMERICAL SIMULATION

In this section, we approximate solutions to the model equations (1) using Python softwre with the initial values $S(0) = 10000$, $H(0) = 100$, $A(0) = 2$, $V(0) = 0$, $P(0) = 0$, $R(0) = 0$ and the parameters in the TABLE 1.

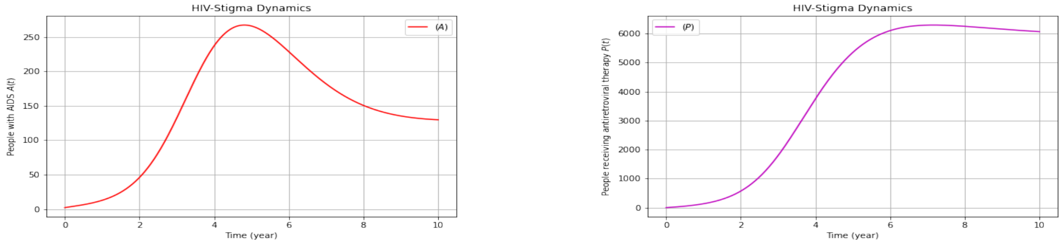
TABLE 1. : The Parameter Description Values of the Model.

| **Parameter** | **Description** | **Value** | **Source** |
|---|---|---|---|
| $\beta$ | Transmission rate | 1.5 | [37] |
| $\mu$ | Natural death rate | 0.0013532 | [37] |
| $\phi_1$ | The rate at which HIV-positive individuals receive antiretroviral therapy | 1.03 | [37] |
| $\phi_2$ | The rate at which AIDS individuals receive antiretroviral therapy | 1.09 | [37] |
| $\psi$ | Progression rate from infected to AIDS | 0.1 | [37] |
| $\varepsilon_1$ | Virally suppressed failure rate of becoming HIV-positive | 0.4 | [37] |
| $\varepsilon_2$ | Virally suppressed failure rate of becoming AIDS | 0.001 | [37] |
| $\delta_1$ | Death rate due to AIDS | 0.1 | [37] |
| $\delta_2$ | Death rate of Virally suppressed individuals | 0.0667 | [37] |
| $\chi$ | The virally suppressed rate | 0.19 | [24] |
| $\delta$ | Intervention rate | 0.50 | [37] |
| $\eta_1$ | Rate of individuals adopting protective action via education | 0.03 | [37] |
| $\eta_2$ | Rate of individuals reverting to their prior habit | 0.003 | [37] |
| $m$ | Modification factor of stigma on transmission | 0.1 | Assumed |
| $k$ | Modification factor of stigma on treatment-seeking behavior | 1 | Assumed |
| $\gamma$ | Progression rate from AIDS to infected | 0.5 | [38] |
| $\sigma$ | Stigma level | 0.2654 | [24] |



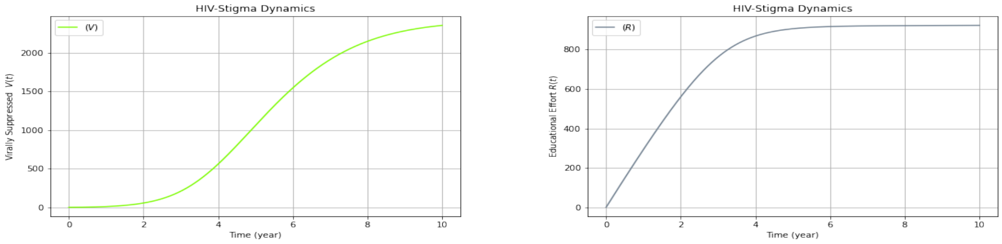
(A) Solution of Susceptible $S$

(B) Solution of HIV positive$(H)$



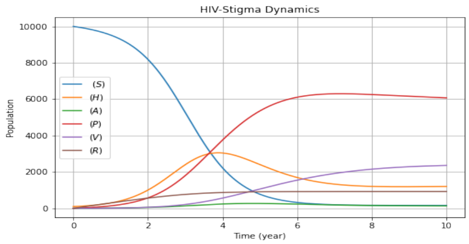
(C) Solution of AIDS population $(A)$

(D) Solution of antiretroviral therapy received population$(V)$



(E) Solution of Virally suppressed population$(V)$

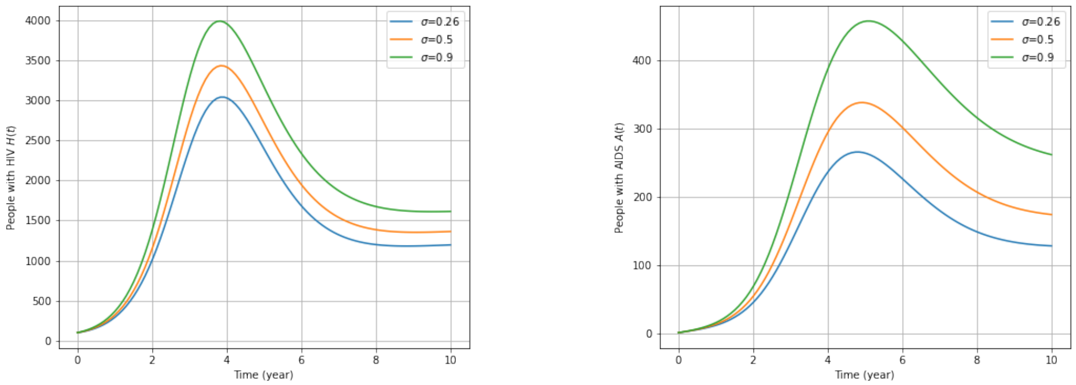
(F) Solution of educational effort on sexual behavior population$(V)$



(G) Combined solutions

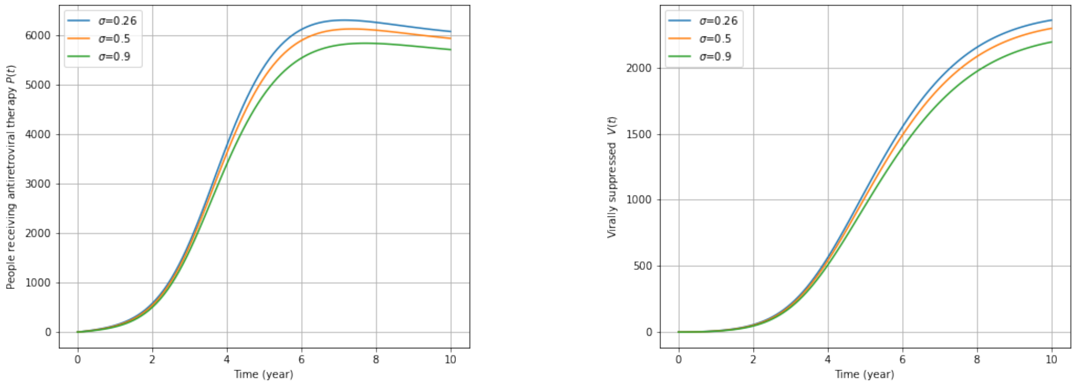
FIGURE 3. The Solution of the Model Equations

In figure (3), it shows the dynamics of model equations. Initially during the outbreak, the susceptible individuals continue to decrease due to the fact that HIV/AIDS is spreading and people are getting infected with HIV/AIDS as well as receiving ART(Anti Retroviral Therapy) in order to suppress $V$ the virus from spreading. When the stigma spread in the population, it affects the susceptible $S$, HIV positive individuals $H$, AIDS individuals $A$ and varially suppressed individuals $V$.



(A) Effects of Stigma on HIV positive$(H)$

(B) Effects of Stigma on AIDS $(A)$

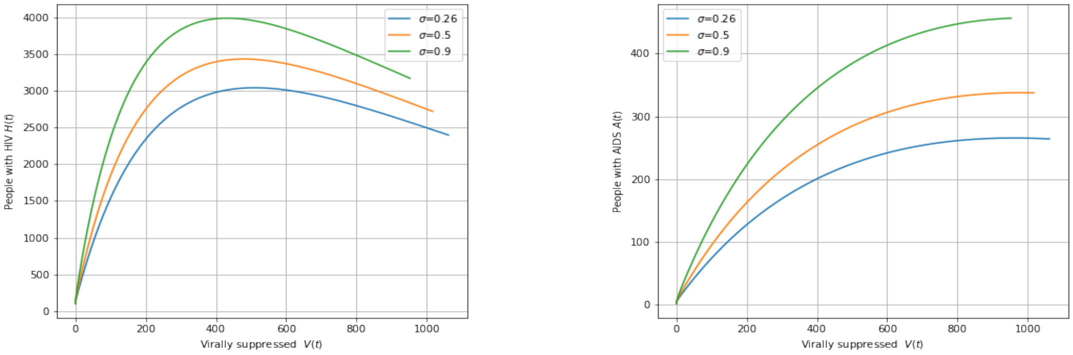


(C) Effects of stigma on people receiving antiretroviral therapy $(P)$

(D) Effects of Stigma on Virally suppressed population$(V)$

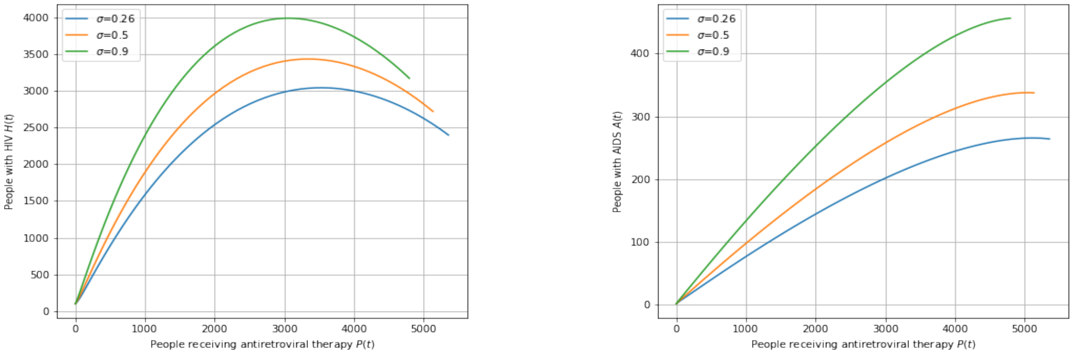
FIGURE 4

Figure (4 A) shows that high stigma $(\sigma = 0.9)$ often results in a more rapid increase of the HIV-positive population reflecting the detrimental impact of high stigma which discourages treatment and allows the disease to spread more freely. Figure (4 B) depicts that higher stigma $(\sigma = 0.9)$ results in a rapid increase in AIDS $A(t)$ due to reduced treatment uptake and faster disease progression. In Figure (4 C), as stigma increases $(\sigma = 0.9)$, people receiving antiretroviral therapy $(P)$ might grow more slowly or even remain low, reflecting the reduced number of people accessing treatment due to stigma-related barriers. In Figure (4 D), lower stigma encourages people to access antiretroviral therapy, leading to higher rates of viral suppression. However, reducing the spread of stigma is crucial to improving health outcomes and controlling the spread of HIV/AIDS.



(A) Effects of Stigma on $V$ vs $H$

(B) Effects of Stigma on $V$ vs $A$



(C) Effects of Stigma on $P$ vs $H$

(D) Effects of Stigma on $P$ vs $A$

FIGURE 5

In Figure (5 A), lower stigma $(\sigma = 0.26)$ indicates that more people living with HIV are receiving and adhering to treatment. Higher stigma $(\sigma = 0.9)$ reduces treatment uptake, resulting in a lower proportion of HIV positive $H(t)$ individuals achieving viral suppression. In Figure (5 B), higher stigma $(\sigma = 0.9)$ reduces the number of virally suppressed individuals, leading to a higher number in the advanced stage AIDS (A(t)). In Figure (5 C), high stigma acts as a barrier to accessing therapy, resulting in fewer treated individuals relative to the number of people living with HIV. In Figure (5 D), higher stigma reduces the number of people on therapy, leading to an increase in the advanced stage population AIDS $(A(t))$. Stigma reduces treatment uptake and viral suppression, leading to worse outcomes in terms of higher numbers in the advanced stages of HIV/AIDS and a lower proportion of virally suppressed individuals.

## 5. CONCLUSION

This shows the impact of HIV stigma on disease dynamics by modeling stigma as a social pathogen. We used a modified compartmental model to look at the interaction between stigma, transmission rates and treatment seeking. The results show that higher stigma leads to more HIV infections and faster progression to AIDS due to lower treatment uptake that suppressed the virus. However, effective stigma reduction interventions can reduce stigma and improve health outcomes for PLWHA. Stigma is the key to the HIV/AIDS epidemic. Treating stigma as a social pathogen, we can understand how it spreads and develop interventions to mitigate the harm. Public health strategies that focus on education, awareness campaigns and supportive policies are key to combating stigma and treatment adherence. Reducing stigma benefits PLWHA and overall public health by reducing transmission and improving treatment outcomes.

## CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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