



Algerian Democratic and Popular Republic
Ministry of Higher Education and Scientific
Research

Laarbi Tébessi University-Tébessa
Faculty of Exact Natural and Life Sciences
Department of Mathematics and Computer Science



**End-of-study Dissertation for a *MASTER* Degree in
Domain: Mathematics and Computer Science
Track: Mathematics
Option: Partial Derivatives and Applications**

The Volterra-Lyapunov for global stability analysis of a model of Reaction-Diffusion system

Presented By:

Chaima Bouchiba

Amel Hadji

The Examination Committee:

Mr. Nouri Boumaza	MCA University Laarbi Tébessi President
Mr. Khalifa Bouaziz	MCB University Laarbi Tébessi Examiner
Mr. Salem Abdelmalek	MCA University Laarbi Tébessi Supervisor
Mr. Mohammed Amroune	MCA University Laarbi Tébessi Invited

Defense Date: June 2020

Abstract

The aim of this work is to study the problem of **global asymptotic stability** for equilibria of a spatially diffusive HIV/AIDS epidemic model with homogeneous Neumann boundary condition. By discretizing the model with respect to the space variable, we first then by incorporating the theory of stable matrix of **Volterra-Lyapunov** into the classical method of the Lyapunov functional ODEs model, and then broaden the construction method into the PDEs model in which either susceptible or infective populations are diffusive. In both cases, we obtain the standard threshold dynamical behaviors, that is, if $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable and if $R_0 > 1$, then the (strictly positive) endemic equilibrium is globally asymptotically stable.

المخلص

إن الهدف من هذا العمل هو دراسة مسألة الاستقرار المقارب العالمي لنموذج وبائي لفيروس نقص المناعة البشرية / الإيدز المنتشر مكانياً مع شروط نيومان المتجانسة. من خلال تفكيك النموذج فيما يتعلق بالمتغير المكاني, نقوم أولاً بدمج نظرية المصفوفة المستقرة لفولتيرا-ليابونوف في الطريقة الكلاسيكية لدالة ليابونوف لنموذج ODES, ثم توسيع طريقة البناء في نموذج PDES, حيث السكان إما عرضة للعدوى أو السكان المعدية منتشرون. في كلتا الحالتين, نحصل على السلوكيات الديناميكية القياسية العتبية, أي إذا كان $R_0 < 1$, فإن التوازن الخالي من الأمراض يكون مستقرًا عالميًا بشكل مقارب وإذا كان $R_0 > 1$, فإن التوازن المستوطن (موجب تمامًا) كذلك مستقر عالميًا بشكل مقارب.

Résumé

Le but de ce travail est d'étudier le problème de la **stabilité asymptotique globale** pour les équilibres d'un modèle épidémique de VIH / SIDA spatialement diffusif avec une condition aux limites de Neumann homogène. En discrétisant le modèle par rapport à la variable spatiale, nous incorporons d'abord la théorie de la matrice stable de **Volterra-Lyapunov** dans la méthode classique du modèle ODE fonctionnel de Lyapunov, puis élargissons la méthode de construction dans le modèle EDPs dans lequel soit sensible ou les populations infectieuses sont diffusives. Dans les deux cas, nous obtenons les comportements dynamiques de seuil standard, c'est-à-dire que si $R_0 < 1$, alors l'équilibre sans maladie est globalement asymptotiquement stable et si $R_0 > 1$, alors l'équilibre endémique (strictement positif) est aussi globalement asymptotiquement stable.

24th September 2020

CONTENTS

0.1 Notations	3
0.2 General Notions	5
1 Reaction-diffusion systems and stability theory	8
1.1 Reaction-diffusion systems	9
1.1.1 Introduction to Reaction-Diffusion Systems	9
1.1.2 Reaction-diffusion model	10
1.1.3 Reaction-diffusion for HIV/AIDS model	12
1.2 Stability of Reaction-diffusion systems	13
1.2.1 Local stability of Reaction-diffusion systems	13
1.2.2 Global stability of Reaction-diffusion systems	15
2 Stability of the HIV/AIDS model	19
2.1 Positivity and boundedness of solutions	21
2.2 Existence the equilibriums points	22
2.2.1 Existence of disease-free equilibrium	22
2.2.2 Existence of endemic equilibrium	23
2.3 Local stability of the equilibriums points of the HIV/AIDS model	24
2.3.1 Local stability of the disease-free equilibrium(DFE)	24
2.3.2 Local stability of endemic equilibrium.	26
2.4 Global stability of the equilibriums points of the HIV/AIDS model	27
2.4.1 Global stability of the disease-free equilibrium(DFE)	27
2.4.2 Global stability of the endemic equilibrium of the HIV/AIDS model	
for $\alpha = 0$	29

3	Reaction-Diffusion system(R-D / PDE) of the model HIV/AIDS	40
3.1	Local stability of the equilibriums points of the model HIV/AIDS	41
3.1.1	Local Stability of disease-free equilibrium(DEF) of the model HIV/AIDS	42
3.1.2	Local stability of endemic equilibrium of the model HIV/AIDS . . .	45
3.2	Global stability of systeme reaction-diffusion	47
3.2.1	Global stability of disease-free equilibrium(DFE) of the model HIV/AIDS	47
3.2.2	Global stabilty of the endemic equilibrium	51

Dedication

- We devote this modest work to all, who from near and far have given us their moral and physical support for the realization of this work.
- To our parents for their support during all studies and who never cease to lavish us with their love.
- To every teacher sacrificed for us to reach this level.
- To all our brothers and sisters who were exemplary in our lives.
- To all our professors at Sheikh Larbi tebessi University, tebesse.
- To all of our colleagues within the Mathematics & Informatics department.

Acknowledgment

In the name of Allah, Most Gracious, Most Merciful, to Whom all praise is due.
The work presented in this thesis has been carried out at the University of Cheikh
LAARBI TEBESSI, in the Institute for Exact Sciences & Sciences of Nature and Life.

Departement of Mathematics & Informatics.

At first, we would like to thank all of our teachers for having given us the necessary knowledge in our educational journey.

As we would like to extend special thanks to our supervisor, Dr. Salem ABDEL-MALEK, and for his tremendous efforts in accompanying us to accomplish this work during these months.

We also wish to thank our colleagues and friends from the University and all of those who are in our hearts, especially our dear parents, for their sacrifices, kindness and never-ending support.

Introduction

HIV (Human Immunodeficiency Virus) and AIDS (Acquired Immune Deficiency Syndrome) is one of the health problems. HIV is now the major cause of years of potential lives lost and the most common cause of death attributed to many infectious diseases. Mathematical modeling over the years has been useful in analyzing various disease dynamics, such as HIV/AIDS, malaria and tuberculosis and also plays an important role in the better understanding of epidemiological patterns for disease control. In modeling frameworks, scientists presented several different models of the HIV/AIDS virus [25, 26, 32]. Marsudi et al [27] studied the impact of educational campaign, screening and HIV therapy on the dynamics of spread of HIV model. Diffusive partial differential equations are important and modern tools used to modeling natural phenomena, particularly in epidemiology, diffusive partial differential equation (PDE) models are frequently used to study the continuous spatiotemporal spread of disease among population, in this work we will the model we consider developed from [8] by including a diffusion term.

This work is divided into three chapters:

- Chapter 1: Reaction-diffusion systems and stability theory

In this chapter, we present an introduction to reaction-diffusion system and applying them in the diffusion of infection in the population, including the diffusion of HIV/AIDS virus, and we also talked about the stability of systems for reaction-diffusion and we focused on stability using Volterra-Lyapunov matrix theory.

- Chapter 2: Stability of EDO system

In this chapter, we analyze a model consisting of ordinary differential equation that describes the dynamics of HIV/AIDS, we will achieve local stability of the equilibrium points, we apply the method of Lyapunov function combined with the Volterra-Lyapunov matrix population which lead to proof the global stability.

- Chapter 3: Stability of PDE system

This is the important chapter, where we study the spatial spread of HIV/AIDS, that is, we analyze of model consisting of partial differential equation in the PDE model, and we studied local and global stability with respect to constant equilibrium points for this model.

Preliminaries

In this chapter, we will present some mathematical notation and some definitions and theories that we will need in the memoire.

This chapter recalls some useful preliminaries that are necessary for the dissertation at hand. We introduce some basic notations and notions.

0.1 Notations

The following are some notations we are used in the memorie.

- The set of the real numbers, is denoted by \mathbb{R} .
- The set of the real numbers of the n-elements, is denoted by \mathbb{R}^n .
- Matrices or variables are denoted by capital characters, e.g $P, D, A...$ etc
- The determinant of real and complex martices, is denoted by $\det(A)$.
- The trace of real and complex matrices, is denoted by $tr(A)$.
- The invers of real and complex matrices, is denoted by A^{-1} .
- The transpose of matrix A , is denoted by A^T .
- The diagonal of real and complex matrices, is denoted by $diag(A)$.
- The real part of a complex number, is denoted by $Re(A)$.
- The space of continuous and derivative functions, is denoted by C^1 .
- The Sobolev spaces, is denoted by $\mathbb{H}^1(\Omega)$.
- The spectral radius of A , is denoted by $\rho(A)$.
- The derived from the variable A with respect to time t , is denoted by $\frac{dx}{dt}$.
- The identity operator, is denoted by I .
- The norme euclidean of A , is denoted by $\|A\|$.
- The Laplacian operator A , is denoted by ΔA , where

$$\Delta A = \sum_{i=1}^n \frac{\partial^2 A}{\partial A_i^2}. \tag{1}$$

- The gradient A , is denoted by ∇A , where

$$\text{Grad } A = \nabla A = \left(\frac{\partial A}{\partial x_1}, \frac{\partial A}{\partial x_2}, \dots, \frac{\partial A}{\partial x_n} \right). \quad (2)$$

- For any $n \times n$ matrix A , let \tilde{A} denote the $(n-1) \times (n-1)$ matrix obtained from A by deleting its last row and last column.
- The set of positive real numbers, is denoted by $\mathbb{R}_+ = [0, +\infty)$.
- U^{**} : Non-constant equilibrium point.
- U^* : Constant equilibrium point.
- The disease-free equilibrium, is denoted by **(DFE)**.
- The ordinary differential equations, is denoted by **(EDO)**.
- The partial differential equations, is denoted by **(PDE)**.
- Ω : open domain in \mathbb{R}^n , where $n > 1$.

0.2 General Notions

The following are some general notions we are used in the memorie.

Definition 1 (*equilibrium point*) We say that X^E an equilibrium point of a system

$$\begin{cases} \frac{dX(t)}{dt} = f(X(t)), \\ X(0) = X_0, \end{cases} \quad (3)$$

if X^E verify the equation

$$f(X^E) = 0. \quad (4)$$

Definition 2 We denote by $\mathbb{L}^2(\Omega)$ the set of integrable square functions on Ω . A function f defined on Ω is called an integrable square if f is measurable. We then define the norme on

$$\|f\|_{\mathbb{L}^2(\Omega)} = \left(\int_{\Omega} |f|^2 \right)^{\frac{1}{2}}. \quad (5)$$

Definition 3 [31] The equilibrium X^E is said to be stable if for everything $\epsilon > 0$, it exists $\eta > 0$, as for all solution $X(t)$ of (3), we have

$$\|X(0) - X^E\| < \eta \implies \|X(t) - X^E\| < \epsilon. \quad (6)$$

Definition 4 [31] (Locally asymptotically stable) Let $J(X^E) = \frac{\partial f}{\partial X}(X^E)$, the Jacobian matrix of f evaluates at point X^E . Consider the following linear system

$$\frac{dX}{dt} = AX, \quad (7)$$

where $A = J(X^E)$ is say the linearized or the linear approximation of the non-linear system (3) in X^E .

The study of the stability of the origin for the linearized allows in certain cases to characterize the stability of the (3). More precisely, we have,

- If all the eigenvalues of the matrix A are of strictly negative real part, then the system (3) is stable.
- If there is at least one eigenvalue of the matrix A of strictly positive real part then, the system (3) is unstable.

Definition 5 [31] (Globally asymptotically stable) The equilibrium point X^E is say to be globally asymptotically stable if it is stable, and for any $X(t)$ solution for (3), we have

$$\lim_{t \rightarrow \infty} \|X(t) - X^E\| = 0. \quad (8)$$

Definition 6 [13] The basic reproduction number R is the spectral radius of the next generation matrix, namely

$$R = \rho(FV^{-1}). \quad (9)$$

The following interpretation is given to the matrix FV^{-1} : Let us consider an infected individual introduced into a compartment FV^{-1} of a population without disease. The entry (i, k) of the matrix V^{-1} is the average time that the individual will spend in compartment i during his life, assuming that the infection has been blocked. The entry (j, i) of matrix F is the speed at which an infected person in compartment i produces infections in compartment j . Thus the entry (j, k) of FV^{-1} is the expected number of new infections in compartment j produced by an infected individual originally introduced into compartment k . The spectral radius of the matrix FV^{-1} is the basic reproduction number. That is to say $R = \rho(FV^{-1})$.

Lemma 7 [30] For all function u of $\mathbb{H}^1(\Omega)$, and all function v of $\mathbb{H}^1(\Omega)$, we have the Green formula

$$\int_{\Omega} (\Delta u) v = \int_{\Omega} \frac{\partial u}{\partial \eta} v d\sigma - \int_{\Omega} \nabla u \nabla v. \quad (10)$$

Proof 8 On suppose $\Delta u = \sum_{i=1}^n \frac{\partial^2 u}{\partial x_i^2}$, the Laplacian of a distribution u . Then, if u is a function of $\mathbb{H}^1(\Omega)$, for all function v of $\mathbb{H}^1(\Omega)$

$$\begin{aligned} - \int_{\Omega} (\Delta u) v &= - \sum_{i=1}^n \int_{\Omega} \frac{\partial^2 u}{\partial x_i^2} v dx, \\ &= \sum_{i=1}^n \left\{ \int_{\Omega} \frac{\partial u}{\partial x_i} \frac{\partial v}{\partial x_i} - \int_{\Omega} \frac{\partial u}{\partial x_i} v \eta_i d\sigma \right\}, \\ &= \sum_{i=1}^n \int_{\Omega} \frac{\partial u}{\partial x_i} \frac{\partial v}{\partial x_i} - \int_{\Omega} \frac{\partial u}{\partial \eta} v d\sigma, \\ &= \int_{\Omega} \nabla u \nabla v - \int_{\partial \Omega} \frac{\partial u}{\partial \eta} v d\sigma. \end{aligned} \quad (11)$$

Theorem 9 [10] (Routh-Hurwitz)

Consider the characteristic equation

$$\det(\lambda I_n - P) = \lambda^n + a_1 \lambda^{n-1} + a_2 \lambda^{n-2} + \dots + a_{n-1} \lambda + a_n,$$

determining the n eigenvalues λ of a real $n \times n$ square matrix P , where I is the identity matrix.

Then the eigenvalues λ all have negative real parts if

$$\Delta_1 > 0, \Delta_2 > 0, \dots, \Delta_k > 0, \dots, \Delta_n > 0, \quad (12)$$

where

$$\Delta_1 = a_1, \Delta_2 = \begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix} = a_1 a_2 - a_3, \quad (13)$$

and

$$\Delta_k = \begin{vmatrix} a_1 & 1 & 0 & 0 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ a_{2k-1} & a_{2k-2} & a_{2k-3} & a_{2k-4} & a_{2k-5} & a_{2k-6} & \cdots & a_k \end{vmatrix}. \quad (14)$$

CHAPTER 1

Reaction-diffusion systems and stability theory

In this chapter, we provide an introduction to the systems for spreading the reaction, and the extent of their application in all fields, especially in the spread of infection (diseases), and we have dedicated to this the spread of HIV/AIDS. We also talked about the stability of these systems and we have devoted to global stability for stability according to Volterra-Lyapunov

1.1 Reaction-diffusion systems

1.1.1 Introduction to Reaction-Diffusion Systems

Due to the extreme importance of reaction–diffusion systems of partial differential equations in modeling real–life applications [29], it in various are well-established in different life science disciplines, they have been attracting the interest of scientists and academics for decades, and has been the subject of much research, especially in the last twenty years. Reaction–diffusion systems are mathematical models which correspond to several physical phenomena. The most common is the change in space and time of the concentration of one or more chemical substances, or infection spread among the population, and diffusion which causes the substances to spread out over a surface in space.

Population dynamic has attracted the interest of many authors in the past as nowadays reaction-diffusion equations are widely used as models for spatial effects in ecology. They support three important types of ecological phenomena: the existence of a minimal patch size necessary to sustain a population, the propagation of wave fronts corresponding to biological invasions, and the formation of spatial patterns in the distributions of populations in homogeneous environments. Reaction-diffusion equations can be, analyzed by means of methods from the theory of partial differential equations and dynamical systems. Reaction–diffusion equations arise as models for the densities of substances or organisms that disperse through space by brownian motion, random walks, hydrodynamic turbulence, or similar mechanisms, and that react to each other and their surroundings in ways that affect their local densities [16]. Reaction–diffusion models are in themselves deterministic, but they can be derived as limits of stochastic processes under suitable scaling. Specifically, they provide a modeling approach that allows us to translate assumptions about stochastic local movement into deterministic descriptions of global densities. Reaction-diffusion models are spatially explicit, describe population densities, and treat space and time as continuous.

Diffusion [17]:

The concept of diffusion originates from physical sciences (Fick’s law is regarded as the fundamental principle of diffusion). In its physical sense diffusion is defined as a phenomenon where a certain particle group as a whole spreads according to the irregular motion of each particle. There by the spread is always directed from regions of higher concentration to regions of lower concentration and the time dependence of the distribution of the particles in space is given by the so called diffusion equation which is the mathematical formulation of the described spread dynamic. The diffusion theory seeks to explain the spread behaviour of a group of particles (rather than spread behaviour of a

single particle) and consequently the variable of interest is the proportion of the particle group which can be found. In this way phenomena like the diffusion of an ink drop in water or diffusion of heat can be described, of diffusion is applied in biology to describe processes of biodiffusion and to model population dynamics, or the spread of infectious diseases among the population, or in a less quantitative way, in social sciences to describe the spread of ideas (diffusion of innovations, lexical diffusion, trans-cultural diffusion).

Reaction [17]:

Based on our gained insights from the theory of the random spread of infection among the population we now develop the general diffusion theory. We are especially interested in deriving spatial distribution results of infection between the dispersing population. In other words we assume a population with a sufficient high number of individuals situated and are interested in the spatial distribution of this population as time progresses. That means we determine evolution of infection of between individuals at assuming that the entire population coexists with each other without taking preventive measures. Here we carry out the transition from discrete considerations in time and space to continuous considerations in time and space.

In mathematical population biology, qualitative results for models are the most important kind, for accurate quantitative results can only occasionally even be expected. The main reason is that models here are much more idealized than in physics and chemistry. Populations are never homogeneous, the environment is never uniform in time or space, and the population is never isolated from other influences. What one can often hope for, however, is some indication as to the effect, with in the total picture, of the few factors and influences specifically being accounted for in the model. Other factors will also leave their marks on the behavior of actual populations.

1.1.2 Reaction-diffusion model

After considering different approaches for describing reproduction and dispersion, whether for infection, prey or population separately we now study the population dynamic in [29] obtained by combining both mechanisms. We allow the population or infection to grow and to disperse at the same time and are interested in the temporal and spatial behaviour of the population size under different growth models (exponential growth, logistic growth,...). We analyse so called diffusion-reaction systems we assume Neumann bound-

ary conditions of the form:

$$\left\{ \begin{array}{l} \frac{\partial U(x,t)}{\partial t} = D\Delta U(x,t) + F(U(x,t)) \quad \text{for all } x \in \Omega, t > 0, \\ \frac{\partial U(x,t)}{\partial \eta} = 0 \quad \text{on } \mathbb{R}_+ \times \partial\Omega, \\ U(x,0) = U_0(x) \quad x \in \Omega. \end{array} \right. \quad (1.1)$$

Where $U(x,t)$ is an m -vector the time and space dependent function $U(x,t)$ again describes the population size at any location x and time t . The mathematical symbol Δ defines the Laplacian operator, which is the mathematical description of the process of moving the infection or population from local spatial regions of high density or the most common infection to those of a lower density or the least infection, D = diagonal matrix (d_1, d_2, \dots, d_m) , the temporal change of the population or infection size at location x is given by the diffusion component $D\Delta U(x,t)$, and the growth component $F = (f_1, f_2, \dots, f_m)$.

For the reaction of water, for example, we take in



The equations describing this reaction are then written according to

$$\left\{ \begin{array}{l} \frac{\partial [H_2]}{\partial t} - d_1\Delta [H_2] = 2(-h[H_2]^2[O_2] + l[H_2O]^2), \\ \frac{\partial [O_2]}{\partial t} - d_2\Delta [O_2] = (-h[H_2]^2[O_2] + l[H_2O]^2) \quad x \in \Omega, t > 0, \\ \frac{\partial [H_2O]}{\partial t} - d_3\Delta [H_2O] = 2(h[H_2]^2[O_2] - l[H_2O]^2), \end{array} \right. \quad (1.3)$$

with boundary conditions (for exemple $\frac{\partial [H_2]}{\partial t} = \frac{\partial [O_2]}{\partial t} = \frac{\partial [H_2O]}{\partial t} = 0, t > 0, x \in \partial\Omega$) and positive initial conditions i.e

$$[H_2]_{t=0} = [H_2]_0 > 0, [O_2]_{t=0} = [O_2]_0 > 0, [H_2O]_{t=0} = [H_2O]_0 > 0.$$

The coefficients h and l are assumed to be positive constants, although they may depend on the temperature:

$$h, l \approx cT^\beta \exp\left(\frac{E}{R}T\right), \quad 1 \leq \beta \leq 2. \quad (1.4)$$

1.1.3 Reaction-diffusion for HIV/AIDS model

AIDS is an infectious disease that suppresses the normal function of the immune system, it is caused by the human immunodeficiency virus (HIV), which destroys the body's ability to fight infections. In order to reduce the risk of transmission to future generations, patients with HIV can accept treatment before becoming AIDS patients. Their results showed that 40% of all HIV/AIDS cases result from mother to child transmission and fewer than 300 infants in the U.S acquired HIV through vertical transmission in 1997. In sub-saharan africa over 2.5 million children under the age of 15 died of AIDS. In the context of modeling, the authors suggested several models for HIV/AIDS virus. We will take the suggested form a side [8] (use of condom, screening of unaware infectives and treatment of infectives). The challenge posed by the number of cases of unaware infectives calls for urgent need for a better understanding of the important parameters in the disease transmission, and to develop an effective and optimal strategies for prevention and control of the spread of HIV/AIDS disease.

$$\left\{ \begin{array}{l} \frac{dS(x, t)}{dt} - a\Delta S(x, t) = Q_0 - \beta_m S(x, t) - \mu S(x, t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dI_1(x, t)}{dt} - b\Delta I_1(x, t) = \beta_m S - (\theta + \delta + \mu) I_1(x, t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dI_2(x, t)}{dt} - c\Delta I_2(x, t) = \theta I_1(x, t) - (\theta + \delta + \pi) I_2(x, t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dA(x, t)}{dt} - d\Delta A(x, t) = \delta I_1(x, t) + (\delta + \pi) I_2(x, t) - \mu A(x, t) \quad \text{in } \mathbb{R}_+ \times \Omega. \end{array} \right. \quad (1.5)$$

Where Ω is an open bounded subset of \mathbb{R}^n with piecewise smooth boundary $\partial\Omega$.

Subject to the homogeneous Neumann boundary condition $\frac{\partial S}{\partial \eta} = \frac{\partial I_1}{\partial \eta} = \frac{\partial I_2}{\partial \eta} = \frac{\partial A}{\partial \eta} = 0$, for all $x \in \partial\Omega$, and positive initial conditions i.e

$$\begin{aligned} S(x, 0) &= S_0(x) > 0, \quad I_1(x, 0) = I_{1_0}(x) > 0, \\ I_2(x, 0) &= I_{2_0}(x) > 0, \quad \text{and } A(x, 0) = A_0(x) > 0, \quad \text{where } x \in \Omega. \end{aligned}$$

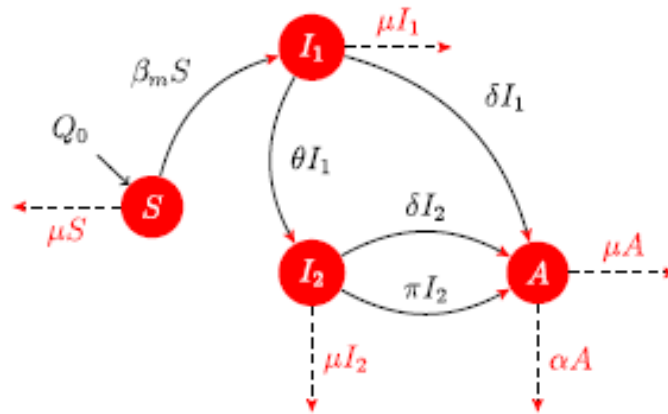


Diagram 1. Flow diagram for HIV/AIDS disease transmission model.

1.2 Stability of Reaction-diffusion systems

The study of the endemic global stability is not only mathematically important, but also essential in predicting the evolution of the disease in the long run so that prevention and intervention strategies can be effectively designed, and public health administrative efforts can be properly scaled. There are some methods, i.e. those based on the monotone dynamical systems (1.1), the geometric approach [20], and Lyapunov functions, [21] to conduct global stability analysis for epidemic models.

Under certain assumptions one might expect that the solution to (1.1) would approach as $t \rightarrow \infty$ to a solution of the system we have:

$$\begin{cases} D\Delta U^{**}(x, t) + F(U^{**}(x, t)) = 0 & x \in \Omega, t > 0, \\ \frac{\partial U^{**}(x, t)}{\partial \eta} = 0 & \text{on } \mathbb{R}_+ \times \partial\Omega, \\ U^{**}(x, 0) = U_0^{**}(x), & x \in \Omega. \end{cases} \quad (1.6)$$

Solutions of (1.6) with the Neumann boundary condition are called non-constant equilibriums solutions.

1.2.1 Local stability of Reaction-diffusion systems

In order to study the local asymptotic stability in the PDE sens [29], one of the most commonly used methods is that of eigenfunction expansion, you we have found that the linear stability analysis of continuous field models isn't as easy as that of non-spatial models.

For the latter, we have a very convenient tool called the Jacobian matrices of system, it is as follows:

Suppose $f_j : \mathbb{R}^m \longrightarrow \mathbb{R}^m$, for $j = 1, \dots, m$, is a function such that each of its first-order partial derivatives exist on \mathbb{R}^m . This function takes a point $x \in \mathbb{R}^m$ as input and produces the vector $f_j(x) \in \mathbb{R}^m$ as output. Then the Jacobian matrix of f_j in the system (1.1) is defined to be an $m \times m$ matrix, denoted by J_{f_j} , whose (m, m) th entry is

$$J_{f_j} = \begin{bmatrix} \frac{\partial f_1}{\partial x_1} & \dots & \frac{\partial f_1}{\partial x_m} \\ \vdots & \ddots & \vdots \\ \frac{\partial f_m}{\partial x_1} & \dots & \frac{\partial f_m}{\partial x_m} \end{bmatrix}. \quad (1.7)$$

Let $0 = \lambda_0 < \lambda_1 \leq \lambda_2 \leq \dots$ be the sequence of eigenvalues for the elliptic operator $(-\Delta)$ subject to the homogeneous Neumann boundary condition $\frac{\partial f_i}{\partial \eta} = 0$ on $\partial\Omega$, where each λ_i for $i = 0, 1, \dots$ has multiplicity $m_i \geq 1$. Also let Φ_{ij} , $1 \leq j \leq i$, (recall that $\Phi_0 = const$ and $\lambda_i \longrightarrow \infty$) be the normalized eigenfunction corresponding to λ_i .

That is, Φ_{ij} and λ_i satisfy

$$-\Delta \Phi_{ij} = \lambda_i \Phi_{ij} \text{ in } \Omega, \quad (1.8)$$

with

$$\frac{\partial \Phi_{ij}}{\partial \nu} = 0 \text{ in } \partial\Omega, \quad (1.9)$$

and

$$\int_{\Omega} \Phi_{ij}^2(x) dx = 1. \quad (1.10)$$

Similar to the **ODE** case, **the asymptotic stability** of the steady state solution $u^*(x, t)$ in the (1.1) can be determined by examining the eigenvalue ξ of the operator J_{f_j} . That is the solution is **asymptotically stable** if all the eigenvalues of J_{f_j} have negative real parts. In order to achieve that, suppose $\psi = (\psi_1, \psi_2, \dots, \psi_n)$ is an eigenfunction of J_{f_j} corresponding to an eigenvalue ξ . By definition [11], we have

$$J_{f_j} \psi^t = \xi \psi^t, \quad (1.11)$$

where

$$(J_{f_j} - \xi I_n) \psi^t = 0, \quad (1.12)$$

this can be rearranged to the form

$$\sum_{0 \leq i \leq \infty, 1 \leq j \leq m_i} (P_i - \xi I_n) \begin{bmatrix} k_{1ij} \\ k_{2ij} \\ \vdots \\ k_{nij} \end{bmatrix} \Phi_{ij} = 0, \quad (1.13)$$

where

$$\psi_1 = \sum_{0 \leq i \leq \infty, 1 \leq j \leq m_i} k_{1ij} \Phi_{ij}, \text{ and } \psi_2 = \sum_{0 \leq i \leq \infty, 1 \leq j \leq m_i} k_{2ij} \Phi_{ij}, \dots, \psi_n = \sum_{0 \leq i \leq \infty, 1 \leq j \leq m_i} k_{nij} \Phi_{ij}$$

The stability of the steady state now reduces to examining the eigenvalues of the matrices P_i ,

$$\det(P_i - \xi I_n) = 0,$$

the sytem (1.1) is asymptotically locally stable if

$$\operatorname{Re}(\xi) < 0. \quad (1.14)$$

If $\operatorname{Re}(\xi) \geq 0$, the sytem (1.1) is unstable.

1.2.2 Global stability of Reaction-diffusion systems

Definition 10 [29] *The $U^*(., t)$ equilibrium point constant of the system (1.1) is globally asymptotically stable, and for any U solution constant to (1.1) we have*

$$\lim_{t \rightarrow \infty} \|U^*(., t) - U\|_{\mathbb{L}^2(\Omega)} = 0. \quad (1.15)$$

Lemma 11 [1] *Let consider a disease model system written in the form*

$$\begin{cases} \frac{dX_1}{dt} = F_i(X_1, X_2) & \text{for } i = 0, 1, \dots, \\ \frac{dX_2}{dt} = G_i(X_1, X_2) & \text{for } i = 0, 1, \dots, \end{cases} \quad (1.16)$$

and

$$G_i(X_1, 0) = 0 \text{ for } i = 0, 1, \dots, \quad (1.17)$$

where $X_1 \in \mathbb{R}^m$ denotes (by its components) the uninfected populations and $X_2 \in \mathbb{R}^n$ de

notes (by its components) the infectious populations, $X_0 = (X_1^E, 0)$. denotes the DFE of the system.

Also assume the following conditions:

(C₁) For $\frac{dX_1}{dt} = F_i(X_1, 0)$ for $i = 0, 1, \dots$. X_1^E is globally asymptotically stable.

(C₂) $G_i(X_1, X_2) = A_i X_2 - \hat{G}(X_1, X_2)$, with $\hat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$ and for $i = 0, 1, \dots$

Where the jacobian matrix $A_i = \frac{\partial G_i}{\partial X_2}(X_1^E, 0)$, for $i = 0, 1, \dots$ has all non-negative off-diagonal elements, and X is the region where the model makes biological sense.

Then the DFE $X_0 = (X_1^E, 0)$ is globally asymptotically stable provided that $R_0 < 1$, for $i = 0, 1, \dots$

Stability Based on Volterra-Lyapunov Method

The construction method of Lyapunov functions and functionals is commonly used to establish global stability results for biologically structured epidemic models, the method of Lyapunov functions has been known for many decades [29]. The challenge in the application of this method is that there is no systematic way to construct Lyapunov functions (particularly, the determination of the appropriate coefficients is often a matter of luck), so that its success largely depends on trial and error as well as on specific problems. Due to the fact that these models belong to infinite dimensional dynamical systems, it is often necessary to formulate some mathematical arguments, for example, the relative compactness (or completely continuous) of the semi-flow generated from system, which ensures the existence of the global attractor and uniform persistence, which ensures the wellposedness of the Lyapunov functions for the steady states, the global asymptotic stability for various epidemic models based on patchy ODEs models with discretized spatial structure have been extensively studied, the global stability analysis of epidemic models as high-dimensional ODEs is relatively easily followed. From this reason, even if we do not know the suitable form of Lyapunov functions for a spatially diffusive epidemic model as PDEs, we can guess it by discretizing the PDEs model into a corresponding ODEs model and paying attention to the known Lyapunov function for the models ODEs

The Lyapunov function is defined as follows

Definition 12 [30] (Lyapunov functional) We say Lyapunov functional associated with a reaction-diffusion system form of m equations, any function

$$V : \mathbb{R}_+ \longrightarrow \mathbb{R}_+,$$

such as

$$\frac{d}{dt} (V (u_1(\cdot, t), u_2(\cdot, t), \dots, u_m(\cdot, t))) \leq 0, \quad (1.18)$$

for all $t > 0$ and any solution $(u_1(x, \cdot), u_2(x, \cdot), \dots, u_m(x, \cdot))$ of system (1.1).

After obtaining the matrix system when applying the Lyapunov function, we prove it is stability by we do incorporate the Volterra–Lyapunov matrix [14, 15] theory into Lyapunov functions which, under certain conditions, eliminates the need of determining the coefficients. by combining this classical approach with the Volterra–Lyapunov matrix analysis, we have leveraged the difficulty of determining specific coefficient values and, as such, wider application of Lyapunov functions to dynamical systems could be promoted, in prove to global stability.

Where we will apply the following characteristics of Volterra-Lyapunov :

Definition 13 [9] *We say a nonsingular $n \times n$ matrix P Volterra-Lyapunov stable if there exists a positive diagonal $n \times n$ matrix M such that*

$$MP + P^T M^T < 0. \quad (1.19)$$

Definition 14 [9] *We say a nonsingular $n \times n$ matrix P is diagonally stable (or positive stable) if here exists a positive diagonal $n \times n$ matrix M such that*

$$MP + P^T M^T > 0. \quad (1.20)$$

Lemma 15 [9] *Let P be an $n \times n$ real matrix. Then all the eigenvalues of P have negative (positive) real parts if and only if there exists a matrix $M > 0$ such that*

$$MP + P^T M^T < 0 (> 0). \quad (1.21)$$

Lemma 16 [9] *Let $P = \begin{bmatrix} p_{11} & p_{12} \\ p_{21} & p_{22} \end{bmatrix}$ be a 2×2 matrix. Then P is Volterra-Lyapunov stable if and only if $p_{11} < 0$, $p_{22} < 0$ and*

$$\det(P) = p_{11}p_{22} - p_{12}p_{21} > 0. \quad (1.22)$$

The characterization of Volterra-Lyapunov stable matrices of higher dimensions, however, is much more difficult.

From definition 12 and 13, it is clear that a matrix P is Volterra-Lyapunov stable if and only if its negative matrix, $-P$ is diagonally stable obtained the following sufficient and necessary conditions for 3×3 diagonally stable matrices.

Lemma 17 [9] Let $P = (p_{ij})$ be a 3×3 real matrix and $D = (d_{ij}) = \det(P)D$ be the adjoint of P , where $D = (d_{ij})$ is the inverse of P is diagonally stable if and only if all the signed principal minors of $-P$ are positive and the inequalities:

$$\begin{cases} (p_{31} + Zp_{13})^2 < 4p_{11}p_{33}Z, \\ (\hat{d}_{31} + Z\hat{b}_{13})^2 < 4\hat{d}_{11}\hat{d}_{33}Z. \end{cases} \quad (1.23)$$

Based on Lemma 16, the following generalized result was obtained by Redheffer which will be frequently used in our global stability analysis. For simplicity, we only state the sufficient condition below.

Lemma 18 [9] Let $P = [P_{ij}]$ be a nonsingular $n \times n$ matrix ($n \geq 2$) and $M = \text{diag}(m_1, \dots, m_n)$ be a positive diagonal $n \times n$ matrix. Let $E = P^{-1}$. Then, if $p_{nn} > 0$. $\widetilde{M}\widetilde{E} + (\widetilde{M}\widetilde{E})^T > 0$, and $\widetilde{M}\widetilde{P} + (\widetilde{M}\widetilde{P})^T > 0$, it is possible to choose $M_n > 0$ such that

$$MP + P^T M^T > 0. \quad (1.24)$$

Finally, we say that reaction–diffusion systems have become important in many areas of life, it in various are well-established in different life science disciplines.

CHAPTER 2

Stability of the HIV/AIDS model

As we have seen before the HIV/AIDS model, in this chapter we will study the local and global stability of the equilibriums points of this model in the case of ordinary differential equations (EDO).

In this chapter, we consider the fourth-order model of [8]. Consider a population of size $N(t)$ at time t into the following sub-populations of susceptible individuals $S(t)$, infective individuals who do not know that they are infected $I_1(t)$, HIV positive individuals who know that they are infected $I_2(t)$ and that of the **AIDS** population $A(t)$. So that

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t). \quad (2.1)$$

We study a system of differentiale quations in the following form:

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = Q_0 - \beta_m S(t) - \mu S(t) \quad t > 0, \\ \frac{dI_1(t)}{dt} = \beta_m S(t) - (\theta + \mu + \delta) I_1(t) \quad t > 0, \\ \frac{dI_2(t)}{dt} = \theta I_1(t) - (\delta + \mu + \pi) I_2(t) \quad t > 0, \\ \frac{dA(t)}{dt} = \delta I_1(t) + \delta I_2(t) + \pi I_2(t) - (\alpha + \mu) A(t) \quad t > 0, \end{array} \right. \quad (2.2)$$

where

$$\beta_m = \frac{(1 - u_1)(\beta_1 c_1 I_1 + \beta_2 c_2 I_2 + \beta_3 c_3 A)}{N}$$

where the terms c_i ($i = 1, 2, 3$) are the number of sexual partners of susceptible individuals with unaware infectives, aware infectives and the **AIDS** individuals respectively in each time period. Also, β_i ($i = 1, 2, 3$) are the probabilities for susceptible individuals with unaware infectives, infectives who are already-aware of their status and **AIDS** individuals respectively. Control $u_1 \in [0, 1]$ is the successful use of condom by susceptibles to protect themselves. The term θ measures the rate at which unaware infectives are detected by a screening method to become aware infectives, the term π measures the progression rate at which the already-aware infective individuals on treatment move to the A class in each time period. Here, δ is the rate by which both types of infectives not on treatment develop **AIDS** ($\pi < \delta$). μ is the natural mortality rate unrelated to **HIV/AIDS** disease and α is the **AIDS** related death rate. It is assumed that the rate of contact of susceptibles with **AIDS** individuals is much less than aware infectives which in turn is much less than that with unaware infectives ($\beta_3 \ll \beta_2 \ll \beta_1$). This is so because, on becoming aware of their infection, the infected persons may choose to use preventive measures and change their behavior and thus may contribute little in spreading the infection. We assume also that the A class is less sexually active. Now, we describe that all solutions of the system with

non-negative initial data will remain non-negative for all time.

2.1 Positivity and boundedness of solutions

For the HIV/AIDS transmission system (2.2) to be epidemiologically meaningful, it is important to prove that all solutions with non-negative initial data will remain non-negative for all time [8].

Theorem 19 *If $S(0), I_1(0), I_2(0)$ and $A(0)$ are non-negative, then so are $S(t), I_1(t), I_2(t)$ and $A(t)$ for all time $t > 0$. Moreover, $\limsup_{t \rightarrow \infty} N(t) \leq \frac{Q_0}{\mu}$.*

Furthermore, if $N(0) \leq \frac{Q_0}{\mu}$, then $N(t) \leq \frac{Q_0}{\mu}$.

Proof 20 *Let $(S(t), I_1(t), I_2(t))$ and $A(t)$ be any solution with positive initial conditions [9]. We have*

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t),$$

the time derivative of $N(t)$ along the solution of (1.1) is

$$\begin{aligned} \frac{dN(t)}{dt} &= \frac{dS(t)}{dt} + \frac{dI_1(t)}{dt} + \frac{dI_2(t)}{dt} + \frac{dA(t)}{dt}, \\ \frac{dN(t)}{dt} &\leq Q_0 - \mu N(t). \end{aligned}$$

Using theory of differential equations, we get:

homogene solution:

$$\frac{dN(t)}{dt} = -\mu N(t), \text{ therefore } N(t) = N_0 e^{-\mu t},$$

non-homogene solution:

$$\frac{dN(t)}{dt} = \left(\frac{dN_0(t)}{dt} - \mu N_0(t) \right) e^{-\mu t}, \text{ hence } N(t) \leq \frac{Q_0}{\mu} (1 - e^{-\mu t}) + N_0 e^{-\mu t},$$

and for $t \rightarrow \infty$, we have

$$\overline{\lim}_{t \rightarrow \infty} N(t) = \limsup_{t \rightarrow \infty} N(t) \leq \frac{Q_0}{\mu}. \quad (2.3)$$

Clearly, it has been proved that all the solutions of (2.2) which initiate in \mathbb{R}_+^4 confined in the region

$$D = \left\{ (S, I_1, I_2, A) \in \mathbb{R}_+^4 : S + I_1 + I_2 + A \leq \frac{Q_0}{\mu} \right\}. \quad (2.4)$$

(i.e) solution are bounded in the interval $[0, \infty)$.

2.2 Existence the equilibriums points

In the section we calculation the quilibriums pionts of model (2.2).

Solving the HIV/AIDS model equation in-terms of β_m , we calculate the equilibrium piont and obtain:

$$Q_0 - \beta_m S - \mu S = 0, \quad (2.5)$$

$$\beta_m S - (\theta + \mu + \delta) I_1 = 0, \quad (2.6)$$

$$\theta I_1 - (\delta + \mu + \pi) I_2 = 0, \quad (2.7)$$

$$\delta I_1 + \delta I_2 + \pi I_2 - (\alpha + \mu) A = 0. \quad (2.8)$$

2.2.1 Existence of disease-free equilibrium

The **disease-free equilibrium (DFE)** of the **HIV/AIDS** model (2.2) existe only when $I_1 = I_2 = A = 0$, it is given by

$$E_0 = \left(\frac{Q_0}{\mu}, 0, 0, 0 \right). \quad (2.9)$$

The basic reproduction number of the model (2.2) with condom use and screening of unaware infective individuals, by defintion 5 it is given as follows:

$$R_0 = \rho(FV^{-1}),$$

where

$$F(E_0) = \begin{bmatrix} (1 - u_1) \beta_1 c_1 & (1 - u_1) \beta_2 c_2 & (1 - u_1) \beta_3 c_3 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad (2.10)$$

and

$$V(E_0) = \begin{bmatrix} (\theta + \delta + \mu) & 0 & 0 \\ -\theta & (\delta + \mu + \pi) & 0 \\ -\delta & -(\delta + \pi) & (\alpha + \mu) \end{bmatrix}, \quad (2.11)$$

since $\det(V) = (\theta + \delta + \mu)(\delta + \mu + \pi)(\alpha + \mu) \neq 0$, therefore the matrix V is inverse,

where inverse is given by

$$V^{-1} = \frac{1}{\det(V)} \begin{bmatrix} (\delta + \mu + \pi)(\alpha + \mu) & 0 & 0 \\ \theta(\alpha + \mu) & (\theta + \delta + \mu)(\alpha + \mu) & 0 \\ \theta(\delta + \pi) + \delta(\theta + \mu + \pi) & (\delta + \pi)(\delta + \mu + \pi) & (\theta + \delta + \mu)(\delta + \mu + \pi) \end{bmatrix}, \quad (2.12)$$

The basic reproduction number is given by

$$R_0 = B + C + D + E, \quad (2.13)$$

where

$$\begin{aligned} B &= \frac{(1 - u_1)\beta_1 c_1}{(\theta + \delta + \mu)}, \\ C &= \frac{(1 - u_1)\beta_2 c_2 \theta}{(\theta + \delta + \mu)(\delta + \mu + \pi)}, \\ D &= \frac{(1 - u_1)\beta_3 c_3 \theta (\delta + \pi)}{(\theta + \delta + \mu)(\delta + \mu + \pi)(\alpha + \mu)}, \\ E &= \frac{(1 - u_1)\beta_3 c_3 \delta}{(\theta + \delta + \mu)(\alpha + \mu)}. \end{aligned}$$

While the basic reproduction number of the model without condom use and screening of unaware infective individuals [8], i.e ($\theta = 0$ and $u_1 = 0$), is the given by

$$R = \frac{(\beta_1 c_1 (\delta + \mu + \pi)(\alpha + \mu) + \beta_3 c_3 \delta (\delta + \pi + \mu))}{(\delta + \mu)(\delta + \mu + \pi)(\alpha + \mu)}. \quad (2.14)$$

2.2.2 Existence of endemic equilibrium

Solving the HIV/AIDS model equation in-terms of β_m^* , we calculate the equilibrium points, and obtain:

$$\begin{aligned} S^* &= \frac{Q_0}{\beta_m^* + \mu}, \\ I_1^* &= \frac{Q_0 \beta_m^*}{(\theta + \delta + \mu)(\beta_m^* + \mu)}, \\ I_2^* &= \frac{Q_0 \beta_m^* \theta}{(\theta + \delta + \mu)(\delta + \mu + \pi)(\beta_m^* + \mu)}, \end{aligned}$$

$$A^* = \frac{Q_0 \beta_m^* ((\pi + \delta)(\delta + \theta) + \delta \mu)}{(\alpha + \mu)(\theta + \delta + \mu)(\delta + \mu + \pi)(\beta_m^* + \mu)}. \quad (2.15)$$

Using theory 20 we obtain:

$$N^* = \frac{Q_0 - \alpha A^*}{\mu}. \quad (2.16)$$

By solving system (2.2) at the equilibrium we obtain $\beta_m^* = 0$ (which corresponds to the **DFE**) or

$$B_1 \beta_m^* + B_0 = 0, \text{ therefore } \beta_m^* = -\frac{B_0}{B_1}, \quad (2.17)$$

where

$$\begin{aligned} B_1 &= (\delta + \alpha + \mu)(\theta + \mu + \delta) + \pi(\alpha + \delta + \mu + \theta) > 0, \\ B_0 &= (\alpha + \mu)(\pi + \delta + \mu)(\delta + \theta + \mu)(1 - R_0). \end{aligned} \quad (2.18)$$

- if $R_0 > 1$ then $\beta_m^* > 0$ therefore the model **HIV** has a unique **endemic equilibrium**.
- if $R_0 < 1$ then $\beta_m^* \leq 0$ therefore the **HIV/AIDS** model has no **endemic equilibrium**.

Proposition 21 *The **HIV** model has a unique endemic equilibrium if and only if $R_0 > 1$.*

Now we will discuss the existence of all possible equilibria of the model system (2.2). We found that the system (2.2) has two possible non-negative equilibria namely the disease-free equilibrium (**DFE**) E_0 and the **endemic equilibrium** E_1 .

2.3 Local stability of the equilibriums points of the HIV/AIDS model

In this section we will study the local stability of equilibres points of the model (2.2).

2.3.1 Local stability of the disease-free equilibrium(DFE)

Let examine the local stability of the disease-free equilibriumis $E_0 = \left(\frac{Q_0}{\mu}, 0, 0, 0\right)$.

Proposition 22 *The disease-free equilibrium (DFE) of the (2.6) is locally asymptotically stable.*

Proof 23 The Jacobian matrix for the model (2.2), evaluated at E_0 , is given by

$$J(E_0) = \begin{bmatrix} -\mu & -\Lambda_1 & -\Lambda_2 & -\Lambda_3 \\ 0 & \Lambda_1 - (\theta + \delta + \mu) & \Lambda_2 & \Lambda_3 \\ 0 & \theta & -(\delta + \mu + \pi) & 0 \\ 0 & \delta & \delta + \pi & -(\alpha + \mu) \end{bmatrix}, \quad (2.19)$$

where

$$\begin{aligned} \Lambda_1 &= (1 - u_1)\beta_1 c_1 \\ \Lambda_2 &= (1 - u_1)\beta_2 c_2, \\ \Lambda_3 &= (1 - u_1)\beta_3 c_3. \end{aligned}$$

It is clear that $-(\mu)$ is an eigenvalue. Hence, by removing the first column and the first row, the Jacobian matrix will be reduced as

$$J(E_0) = \begin{bmatrix} \Lambda_1 - (\theta + \delta + \mu) & \Lambda_2 & \Lambda_3 \\ \theta & -(\delta + \mu + \pi) & 0 \\ \delta & \delta + \pi & -(\alpha + \mu) \end{bmatrix}. \quad (2.20)$$

We therefore calculate the eigenvalues of the reduced matrix. Solving the eigenvalues of $J(E_0)$, requires that

$$\det(J(E_0) - \xi) = 0,$$

which leads to the following characteristic polynomial,

$$\xi^3 + a_1 \xi^2 + a_2 \xi + a_3 = 0, \quad (2.21)$$

here

$$\begin{aligned} a_1 &= (\alpha + \mu) + (\delta + \mu + \pi) + (\theta + \delta + \mu)(1 - A), \\ a_2 &= (\alpha + \mu)(\delta + \mu + \pi) \\ &\quad + (\delta + \mu + \pi)(\theta + \delta + \mu)(1 - A - B) \\ &\quad + (\alpha + \mu)(\theta + \delta + \mu)(1 - A - D) \\ a_3 &= (\theta + \delta + \mu)(\delta + \mu + \pi)(\alpha + \mu)(1 - R_0). \end{aligned}$$

Now we applying theorem 9 stability conditions

$$a_1 > 0, \quad a_3 > 0, \quad a_1 a_2 - a_3 > 0. \quad (2.22)$$

We examine the conditions (2.22) for the equation (2.21) $a_3 > 0$, will be resulted if $R_0 < 1$.

Since $A > 0$, then with respect to (2.13) we can conclude $A < R_0$. Thus, if $R_0 < 1$ then $a_1 > 0$. Finally, we investigate the third stability condition. With some algebraic computations, we have

$$\begin{aligned} a_1 a_2 - a_3 &= (\alpha + \mu)^2 (\pi + \delta + \mu) + (\alpha + \mu)^2 (\theta + \delta + \mu) (1 - B - E) \\ &+ (\delta + \mu + \pi)^2 (\alpha + \mu) + (\delta + \mu + \pi)^2 (\theta + \delta + \mu) (1 - B - C) \\ &+ (\delta + \mu + \pi) (\alpha + \mu) (\theta + \delta + \mu) [2(1 - B) + D] \\ &+ (\theta + \delta + \mu)^2 (\delta + \mu + \pi) (1 - B) (1 - B - C) \\ &+ (\theta + \delta + \mu)^2 (\alpha + \mu) (1 - B - E) (1 - B). \end{aligned}$$

Since all the parameters B, C, D , and E are smaller than R_0 , hence if $R_0 < 1$, then from the above relation. With these assumptions, all the stability conditions (2.22) are satisfied and the disease-free equilibrium E_0 is locally asymptotically stable.

2.3.2 Local stability of endemic equilibrium.

Let $R_0 > 1$, we will study the stability local of endemic equilibrium E_1 in (2.15) in the system (2.2).

We evaluate the Jacobian matrix of the model at the **endemic equilibrium** E_1 and we obtain

$$J_{E_1} = \begin{bmatrix} -G - \mu & J_1 & J_2 & J_3 \\ G & -J_1 - B & -J_2 & -J_3 \\ 0 & \theta & -C & 0 \\ 0 & \delta & D & -E \end{bmatrix}, \quad (2.23)$$

where: $B = \theta + \delta + \mu$, $C = \delta + \mu + \pi$, $D = \delta + \pi$, $E = \alpha + \mu$.

$$\begin{aligned} G &= \frac{(N^* - S^*)}{N^*} \beta_m^* > 0, \\ J_1 &= S^* \left(\beta_m^* - \frac{(1 - u_1)}{N^*} \beta_1 C_1 \right) > 0, \\ J_2 &= S^* \left(\beta_m^* - \frac{(1 - u_1)}{N^*} \beta_2 C_2 \right) > 0, \\ J_3 &= S^* \left(\beta_m^* - \frac{(1 - u_1)}{N^*} \beta_3 C_3 \right) > 0. \end{aligned} \quad (2.24)$$

The characteristic equation corresponding to J_{E_1} is

$$\det (J_{E_1} - \lambda I_4) = \lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4, \quad (2.25)$$

where

$$\begin{aligned} b_1 &= G + \mu + B + C + E + J_1 > 0, \\ b_2 &= (B + C)(\mu + E + G) + E(J_1 + G + \mu) + (\mu + C) J_1 + CB + \theta J_2 + \delta J_3 > 0, \\ b_3 &= (\mu + G)(C + E)B + (C + \mu)(J_1 + \delta) + \theta D J_3 + \theta J_2(E + \mu) + C\mu J_1 > 0, \\ b_4 &= E(C(G + \mu)(B + J_1) + \theta\mu J_2) + (\theta D + C\delta)\mu J_3 > 0. \end{aligned} \quad (2.26)$$

It is clear that

$$b_i > 0 \text{ for } j = 1, 2, 3, 4,$$

using theorem 9, E_1 is locally asymptotically stable if the following conditions hold

$$\begin{aligned} \text{(i)} \quad & b_1 b_2 - b_3 > 0, \\ \text{(ii)} \quad & b_3(b_1 b_2 - b_3) - b_1^2 b_4 > 0. \end{aligned} \quad (2.27)$$

Hence we have the following theorem:

Theorem 24 *L'équilibre endémique E_1 du modèle VIH / SIDA est localement asymptotiquement stable avec les conditions (i) et (ii) satisfaites..*

2.4 Global stability of the equilibriums points of the HIV/AIDS model

In this section we will study the global stability of the equilibriums points of the model HIV/AIDS (2.2).

2.4.1 Global stability of the disease-free equilibrium(DFE)

We will study the global stability of the disease-free equilibrium (DFE) of system (2.11) using lemma 11.

Theorem 25 [9] *The fixed point $E_0 = \left(\frac{Q_0}{\mu}, 0, 0, 0\right)$ is a globally asymptotically stable equilibrium of system (2.2) if $R_0 < 1$.*

Proof 26 *Applying Lemma 11 to model system (2.2), consider*

$$X_1 = S, \tag{2.28}$$

and

$$X_2 = \begin{bmatrix} I_1 \\ I_2 \\ A \end{bmatrix}. \tag{2.29}$$

When $I_1 = I_2 = A = 0$, the uninfected subsystem (i.e the equation for S) becomes

$$\frac{dS}{dt} = Q_0 - \mu S, \tag{2.30}$$

which has the solution

$$S(t) = \frac{Q_0}{\mu} + e^{-\mu t} \left(S(0) - \frac{Q_0}{\mu} \right), \tag{2.31}$$

obviously $S(t) \rightarrow \frac{Q_0}{\mu}$ as $t \rightarrow \infty$ regardless of the initial value $S(0)$. Therefore, it show that condition (C_1) in lemma 11 for our model (2.2) next, the right-hand side of the infectious subsysteme (i.e the equations for I_1, I_2, A) can be written as

$$\begin{aligned} \frac{dX_2}{dt} = G(X_1, X_2) &= \begin{bmatrix} \frac{dI_1}{dt} \\ \frac{dI_2}{dt} \\ \frac{dA}{dt} \end{bmatrix} \\ &= \begin{bmatrix} -(\theta + \delta + \mu) I_1 + \frac{1-u_1}{N} \beta_1 c_1 \frac{Q_0}{\mu} I_1 + \frac{1-u_1}{N} \beta_2 c_2 \frac{Q_0}{\mu} I_2 + \frac{1-u_1}{N} \beta_3 c_3 \frac{Q_0}{\mu} A \\ -\beta_m \frac{Q_0}{\mu} + \beta_m S \\ \theta I_1 - (\theta + \delta + \mu) I_2 \\ \delta I_1 + (\delta + \pi) I_2 - (\alpha + \mu) A \end{bmatrix}. \end{aligned}$$

$$\begin{aligned}
 &= \begin{bmatrix} -(\theta + \delta + \mu) + \frac{1 - u_1}{N} \beta_1 c_1 \frac{Q_0}{\mu} & \frac{1 - u_1}{N} \beta_2 c_2 \frac{Q_0}{\mu} & \frac{1 - u_1}{N} \beta_3 c_3 \frac{Q_0}{\mu} \\ \theta & -(\theta + \delta + \mu) & 0 \\ \delta & (\delta + \pi) & -(\alpha + \mu) \end{bmatrix} \times \begin{bmatrix} I_1 \\ I_2 \\ A \end{bmatrix} \\
 &\quad - \begin{bmatrix} \beta_m \frac{Q_0}{\mu} - \beta_m S \\ 0 \\ 0 \end{bmatrix}. \\
 &= MX_2 - \hat{G}(X_1, X_2), \tag{2.32}
 \end{aligned}$$

where

$$M = \begin{bmatrix} -(\theta + \delta + \mu) + \frac{1 - u_1}{N} \beta_1 c_1 \frac{Q_0}{\mu} & \frac{1 - u_1}{N} \beta_2 c_2 \frac{Q_0}{\mu} & \frac{1 - u_1}{N} \beta_3 c_3 \frac{Q_0}{\mu} \\ \theta & -(\theta + \delta + \mu) & 0 \\ \delta & (\delta + \pi) & -(\alpha + \mu) \end{bmatrix}, \tag{2.33}$$

and

$$\hat{G}(X_1, X_2) = \begin{bmatrix} \beta_m \frac{Q_0}{\mu} - \beta_m S \\ 0 \\ 0 \end{bmatrix}. \tag{2.34}$$

It is obvious that $S \leq \frac{Q_0}{\mu}$, hence it is clear that $\hat{G}(X_1, X_2) \geq 0$ for all $(X_1, X_2) \in \mathbb{R}_+^3$. We also notice that the matrix M is an M -matrix since all its off-diagonal elements are non-negative. Hence, this proves the **global stability** of the **DFE** (E_0).

2.4.2 Global stability of the endemic equilibrium of the HIV/AIDS model for $\alpha = 0$

Our goal here is to show that the endemic equilibrium of the HIV model for $\alpha = 0$ is globally asymptotically stable [9] if $R_0 > 1$. It is, however, interesting to note that the classical method of Lyapunov functions combined with the Volterra–Lyapunov matrix properties [14, 15] can lead to the proof of the endemic global stability. The details are provided below. We will study the system (2.2) in the biologically feasible domain

$$D = \left\{ (S, I_1, I_2, A) \in \mathbb{R}_+^4 : S + I_1 + I_2 + A \leq \frac{Q_0}{\mu} \right\},$$

which is clearly a positively invariant set in \mathbb{R}_+^4 . Further, $N(t) = N^*(t) = \frac{Q_0}{\mu}$ as $t \rightarrow \infty$.

Thus, we have the following limiting system:

$$\begin{aligned} \frac{dS(t)}{dt} &= Q_0 - \frac{1-u_1}{N^*} (\beta_1 c_1 I_1(t) + \beta_2 c_2 I_2(t) + \beta_3 c_3 A(t)) S(t) - \mu S(t) \quad t > 0, \\ \frac{dI_1(t)}{dt} &= \frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1(t) + \beta_2 c_2 I_2(t) + \beta_3 c_3 A(t)) S(t) - (\theta + \mu + \delta) I_1(t) \quad t > 0, \\ \frac{dI_2(t)}{dt} &= \theta I_1(t) - (\delta + \mu + \pi) I_2(t) \quad t > 0, \\ \frac{dA(t)}{dt} &= \delta I_1(t) + \delta I_2(t) + \pi I_2(t) - \mu A(t) \quad t > 0. \end{aligned} \tag{2.35}$$

To prove global stability result we propose the following **Lyapunov** function

$$V = m_1 (S - S^*)^2 + m_2 (I_1 - I_1^*)^2 + m_3 (I_2 - I_2^*)^2 + m_4 (A - A^*)^2, \tag{2.36}$$

where m_1, m_2, m_3 and m_4 are positive constants calculating the time derivative of V along the trajectories of system (2.2), we obtain

$$\begin{aligned} \frac{dV}{dt} &= \left[2m_1 (S - S^*) \frac{dS}{dt} + 2m_2 (I_1 - I_1^*) \frac{dI_1}{dt} + 2m_3 (I_2 - I_2^*) \frac{dI_2}{dt} + 2m_4 (A - A^*) \frac{dA}{dt} \right] \\ &= 2m_1 \left[-\frac{1-u_1}{N^*} (\beta_1 c_1 (I_1 - I_1^*) + \beta_2 c_2 (I_2 - I_2^*) + \beta_3 c_3 (A - A^*)) (S - S^*) - \mu (S - S^*) \right] (S - S^*) \\ &\quad + 2m_2 (I_1 - I_1^*) \left[\frac{1-u_1}{N^*} (\beta_1 c_1 (I_1 - I_1^*) + \beta_2 c_2 (I_2 - I_2^*) + \beta_3 c_3 (A - A^*)) (S - S^*) \right. \\ &\quad \left. - (\theta + \delta + \mu) (I_1 - I_1^*) \right] + 2m_3 (I_2 - I_2^*) [\theta (I_1 - I_1^*) - (\delta + \mu + \pi) (I_2 - I_2^*)] \\ &\quad + 2m_4 (A - A^*) [\delta (I_1 - I_1^*) - (\delta + \pi) (I_2 - I_2^*) - \mu (A - A^*)], \\ \frac{dV}{dt} &= 2m_1 \left[-\frac{(1-u_1)}{N^*} (\beta_1 c_1 (I_1 S - I_1^* S^*) + \beta_2 c_2 (I_2 S - I_2^* S^*) + \beta_3 c_3 (AS - A^* S^*)) (S - S^*) \right. \\ &\quad \left. - \mu (S - S^*)^2 \right] + 2m_2 \left[\frac{(1-u_1)}{N^*} (\beta_1 c_1 (I_1 S - I_1^* S^*) + \beta_2 c_2 (I_2 S - I_2^* S^*) + \beta_3 c_3 (AS - A^* S^*)) (I_1 - I_1^*) \right. \\ &\quad \left. - (\theta + \delta + \mu) (I_1 - I_1^*)^2 \right] + 2m_3 [\theta (I_1 - I_1^*) (I_2 - I_2^*) - (\delta + \mu + \pi) (I_2 - I_2^*)^2] \\ &\quad + 2m_4 [\delta (I_1 - I_1^*) (A - A^*) - (\delta + \pi) (I_2 - I_2^*) (A - A^*) - \mu (A - A^*)^2], \end{aligned}$$

Then, we add the expression $\beta_1 c_1 I_1 S^*$, $\beta_2 c_2 I_2 S^*$ and $\beta_3 c_3 A S^*$ into the first and second square brackets. As a result, we obtain:

$$\begin{aligned} \frac{dV}{dt} = & 2m_1 \left[-\frac{(1-u_1)}{N^*} (\beta_1 c_1 (I_1 S - I_1^* S^*) + \beta_1 c_1 I_1 S^* - \beta_1 c_1 I_1 S^* + \beta_2 c_2 (I_2 S - I_2^* S^*)) \right. \\ & + \beta_2 c_2 I_2 S^* - \beta_2 c_2 I_2 S^* + \beta_3 c_3 (A S - A^* S^*) + \beta_3 c_3 A S^* - \beta_3 c_3 A S^* (S - S^*) - \mu (S - S^*)^2 \left. \right] \\ & + 2m_2 \left[\frac{(1-u_1)}{N^*} (\beta_1 c_1 (I_1 S - I_1^* S^*) + \beta_1 c_1 I_1 S^* - \beta_1 c_1 I_1 S^* + \beta_2 c_2 (I_2 S - I_2^* S^*)) \right. \\ & + \beta_2 c_2 I_2 S^* - \beta_2 c_2 I_2 S^* + \beta_3 c_3 (A S - A^* S^*) + \beta_3 c_3 A S^* - \beta_3 c_3 A S^* (I_1 - I_1^*) - (\theta + \delta + \mu) (I_1 - I_1^*)^2 \left. \right] \\ & + 2m_3 [\theta (I_1 - I_1^*) (I_2 - I_2^*) - (\delta + \mu + \pi) (I_2 - I_2^*)^2] + 2m_4 [\delta (I_1 - I_1^*) (A - A^*) \\ & - (\delta + \pi) (I_2 - I_2^*) (A - A^*) - \mu (A - A^*)^2], \end{aligned}$$

therefore, we have

$$\begin{aligned} \frac{dV}{dt} = & 2m_1 \left[-\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 (S - S^*) + \beta_1 c_1 S^* (I_1 - I_1^*) + \beta_2 c_2 I_2 (S - S^*) + \beta_2 c_2 S^* (I_2 - I_2^*)) \right. \\ & \left. + \beta_3 c_3 A (S - S^*) + \beta_3 c_3 S^* (A - A^*) - \mu (S - S^*)^2 \right] \\ & + 2m_2 \left[\frac{(1-u_1)}{N^*} (I_1 - I_1^*) (\beta_1 c_1 I_1 (S - S^*) + \beta_1 c_1 S^* (I_1 - I_1^*) + \beta_2 c_2 I_2 (S - S^*) \right. \\ & \left. + \beta_2 c_2 S^* (I_2 - I_2^*) + \beta_3 c_3 A (S - S^*) + \beta_3 c_3 S^* (A - A^*)) - (\theta + \delta + \mu) (I_1 - I_1^*)^2 \right] \\ & + 2m_3 [\theta (I_1 - I_1^*) (I_2 - I_2^*) - (\delta + \mu + \pi) (I_2 - I_2^*)^2] + 2m_4 \delta (I_1 - I_1^*) (A - A^*) \\ & - (\delta + \pi) (I_2 - I_2^*) (A - A^*) - \mu (A - A^*)^2], \\ = & 2m_1 \left[-\frac{(1-u_1)}{N^*} \{ (\beta_1 c_1 I_1^* + \beta_2 c_2 I_2^* + \beta_3 c_3 A^*) (S - S^*)^2 + \beta_1 c_1 S^* (I_1 - I_1^*) (S - S^*) \right. \\ & \left. + \beta_2 c_2 S^* (I_2 - I_2^*) (S - S^*) + \beta_3 c_3 S^* (A - A^*) (S - S^*) \} - \mu (S - S^*)^2 \right] \\ & + 2m_2 \left[\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1^* + \beta_2 c_2 I_2^* + \beta_3 c_3 A^*) (S - S^*) (I_1 - I_1^*) + \beta_1 c_1 S^* (I_1 - I_1^*) \right. \end{aligned}$$

Proof 28 From (2.38), we obtain

$$D = -\tilde{P} = \begin{bmatrix} \frac{1-u_1}{N^*}(\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^* + \beta_3 C_3 A^*) + \mu & \frac{1-u_1}{N^*} \beta_1 c_1 S^* \\ -\frac{1-u_1}{N^*}(\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^* + \beta_3 C_3 A^*) & -\frac{1-u_1}{N^*} \beta_1 c_1 S^* + (\theta + \delta + \mu) \\ 0 & -\theta \\ & \frac{1-u_1}{N^*} \beta_2 c_2 S^* \\ & -\frac{1-u_1}{N^*} \beta_2 c_2 S^* \\ & (\mu + \delta + \pi) \end{bmatrix}. \quad (2.39)$$

To prove the diagonal stability of D and based on lemma 14 we need to show that the following three conditions are satisfied.

Condition 1. We show that the matrix \widetilde{D} is diagonal stable. For this purpose, it is necessary to show that $-\widetilde{D}$ is Volterra-Lyapunov stable:

$$-\widetilde{D} = \begin{bmatrix} -\frac{(1-u_1)}{N^*}(\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) - \mu & -\frac{1-u_1}{N^*} \beta_1 c_1 S^* \\ \frac{(1-u_1)}{N^*}(\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) & \frac{1-u_1}{N^*} \beta_1 c_1 S^* - (\theta + \delta + \mu) \end{bmatrix}. \quad (2.40)$$

Clearly $-\widetilde{D}_{11} = -\frac{(1-u_1)}{N^*}(\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) - \mu < 0$, note that $u_1 \in [0, 1]$. Next we show $-\widetilde{D}_{22} = \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* - (\theta + \delta + \mu) < 0$.

according (2.6), we have:

$$\beta_m^* S^* = (\theta + \delta + \mu) I_1^*,$$

therefore

$$\frac{(1-u_1)(\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^* + \beta_3 C_3 A^*)}{N^*} S^* = (\theta + \delta + \mu) I_1^*,$$

then

$$\frac{(1-u_1)(\beta_1 c_1 I_1^*)}{N^*} S^* < (\theta + \delta + \mu) I_1^*,$$

therefore

$$\frac{(1-u_1)(\beta_1 c_1)}{N^*} S^* < (\theta + \delta + \mu). \quad (2.41)$$

Also, it is

$$\begin{aligned} \det(-\tilde{D}) &= \left(-\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) - \mu \right) \left(\frac{1-u_1}{N^*} \beta_1 c_1 S^* - (\theta + \delta + \mu) \right) \\ &\quad + \left(\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) \right) \left(\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* \right). \end{aligned} \quad (2.42)$$

hence $-\widetilde{D}_{22} < 0$, since $-\widetilde{D}_{11} \times -\widetilde{D}_{22} > 0$ and $-\widetilde{D}_{21} > 0$, and $-\widetilde{D}_{12} < 0$ to see $\det(-\widetilde{D}) > 0$. Therefore, $-\widetilde{D}$ is Volterra-Lyapunov stable hence \widetilde{D} is diagonal stable, based on lemma 14.

Condition 2. We show that the matrix \widetilde{D}^{-1} is diagonal stable. In fact, we show that $-\widetilde{D}^{-1}$ is Volterra-Lyapunov stable:

$$-\widetilde{D}^{-1} = \frac{1}{\det(\widetilde{D})} \begin{bmatrix} -\widetilde{D}_{11}^{-1} & -\widetilde{D}_{12}^{-1} \\ -\widetilde{D}_{21}^{-1} & -\widetilde{D}_{22}^{-1} \end{bmatrix}. \quad (2.43)$$

Where

$$\begin{aligned} \det(D) &= (\beta_m + \mu) \left[\left(-\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* + (\theta + \delta + \mu) \right) (\mu + \delta + \pi) \right. \\ &\quad \left. - \frac{(1-u_1)}{N^*} \beta_2 c_2 S^* \theta \right] + \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* \left[(1-u_1) \beta_m (\mu + \delta + \pi) \right] + \frac{(1-u_1)}{N^*} \beta_2 c_2 S^* \left[(1-u_1) \beta_m \theta \right], \\ &= (\beta_m + \mu) \left[\left(-\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* + (\theta + \delta + \mu) \right) (\mu + \delta + \pi) - \frac{(1-u_1)}{N^*} \beta_2 c_2 S^* \theta \right] \\ &\quad + \beta_m \left[\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* (\mu + \delta + \pi) + \frac{(1-u_1)}{N^*} \beta_2 c_2 S^* \theta \right], \end{aligned} \quad (2.44)$$

multiplying the above equality by θ , we have

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 \theta I_1^* S^*) + \frac{(1-u_1)}{N^*} (\beta_2 C_2 \theta I_2^* S^*) \leq (\theta + \delta + \mu) \theta I_1^*,$$

therefore

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 (\delta + \mu + \pi) I_2^* S^*) + \frac{(1-u_1)}{N^*} (\beta_2 C_2 \theta I_2^* S^*) \leq (\delta + \mu + \pi) (\theta + \delta + \mu) I_2^*,$$

then

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 (\delta + \mu + \pi) S^*) + \frac{(1-u_1)}{N^*} (\beta_2 C_2 \theta S^*) \leq (\delta + \mu + \pi) (\theta + \delta + \mu),$$

furthermore

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 (\delta + \mu + \pi) + \beta_2 C_2 \theta) S^* \leq (\delta + \mu + \pi) (\theta + \delta + \mu),$$

now, we get

$$\det(D) > 0, \tag{2.45}$$

therefore, we have:

$$\begin{aligned} -\widetilde{D}_{11}^{-1} &= \left[\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* (\delta + \mu + \pi) - (\delta + \mu + \pi) (\theta + \delta + \mu) \right] + \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* \theta, \\ -\widetilde{D}_{12}^{-1} &= \frac{(1-u_1)}{N^*} (\beta_1 c_1 S^* (\delta + \mu + \pi)) + \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* \theta, \\ -\widetilde{D}_{21}^{-1} &= -\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) (\delta + \mu + \pi), \\ -\widetilde{D}_{22}^{-1} &= -\left(\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) + \mu \right) (\delta + \mu + \pi). \end{aligned} \tag{2.46}$$

According to (2.6), we have:

$$\beta_m^* S^* = (\theta + \delta + \mu) I_1^* \quad \text{So} \quad \frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^* + \beta_3 C_3 A^*) S^* = (\theta + \delta + \mu) I_1^*,$$

therefor

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^*) S^* < (\theta + \delta + \mu) I_1^*. \tag{2.47}$$

multiplying the resulting inequality by θ , we have:

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 \theta I_1^* + \beta_2 C_2 \theta I_2^*) S^* < (\theta + \delta + \mu) \theta I_1^*, \tag{2.48}$$

using (2.7) and (2.50), we have:

$$\frac{(1-u_1)}{N^*} (\beta_1 c_1 (\delta + \mu + \pi) I_2^* S^* + \beta_2 C_2 \theta I_2^* S^*) < (\theta + \delta + \mu) (\delta + \mu + \pi) I_2^*, \quad . \tag{2.49}$$

where

$$\frac{(1-u_1)}{N^*}(\beta_1 c_1 (\delta + \mu + \pi) S^* + \beta_2 C_2 \theta S^*) < (\theta + \delta + \mu) (\delta + \mu + \pi),$$

therefore, we have

$$-\widetilde{D}_{11}^{-1} = \left[\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* (\delta + \mu + \pi) - (\delta + \mu + \pi) (\theta + \delta + \mu) \right] + \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* \theta < 0, \quad (2.50)$$

hence, $-\widetilde{D}_{11}^{-1} < 0$. It is easy to see $\det(D) > 0$. Therefore, $-\widetilde{D}^{-1}$ is Volterra-Lyapunov stable based on lemma 14.

Condition 3. It is obvious that $D_{33} = (\delta + \mu + \pi) > 0$.

Hence, lemma 16 guarantees that $D = -\widetilde{P}$ is diagonal stable.

Lemma 29 [9] For the matrix P defined (2.38) matrix $E = -\widetilde{P}^{-1}$ is diagonal stable.

Proof 30 To prove diagonal stability of E , once again use lemma 16 as follows:

- \widetilde{E} is diagonal stable.
- \widetilde{E}^{-1} is diagonal stable.
- $E_{33} > 0$.

From (2.38), we obtain

$$E = -\widetilde{P}^{-1} = \frac{1}{\det(-P)} \begin{bmatrix} -\widetilde{P}_{11}^{-1} & -\widetilde{P}_{12}^{-1} & -\widetilde{P}_{13}^{-1} \\ -\widetilde{P}_{21}^{-1} & -\widetilde{P}_{22}^{-1} & -\widetilde{P}_{23}^{-1} \\ -\widetilde{P}_{31}^{-1} & -\widetilde{P}_{32}^{-1} & -\widetilde{P}_{33}^{-1} \end{bmatrix}, \quad (2.51)$$

where

$$\begin{aligned} -\widetilde{P}_{11}^{-1} &= \frac{(1-u_1)}{N^*} - \beta_3 C_3 S^* ([\theta (\delta + \pi) + \delta (\delta + \mu + \pi)] + \mu \{ (\delta + \mu + \pi) (\frac{(1-u_1)}{N^*} (\beta_1 c_1 S^*) \\ &\quad + (\theta + \delta + \mu)) - \theta \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* \}, \\ -\widetilde{P}_{12}^{-1} &= \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* (\theta (\delta + \pi) + \delta (\delta + \mu + \pi)) - \mu \frac{(1-u_1)}{N^*} (-\beta_1 c_1 S^* (\delta + \mu + \pi) + \beta_2 C_2 S^* \theta), \\ -\widetilde{P}_{13}^{-1} &= -\frac{(1-u_1)}{N^*} (\theta + \delta + \mu) S^* [\mu \beta_2 C_2 + (\delta + \pi) \beta_3 C_3], \end{aligned}$$

$$\begin{aligned}
 -\widetilde{P}_{21}^{-1} &= \frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) (\mu (\delta + \mu + \pi)), \\
 -\widetilde{P}_{22}^{-1} &= \left(\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) + \mu \right) (\mu (\delta + \mu + \pi)), \\
 -\widetilde{P}_{23}^{-1} &= \mu \frac{(1-u_1)}{N^*} S^* [\mu \beta_2 C_2 + (\delta + \pi) \beta_3 C_3], \\
 -\widetilde{P}_{31}^{-1} &= \frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) (\mu \theta), \\
 -\widetilde{P}_{32}^{-1} &= \left(\frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) + \mu \right) (\mu \theta), \\
 -\widetilde{P}_{33}^{-1} &= -\mu \left[\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* \mu + \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* \delta - \mu (\theta + \delta + \mu) \right] \\
 &\quad + \mu \frac{(1-u_1)}{N^*} (\beta_1 c_1 I_1 + \beta_2 C_2 I_2 + \beta_3 C_3 A) (\theta + \delta + \mu).
 \end{aligned}$$

and

$$-\widetilde{E} = \begin{bmatrix} \widetilde{P}_{11}^{-1} & \widetilde{P}_{21}^{-1} \\ \widetilde{P}_{12}^{-1} & \widetilde{P}_{22}^{-1} \end{bmatrix}. \tag{2.52}$$

Now, we show that the $-\widetilde{E}$ is Volterra-Lyapunov stable. As we know

$$\begin{aligned}
 -\widetilde{P}_{11}^{-1} &= \frac{(1-u_1)}{N^*} (-\beta_3 C_3 S^*) ([\theta (\delta + \pi) + \delta (\delta + \mu + \pi)] + \mu) \times \{ (\delta + \mu + \pi) \left[\frac{(1-u_1)}{N^*} (\beta_1 c_1 S^*) \right. \\
 &\quad \left. + (\theta + \delta + \mu) \right] - \theta \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* \},
 \end{aligned}$$

from (2.5), we have

$$\frac{(1-u_1) (\beta_1 c_1 I_1^* + \beta_2 C_2 I_2^* + \beta_3 C_3 A^*)}{N^*} S^* = (\theta + \delta + \mu) I_1^*,$$

multiplying the above equality by $(\theta \mu)$, using (2.6) and (2.7), we have

$$\begin{aligned}
 \mu \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* (\delta + \mu + \pi) I_2^* + \theta \mu \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* I_2^* + \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* I_2^* [\delta (\delta + \mu + \pi) + \theta (\delta + \pi)] \\
 = \mu (\theta + \delta + \mu) (\delta + \mu + \pi) I_2^*,
 \end{aligned} \tag{2.53}$$

and

$$\begin{aligned} \det(-P) &= (\beta_m^* + \mu) \left[- \left(\frac{(1-u_1)}{N^*} \beta_1 c_1 S^* + (\theta + \delta + \mu) \right) (\mu (\delta + \mu + \pi)) \right. \\ &+ \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* (-\theta \mu) - \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* (\theta (\delta + \pi) + \delta (\delta + \mu + \pi)) \left. \right] - \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* \\ &\times [-\beta_m^* \mu (\delta + \mu + \pi)] + \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* [\beta_m^* (\mu \theta)] + \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* [-\beta_m^* \theta (\delta + \pi) + \delta (\delta + \mu + \pi)] \\ &+ \delta (\delta + \mu + \pi)], \end{aligned}$$

therefore

$$\det(-P) = (\beta_m^* + \mu) \widetilde{P}_{11}^{-1} - \beta_m^* \widetilde{P}_{12}^{-1}, \quad (2.54)$$

This implies that $\widetilde{P}_{11}^{-1} = 0$. It is obvious that $\widetilde{P}_{11}^{-1} < 0$, and $\widetilde{P}_{12}^{-1} < 0$, and since $\det(-P) > 0$ one can conclude that $-\widetilde{E}$ is Volterra-Lyapunov stable.

Our next goal here is to show that $-\widetilde{E}^{-1}$ is Volterra-Lyapunov stable. Let us consider

$$-\widetilde{E}^{-1} = \frac{1}{\det(E)} \begin{bmatrix} -e_{11} & -e_{12} \\ -e_{21} & -e_{22} \end{bmatrix}, \quad (2.55)$$

where

$$\begin{aligned} -e_{11} &= -\mu (\beta_m^* + \mu) \mu \left\{ (\delta + \mu + \pi) \mu \frac{(1-u_1)}{N^*} (-\beta_1 c_1 S^*) - \delta (\delta + \mu + \pi) \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* \right. \\ &\quad \left. - \theta \mu \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* + \theta \mu \frac{(1-u_1)}{N^*} \beta_3 C_3 S^* + \mu (\theta + \delta + \mu) (\delta + \mu + \pi) \right\} \\ &\quad - \beta_m^* (\delta + \mu + \pi) (\theta + \delta + \mu) \mu^2 \end{aligned} \quad (2.56)$$

multiplying the above equality by $(\theta \mu)$, using (2.6) and (2.7), we have

$$\begin{aligned} &\mu (\delta + \mu + \pi) \frac{(1-u_1)}{N^*} \beta_1 c_1 S^* + \theta \mu \frac{(1-u_1)}{N^*} \beta_2 C_2 S^* + [\delta (\delta + \mu + \pi) + \theta (\delta + \pi)] \frac{(1-u_1)}{N^*} \beta_3 C_3 S^*, \\ &= \mu (\delta + \mu + \pi) (\theta + \delta + \mu), \end{aligned} \quad (2.57)$$

this implies that $-e_{11} < 0$. It is easy to see that $-e_{12} < 0$, $-e_{21} > 0$ and $-e_{22} < 0$ and since:

$$\begin{aligned} \det(E) &= \frac{1}{(\det(-P))^3} \beta_m^* \mu e_{12} + \frac{1}{(\det(-P))^3} \beta_m^* \theta \mu \frac{(1-u_1)}{N^*} \beta_m^* \mu [\beta_2 C_2 S^* \\ &\quad + \beta_m^* (\delta + \pi) \beta_3 C_3 S^*], \end{aligned}$$

therefore

$$\det(E) > 0, \tag{2.58}$$

one can conclude that $-\widetilde{E}^{-1}$ is Volterra-Lyapunov stable.

Theorem 31 [9] The matrix P defined in (2.38) is Volterra-Lyapunov stable.

Proof 32 Based on lemmas 16 and 26 and since $-P_{44} = \mu > 0$, there exists a positive diagonal matrix M such that $M(-P) + (-P)^T M^T > 0$. Thus

$$MP + P^T M^T < 0. \tag{2.59}$$

Theorem 33 [9] The endemic equilibrium, $E_1 = (S^*, I_1^*, I_2^*, A^*)$, of system (2.2) is globally asymptotically stable if $R_0 > 1$.

Proof 34 Based on, lemma 16, 26 and theorem 30, we obtain $\frac{dV}{dt} < 0$ when $X \neq X^*$ and X is not on the s -axis (a set measure zero). It implies that the endemic equilibrium of the model system (2.2) is globally asymptotically stable.

CHAPTER 3

Reaction-Diffusion system(R-D / PDE) of the model HIV/AIDS

In this chapter we will devote to the spatial diffusion of HIV /AIDS, and examine the local and global stability of the constant equilibriums points for this PDE model.

In the previous chapter, we studied the changes of infection **HIV/AIDS** in relation to time, and in this chapter we will study the change in time the place of the spread of infection **HIV/AIDS** spreads to the population, by increasing the mathematical factor Δ in each equation of the system (2.2). Where and we will use the same equilibriums points for the **EDO** system, we will study the local stability and the global stability of the **PDE** system for equilibriums points of the model **HIV/AIDS**.

Let the systeme the reaction-diffusion of the model HIV/AIDS is the form:

$$\left\{ \begin{array}{l} \frac{dS(x,t)}{dt} - a\Delta S(x,t) = Q_0(x,t) - \beta_m S(x,t) - \mu S(x,t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dI_1(x,t)}{dt} - b\Delta I_1(x,t) = \beta_m S(x,t) - (\theta + \delta + \mu) I_1(x,t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dI_2(x,t)}{dt} - c\Delta I_2(x,t) = \theta I_1(x,t) - (\theta + \delta + \pi) I_2(x,t) \quad \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{dA(x,t)}{dt} - d\Delta A(x,t) = \delta I_1(x,t) + (\delta + \pi) I_2(x,t) - \mu A(x,t) \quad \text{in } \mathbb{R}_+ \times \Omega. \end{array} \right. \quad (3.1)$$

Where Ω is an open bounded subset of \mathbb{R}^n with piecewise smooth boundary $\partial\Omega$.

Subject to the homogeneous Neumann boundary condition $\frac{\partial S}{\partial \eta} = \frac{\partial I_1}{\partial \eta} = \frac{\partial I_2}{\partial \eta} = \frac{\partial A}{\partial \eta} = 0$, for all $x \in \partial\Omega$. and positive initial conditions i.e

$$\begin{aligned} S(x,0) &= S_0(x) > 0, \quad I_1(x,0) = I_{1_0}(x) > 0, \quad \text{and} \\ I_2(x,0) &= I_{2_0}(x) > 0, \quad A(x,0) = A_0(x) > 0, \quad \text{where } x \in \Omega. \end{aligned}$$

Let $0 = \lambda_0 < \lambda_1 \leq \lambda_2 \leq \dots$ be the sequence of eigenvalue for the elliptic operator $(-\Delta)$, where each λ_i has multiplicity $m_i \geq 1$. Also let Φ_{ij} , $1 \leq j \leq m_i$, (recall that $\Phi_0 = \text{const}$ and $\lambda_i \rightarrow \infty$) be the normalized eigenfunction corresponding to λ_i , for $i = 0, 1, \dots$. That is, Φ_{ij} and λ_i satisfy $-\Delta\Phi_{ij} = \lambda_i\Phi_{ij}$ in Ω , with $\frac{\partial\Phi_{ij}}{\partial\nu} = 0$ in $\partial\Omega$, and $\int_{\Omega} \Phi_{ij}^2(x)dx = 1$.

3.1 Local stability of the equilibriums pionts of the model HIV/AIDS

In the section we will study the local stability of the pionts equilibriums of model (PDE) (3.1).

3.1.1 Local Stability of disease-free equilibrium(DEF) of the model HIV/AIDS

In sub-section we will study the local Stability of disease-free equilibrium (DEF) an the system **PDE**.

Let us examine the local stability of the disease-free equilibriumis E_0 , Applying the next generation method Now, we compute The basic reproduction number of the model (3.1) with condom use and screening of unaware infective individuals, by defintion 5, we get:

$$R_i = \rho(FV_i^{-1}), \text{ for } i = 0, 1, \dots,$$

where ξ it's eigenvalues in the matrix FV_i^{-1} ,

we have:

$$F = (1 - u_1) \begin{bmatrix} \beta_1 c_1 & \beta_2 c_2 & \beta_3 c_3 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}. \quad (3.2)$$

and, for $i = 0, 1, \dots$, we have

$$V_i = \begin{bmatrix} (\theta + \delta + \mu + b\lambda_i) & 0 & 0 \\ -\theta & (\delta + \mu + \pi + c\lambda_i) & 0 \\ -\delta & -(\delta + \pi) & (\alpha + \mu + d\lambda_i) \end{bmatrix}, \quad (3.3)$$

since $\det(V_i) = (\theta + \delta + \mu + b\lambda_i) (\delta + \mu + \pi + c\lambda_i) (\alpha + \mu + d\lambda_i) \neq 0$, so the matrix V_i is inverse for $i = 0, 1, \dots$,we get by

$$V_i^{-1} = \frac{1}{\det(V_i)} \begin{bmatrix} (\delta + \mu + \pi + c\lambda_i) (\alpha + \mu + d\lambda_i) & 0 & 0 \\ \theta (\alpha + \mu + d\lambda_i) & (\theta + \delta + \mu + b\lambda_i) (\alpha + \mu + d\lambda_i) & 0 \\ \theta (\delta + \pi) + \delta (\delta + \mu + \pi + c\lambda_i) & (\theta + \delta + \mu + b\lambda_i) (\delta + \pi) & (\theta + \delta + \mu + b\lambda_i) (\delta + \pi + \mu + c\lambda_i) \end{bmatrix}.$$

therefore

$$\begin{aligned} R_i &= \rho(FV_i^{-1}) \\ &= B_i + C_i + D_i + E_i, \text{ for } i = 0, 1, \dots, \end{aligned} \quad (3.4)$$

where

$$\begin{aligned}
 B_i &= \frac{(1 - u_1)\beta_1 c_1}{(\theta + \delta + \mu + d_2 \lambda_i)}, \\
 C_i &= \frac{(1 - u_1)\beta_2 c_2 \theta}{(\theta + \delta + \mu + d_2 \lambda_i)(\delta + \mu + \pi + d_3 \lambda_i)}, \\
 D_i &= \frac{(1 - u_1)\beta_3 c_3 \theta (\delta + \pi)}{(\theta + \delta + \mu + d_2 \lambda_i)(\delta + \mu + \pi + d_3 \lambda_i)(\alpha + \mu + d_4 \lambda_i)}, \\
 E_i &= \frac{(1 - u_1)\beta_3 c_3 \delta}{(\theta + \delta + \mu + d_2 \lambda_i)(\alpha + \mu + d_4 \lambda_i)}.
 \end{aligned} \tag{3.5}$$

It is clear to $R_i < R_0$, for $i = 1, 2, \dots$

While the basic reproduction number of the model without condom use and screening of unaware infective individuals is then given by $\theta = 0$ and $u_1 = 0$ so for $i = 0, 1, \dots$ that

$$R_i^* = (1 - u_1) \frac{\beta_1 c_1 (\delta + \mu + \pi + c \lambda_i) (\alpha + \mu + d \lambda_i) + \beta_3 c_3 \delta (\delta + \mu + \pi + c \lambda_i)}{(\delta + \mu + b \lambda_i) (\delta + \mu + \pi + c \lambda_i) (\alpha + \mu + d \lambda_i)}. \tag{3.6}$$

Theorem 35 *The disease free equilibrium E_0 of the model (3.1) is locally asymptotically stable if $R_0 < 1$, but unstable $R_0 > 1$.*

Proof 36 *The Jacobian matrix for the model (3.1), evaluated at E_0 , is given by*

$$J_i(E_0) = \begin{bmatrix} -(\mu + d_1 \lambda_i) & -\Lambda_1 & -\Lambda_2 & -\Lambda_3 \\ 0 & \Lambda_1 - (\theta + \delta + \mu + d_2 \lambda_i) & \Lambda_2 & \Lambda_3 \\ 0 & \theta & -(\delta + \mu + \pi + d_3 \lambda_i) & 0 \\ 0 & \delta & \delta + \pi & -(\alpha + \mu + d_4 \lambda_i) \end{bmatrix}.$$

It is clear that $-(\mu + d_1 \lambda_i)$ is an eigenvalue for $i = 0, 1, \dots$. Hence, by removing the first column and the first row, the Jacobian matrix will be reduced as

$$J_i(E_0) = \begin{bmatrix} \Lambda_1 - (\theta + \delta + \mu + d_2 \lambda_i) & \Lambda_2 & \Lambda_3 \\ \theta & -(\delta + \mu + \pi + d_3 \lambda_i) & 0 \\ \delta & \delta + \pi & -(\alpha + \mu + d_4 \lambda_i) \end{bmatrix},$$

we therefore calculate the eigenvalues ζ of the reduced matrix. Solving the eigenvalues of $J_i(E_0)$, for $i = 0, 1, \dots$ requires that

$$\det(J_i(E_0) - \zeta I_3) = 0, \quad \text{for } i = 0, 1, \dots$$

which leads to the following characteristic polynomial,

$$\zeta^3 + a_{i_1}\zeta^2 + a_{i_2}\zeta + a_{i_3} = 0 \quad \text{for } i = 0, 1, \dots, \quad (3.7)$$

where

$$\begin{aligned} a_{i_1} &= (\alpha + \mu + d_4\lambda_i) + (\delta + \mu + \pi + d_3\lambda_i) + (\theta + \delta + \mu + d_2\lambda_i)(1 - B_i), \\ a_{i_2} &= (\alpha + \mu + d_4\lambda_i)(\delta + \mu + \pi + d_3\lambda_i) \\ &+ (\delta + \mu + \pi + d_3\lambda_i)(\theta + \delta + \mu + d_2\lambda_i)(1 - B_i - C_i) \\ &+ (\alpha + \mu + d_4\lambda_i)(\theta + \delta + \mu + d_2\lambda_i)(1 - B_i - E_i), \\ a_{i_3} &= (\theta + \delta + \mu + d_2\lambda_i)(\delta + \mu + \pi + d_3\lambda_i)(\alpha + \mu + d_4\lambda_i)(1 - R_i). \end{aligned} \quad (3.8)$$

Now, we applying theorem 9 we have:

$$a_{i_1} > 0, a_{i_3} > 0, \text{ and } a_{i_1}a_{i_2} - a_{i_3}, \text{ for } i = 0, 1, \dots \quad (3.9)$$

we examine the conditions (3.9) for the equation (3.7) $a_{i_3} > 0$ will be resulted if $R_i < 1$.

Since $B_i > 0$, then, with respect to (3.4) we can conclude $B_i < R_i$. Thus, if $R_i < 1$ then $a_{i_1} > 0$, for $i = 0, 1, \dots$. Finally, we investigate the third stability condition. With some algebraic computations, we have

$$\begin{aligned} a_{i_1}a_{i_2} - a_{i_3} &= (\alpha + \mu + d_4\lambda_i)^2 (\pi + \delta + \mu + d_3\lambda_i) \\ &+ (\alpha + \mu + d_4\lambda_i)^2 (\theta + \delta + \mu + d_2\lambda_i)(1 - B_i - E_i) \\ &+ (\delta + \mu + \pi + d_3\lambda_i)^2 (\alpha + \mu + d_4\lambda_i) \\ &+ (\delta + \mu + \pi + d_3\lambda_i)^2 (\theta + \delta + \mu + d_2\lambda_i)(1 - B_i - C_i) \\ &+ (\delta + \mu + \pi + d_3\lambda_i)(\alpha + \mu + d_4\lambda_i)(\theta + \delta + \mu + d_2\lambda_i)[2(1 - B_i) + D_i] \\ &+ (\theta + \delta + \mu + d_2\lambda_i)^2 (\delta + \mu + \pi + d_3\lambda_i)(1 - B_i)(-B_i - C_i) \\ &+ (\theta + \delta + \mu + d_2\lambda_i)^2 (\alpha + \mu + d_4\lambda_i)(1 - B_i - E_i)(1 - B_i) \end{aligned}$$

Since all the parameters B_i, C_i, D_i , and E_i for $i = 0, 1, \dots$ are smaller than R_i , hence if $R_i < 1$, then from the above relation, for $i = 0, 1, \dots$. With these assumptions, all the stability conditions (3.8) are satisfied and the disease-free equilibrium E_0 is locally asymptotically stable.

3.1.2 Local stability of endemic equilibrium of the model HIV/AIDS

Let $R_0 > 1$, in the sub-section we will study the local stability of endemic equilibrium an the system (3.1).

Let us define the linearizing operator

$$J = \begin{bmatrix} a\Delta S - G - \mu & J_1 & J_2 & J_3 \\ G & b\Delta I_1 - J_1 - B & -J_2 & -J_3 \\ 0 & \theta & c\Delta I_2 - C & 0 \\ 0 & \delta & D & d\Delta A - E \end{bmatrix}, \quad (3.10)$$

therefore for $i = 0, 1, \dots$

$$J_{\lambda_i} = \begin{bmatrix} -a\lambda_i - G - \mu & J_1 & J_2 & J_3 \\ G & -b\lambda_i - J_1 - B & -J_2 & -J_3 \\ 0 & \theta & -c\lambda_i - C & 0 \\ 0 & \delta & D & -d\lambda_i - E \end{bmatrix},$$

where $B = (\theta + \delta + \mu)$, $C = (\delta + \mu + \pi)$, and $D = (\delta + \pi)$, $E = (\alpha + \mu)$,

$$\left\{ \begin{array}{l} G = \frac{(N^* - S^*)}{N^*} \beta_m^*, \\ J_1 = \frac{(1 - u_1)}{N^*} S^* (\beta_m^* - \beta_1 C_1), \\ J_2 = \frac{(1 - u_1)}{N^*} S^* (\beta_m^* - \beta_2 C_2), \\ J_3 = \frac{(1 - u_1)}{N^*} S^* (\beta_m^* - \beta_2 C_2). \end{array} \right. \quad (3.11)$$

Similar to the **ODE** case, **the asymptotic stability** of the endemic equilibrium (S^*, I_1^*, I_2^*, A^*) can be determined by examining the eigenvalue of the operator J_{λ_i} , for $i = 0, 1, \dots$. That is the solution is **asymptotically stable** if all the eigenvalues of J_{λ_i} have negative real parts. In order to achieve that, suppose $(\phi(x), \Psi(x); \Upsilon(x), \psi(x))$ is an eigenfunction of J_{λ_i} corresponding to an eigenvalue ξ . By definition [11], we have

$$J(\phi(x), \Psi(x); \Upsilon(x), \psi(x))^t = \xi(\phi(x), \Psi(x); \Upsilon(x), \psi(x))^t.$$

Leading to

$$(J - \xi I_4) \begin{bmatrix} \phi \\ \Psi \\ \Upsilon \\ \psi \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

this can be rearranged to the form

$$\sum_{0 \leq i \leq \infty, 1 \leq j \leq m_i} (M_i - \xi I_4) \begin{bmatrix} a_{ij} \\ b_{ij} \\ c_{ij} \\ d_{ij} \end{bmatrix} \Phi_{ij} = \begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad (3.12)$$

where, for $i = 0, 1, \dots$

$$M_i = \begin{bmatrix} -a\lambda_i - G - \mu & J_1 & J_2 & J_3 \\ G & -b\lambda_i - J_1 - B & -J_2 & -J_3 \\ 0 & \theta & -c\lambda_i - C & 0 \\ 0 & \delta & D & -d\lambda_i - E \end{bmatrix}. \quad (3.13)$$

The characteristic equation corresponding to J_{E_1} is for $i = 0, 1, \dots$,

$$\xi^4 + b_{i_1}\xi^3 + b_{i_2}\xi^2 + b_{i_3}\xi + b_{i_4} = 0, \quad (3.14)$$

where, for $i = 0, 1, \dots$

$$b_{i_1} = G + \mu + B + C + E + J_1 + (b + c + d + a)\lambda_i > 0,$$

$$\begin{aligned} b_{i_2} = & [a(b + c + d) + (b(c + d) + cd)]\lambda_i^2 + \lambda_i[(b + c + d + E + C + B)\mu \\ & + (E + J_1 + C)a + (B + E + C + b + c + d)G + B(C + E + d + a + c) \\ & + C(E + b + d) + (C + a + c + d + E + \mu)J_1 + (b + 1)Ec + \theta J_2 + \delta J_3] \\ & (B + E + C)G + B(C + E) + C(E) + (C + E + \mu)J_1 + \theta J_2 + \delta J_3 + \mu(B + C + E)) > 0 \end{aligned}$$

$$\begin{aligned} b_{i_3} = & [BCbcd]\lambda_i^4 + [ab(c + d) + (a + b)cd]\lambda_i^3 + \lambda_i^2[bc(1 + d) \\ & + (ab + (a + b)c)E + Ba(c + d) + Ca(b + d) + G(bc + (b + c)d) \\ & + (ac + (a + c)d)J_1] + \lambda_i[Ba(E + C) + aC(J_1 + E) + (a + d)\theta J_2] \\ & + ((\mu + E)J_1 + \delta J_3)(c + d) + (E(b + c) + C(d + b)) + (B(c + d))(G + \mu) \\ & + [B(G + \mu)(C + E) + C(J_1 + \mu J_1 + \delta) + \mu(\theta J_2 + \delta + J_1) + \theta(DJ_3 + EJ_2)] \end{aligned}$$

$$\begin{aligned}
 & +((\mu + E)J_1 + \delta J_3)(c + d) + (E(b + c) + C(d + b)) + (B(c + d))(G + \mu)] \\
 & +[B(G + \mu)(C + E) + C(J_1 + \mu J_1 + \delta) + \mu(\theta J_2 + \delta + J_1) + \theta(DJ_3 + EJ_2)] \\
 b_{i_4} = & [BCabcd^2]\lambda_i^5 + bcd\lambda_i^4[a + BCd\mu + BCGd] \\
 & +c\lambda_i^3[bd\mu + abE + adJ_1 + Gbd] + [c\mu(bE + dJ_1) \\
 & +a(cE + Cd)(B + J_1) + a(d\theta J_2 + c\delta J_3) + CabE]\lambda_i^2 \\
 & +\lambda_i[E((Bc + cJ_1)\mu + a(J_1 + B + \theta J_2)) + BGc] + Cd\mu J_1 \\
 & +(Eb + Bd)C(\mu + G) + d\theta\mu J_2 + (c\mu\delta + a(C\delta + \theta D))J_3] \\
 & +BCE(G + \mu) + (CJ_1 + \theta J_2)\mu E + (C\delta + \theta D)\mu J_3 > 0.
 \end{aligned}$$

It is clear that

$$b_{i_j} > 0 \text{ for } j = 1, 2, 3, 4,$$

by theorem 9, E_1 is locally asymptotically stable if the following conditions hold

- (i) $b_{i_1}b_{i_2} - b_{i_3} > 0$, for $i = 0, 1, \dots$,
- (ii) $b_{i_3}(b_{i_1}b_{i_2} - b_{i_3}) - b_{i_1}^2 b_{i_4} > 0$ for $i = 0, 1, \dots$

Hence we have the following theorem:

Theorem 37 *The endemic equilibrium $E_1 = (S^*, I_1^*, I_2^*, A^*)$ is locally asymptotically stable with the condition (i) and (ii) are satisfied., for $i = 0, 1, \dots$*

3.2 Global stability of systeme reaction-diffusion

In the section we will study the global stability of the pionts equilibriums of PDE model

(3.1)

3.2.1 Global stability of disease-free equilibrium(DFE) of the model HIV/AIDS

In the sub-section we will study the global stability of disease-free equilibrium (DFE) of PDE system (3.1).

Theorem 38 *The fixed point $E_0 = (\frac{Q_0}{\mu}, 0, 0, 0)$ is a globally asymptotically stable equilibrium of system (3.1), provided that $R_0 < 1$.*

Proof 39 Applying Lemma 6 to model system (3.1), consider

$$X_1 = S(x, t), \tag{3.15}$$

and

$$X_2 = \begin{bmatrix} I_1(x, t) \\ I_2(x, t) \\ A(x, t) \end{bmatrix}. \tag{3.16}$$

When $I_1 = I_2 = A = 0$, the uninfected subsystem (i.e. the equation for S) becomes

$$\frac{dX_1}{dt} = \frac{dS}{dt} = Q_0 - \mu S + a\Delta S, \tag{3.17}$$

from where we solve the following problem

$$\begin{cases} \frac{dS(x, t)}{dt} - a\Delta S(x, t) = Q_0 - \mu S(x, t) & \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{\partial S(0, t)}{\partial \eta} = 0, & \text{in } \mathbb{R}_+ \times \partial\Omega, \\ S_0(x) = \frac{Q_0}{\mu} & \text{in } \Omega. \end{cases} \tag{3.18}$$

Using the method of separating variables:

Solution homogene

$$\begin{cases} \frac{dS(x, t)}{dt} - a\Delta S(x, t) = 0 & \text{in } \mathbb{R}_+ \times \Omega, \\ \frac{\partial S(0, t)}{\partial \eta} = 0, & \text{in } \mathbb{R}_+ \times \partial\Omega, \\ S_0(x) = \frac{Q_0}{\mu} & \text{in } \Omega. \end{cases} \tag{3.19}$$

The solution is given in the form

$$S(x, t) = T(t)X(x),$$

therefor

$$\frac{1}{a} \frac{\partial T(t)}{\partial T(t)} = \frac{\partial^2 X(x)}{\partial X(x)} = -\lambda,$$

where

$$\begin{cases} \frac{\partial^2 X(x)}{\partial X(x)} - \lambda X(x) = 0, \\ \frac{\partial X(0,t)}{\partial \eta} = 0, \end{cases}$$

Solutions depend on the constant λ ,

If $\lambda > 0$ then the solutions of the differential equation are

$$X(x) = C_1 \exp(\sqrt{\lambda}x) + C_2 \exp(-\sqrt{\lambda}x)$$

with condition $\frac{\partial X(0,t)}{\partial \eta} = 0$, the solution is given by

$$X_n(x) = C_1 \cos(n\pi x).$$

If $\lambda = 0$ then the solutions of the differential equation are

$$X_n(x) = 0.$$

If $\lambda < 0$ then the solutions of the differential equation are

$$X_n(x) = 0.$$

and

$$\begin{cases} \frac{\partial T(t)}{\partial T(t)} + a\lambda T(t) = 0, \\ T(0) = \frac{Q_0}{\mu}. \end{cases}$$

solution is given by

$$T_n(t) = \frac{Q_0}{\mu} \exp(-an\pi t)$$

The solution is given by

$$S(x,t) = \frac{Q_0}{\mu} C_1 \cos(n\pi x) \exp(-an\pi t),$$

According to Fourier development we have

$$S(x,t) = \sum_{n \geq 0}^{\infty} \frac{Q_0}{\mu} C_1 \cos(n\pi x) \exp(-an\pi t). \quad (3.20)$$

Using Strum-Loviulle theory for the presence of non-homogene solution

$$S(x, t) = \frac{Q_0}{\mu} + \sum_{n \geq 1} \frac{Q_0}{\mu} C_1 \cos(n\pi x) \exp(-an\pi t). \quad (3.21)$$

For $t \rightarrow \infty$, then $S(x, t) \rightarrow \frac{Q_0}{\mu}$

obviously $S(x, t) \rightarrow \frac{Q_0}{\mu}$ as $t \rightarrow \infty$ regardless of the initial value $S_0(x)$. Therefore, it shows that condition (C1) in Lemma 11 holds for our model.

Next, the right-hand side of the infectious subsystem (i.e the equations for I_1 , I_2 and A) can be written as

$$\frac{dX_2}{dt} = G(X_1, X_2) = \begin{bmatrix} \frac{dI_1(x, t)}{dt} - \beta_m \frac{Q_0}{\mu} + \beta_m S(x, t) \\ \frac{dI_2(x, t)}{dt} \\ \frac{dA(x, t)}{dt} \end{bmatrix}, \quad (3.22)$$

then, we have for $i = 0, 1, \dots$

$$\frac{dX_2}{dt} = G_i(X_1, X_2) = \begin{bmatrix} \beta_m S(x, t) - (\theta + \delta + \mu + b\lambda_i) I_1(x, t) - \beta_m \frac{Q_0}{\mu} + \beta_m S(x, t) \\ \theta I_1(x, t) - (\delta + \mu + \pi + c\lambda_i) \\ \delta I_1(x, t) + \delta I_2(x, t) + \pi I_2(x, t) - (\alpha + \mu A + d\lambda_i) \end{bmatrix},$$

therefore, we have for $i = 0, 1, \dots$

$$\frac{dX_2}{dt} = G_i(X_1, X_2) = \begin{bmatrix} \frac{(1 - u_1)(\beta_1 c_1 I_1 + \beta_2 c_2 I_2 + \beta_3 c_3 A) Q_0}{N} - (\theta + \delta + \mu + b\lambda_i) I_1 - \beta_m \frac{Q_0}{\mu} + \beta_m S \\ \theta I_1 - (\delta + \mu + \pi + c\lambda_i) \\ \delta I_1 + \delta I_2 + \pi I_2 - (\alpha + \mu A + d\lambda_i) \end{bmatrix},$$

here, for $i = 0, 1, \dots$

$$= \begin{bmatrix} \frac{(1 - u_1)(\beta_1 c_1 I_1 + \beta_2 c_2 I_2 + \beta_3 c_3 A) Q_0}{N} - (\theta + \delta + \mu + b\lambda_i) I_1 \\ \theta I_1 - (\delta + \mu + \pi + c\lambda_i) I_2 \\ \delta I_1 + \delta I_2 + \pi I_2 - (\alpha + \mu A + d\lambda_i) A \end{bmatrix} - \begin{bmatrix} \beta_m \frac{Q_0}{\mu} - \beta_m S \\ 0 \\ 0 \end{bmatrix},$$

therefore, for $i = 0, 1, \dots$

$$\frac{dX_2}{dt} = G_i(X_1, X_2) = \begin{bmatrix} -(\theta + \delta + \mu + b\lambda_i) + \frac{(1-u_1)}{N}\beta_1c_1\frac{Q_0}{\mu} & \frac{(1-u_1)}{N}\beta_2c_2\frac{Q_0}{\mu} \\ \theta & -(\delta + \mu + \pi + c\lambda_i) \\ \delta & \delta + \pi \\ \frac{(1-u_1)}{N}\beta_3c_3\frac{Q_0}{\mu} & \\ 0 & \\ -(\alpha + \mu A + d\lambda_i) & \end{bmatrix} \times \begin{bmatrix} I_1 \\ I_2 \\ A \end{bmatrix} - \begin{bmatrix} \beta_m\frac{Q_0}{\mu} - \beta_m S \\ 0 \\ 0 \end{bmatrix},$$

$$\frac{dX_2}{dt} = G_i(X_1, X_2) = M_i X_2 - \hat{G}(X_1, X_2), \text{ for } i = 0, 1, \dots \quad (3.23)$$

where, for $i = 0, 1, \dots$

$$M_i = \begin{bmatrix} -(\theta + \delta + \mu + b\lambda_i) + \frac{(1-u_1)}{N}\beta_1c_1\frac{Q_0}{\mu} & \frac{(1-u_1)}{N}\beta_2c_2\frac{Q_0}{\mu} & \frac{(1-u_1)}{N}\beta_3c_3\frac{Q_0}{\mu} \\ \theta & -(\delta + \mu + \pi + c\lambda_i) & 0 \\ \delta & \delta + \pi & -(\alpha + \mu A + d\lambda_i) \end{bmatrix}, \quad (3.24)$$

and

$$\hat{G}(X_1, X_2) = \begin{bmatrix} \beta_m\frac{Q_0}{\mu} - \beta_m S(x, t) \\ 0 \\ 0 \end{bmatrix}. \quad (3.25)$$

It is obvious that $S(x, t) \leq \frac{Q_0}{\mu}$, hence it is clear that $\hat{G}(X_1, X_2) \geq 0$ for all $(X_1, X_2) \in \mathbb{R}_+^3$. We also notice that the matrices M_i is an M-matrices, for $i = 0, 1, \dots$ since all its off-diagonal elements are non-negative. this proves the global stability of the **DFE** E_0 .

3.2.2 Global stability of the endemic equilibrium

In sub-section we will study the global stability of the endemic equilibrium in the **PDE** system(3.1), we will use the Lyapunov function in the **EDO** system of [9] and methode of [11].

Theorem 40 *Let*

$$V(t) = \int_{\Omega} [m_1 (S - S^*)^2 + m_2 (I_1 - I_1^*)^2 + m_3 (I_2 - I_2^*)^2 + m_4 (A - A^*)^2] dx. \quad (3.26)$$

Then $V(t)$ is non-negative it is a valid Lyapunov functional. Hence (S^*, I_1^*, I_2^*, A^*) is

globally asymptotically stable if $R_0 > 1$.

Proof 41 To prove that the endemic equilibrium $E_1 = (S^*, I_1^*, I_2^*, A^*)$ is globally asymptotically stable, we need to establish that $V(t)$ is a Lyapunov functional. First, we differentiate $V(t)$ with respect to time

$$\frac{dV(t)}{dt} = \int_{\Omega} \left[2m_1 (S - S^*) \frac{dS}{dt} + 2m_2 (I_1 - I_1^*) \frac{dI_1}{dt} + 2m_3 (I_2 - I_2^*) \frac{dI_2}{dt} + 2m_4 (A - A^*) \frac{dA}{dt} \right] dx. \quad (3.27)$$

Substituting the time derivatives with their values form [\(3.20\)](#) yields

$$\begin{aligned} \frac{dV(t)}{dt} &= \int_{\Omega} [2m_1 (-\beta_m S - \mu S + a\Delta S) (S - S^*)] dx \\ &\quad + \int_{\Omega} [2m_2 (\beta_m S - (\theta + \delta + \mu) I_1 + b\Delta I_1) (I_1 - I_1^*)] dx \\ &\quad + \int_{\Omega} [2m_3 (\theta I_1 - (\theta + \delta + \pi) I_2 + c\Delta I_2) (I_2 - I_2^*)] dx \\ &\quad + \int_{\Omega} [2m_4 (\delta I_1 + (\delta + \pi) I_2 - \mu A + -d\Delta A) (A - A^*)] dx, \\ &= \int_{\Omega} [2m_1 (-\beta_m S - \mu S) (S - S^*)] dx + \int_{\Omega} [2m_1 (a\Delta S) (S - S^*)] dx \\ &\quad + \int_{\Omega} [2m_2 (\beta_m S - (\theta + \delta + \mu) I_1) (I_1 - I_1^*)] dx \\ &\quad + \int_{\Omega} [2m_2 (b\Delta I_1) (I_1 - I_1^*)] dx + \int_{\Omega} [2m_3 (\theta I_1 - (\theta + \delta + \pi) I_2) (I_2 - I_2^*)] dx \\ &\quad + \int_{\Omega} [2m_3 (c\Delta I_2) (I_2 - I_2^*)] dx \\ &\quad + \int_{\Omega} [2m_4 (\delta I_1 + (\delta + \pi) I_2 - \mu A) (A - A^*)] dx + \int_{\Omega} [2m_4 (d\Delta A) (A - A^*)] dx, \\ &\quad \int_{\Omega} [2m_1 (a\Delta S) (S - S^*)] dx + \int_{\Omega} [2m_2 (b\Delta I_1) (I_1 - I_1^*)] dx + \int_{\Omega} [2m_3 (c\Delta I_2) (I_2 - I_2^*)] dx \\ &\quad + \int_{\Omega} [2m_4 (d\Delta A) (A - A^*)] dx + \int_{\Omega} [2m_1 (-\beta_m S - \mu S) (S - S^*)] dx \\ &\quad + \int_{\Omega} [2m_2 (\beta_m S - (\theta + \delta + \mu) I_1) (I_1 - I_1^*)] dx \\ &\quad + \int_{\Omega} [2m_3 (\theta I_1 - (\theta + \delta + \pi) I_2) (I_2 - I_2^*)] dx \\ &\quad + \int_{\Omega} [2m_4 (\delta I_1 + (\delta + \pi) I_2 - \mu A) (A - A^*)] dx, \\ &= I + J. \end{aligned} \quad (3.28)$$

The first part is

$$I = I_1 + I_2 + I_3 + I_4, \quad (3.29)$$

where

$$I_1 = 2 \int_{\Omega} m_1 a \Delta S (S - S^*) dx, \quad (3.30)$$

$$I_2 = 2 \int_{\Omega} m_2 b \Delta I_1 (I_1 - I_1^*) dx, \quad (3.31)$$

$$I_3 = 2 \int_{\Omega} m_3 c \Delta I_2 (I_2 - I_2^*) dx, \quad (3.32)$$

$$I_4 = 2 \int_{\Omega} m_4 d \Delta A (A - A^*) dx. \quad (3.33)$$

The second part of the derivative is

$$\begin{aligned} J = & \int_{\Omega} [2m_1 (-\beta_m S - \mu S) (S - S^*)] dx + \int_{\Omega} [2m_2 (\beta_m S - (\theta + \delta + \mu) I_1) (I_1 - I_1^*)] dx \\ & + \int_{\Omega} [2m_3 (\theta I_1 - (\theta + \delta + \pi) I_2) (I_2 - I_2^*)] dx + \int_{\Omega} [2m_4 (\delta I_1 + (\delta + \pi) I_2 - \mu A) (A - A^*)] dx, \end{aligned} \quad (3.34)$$

form theorem 32 [9] in **EDO** system, we have

$$J < 0.$$

We start by looking at I Using Green's formula and assuming the Neumann boundary conditions, we obtain

$$\begin{aligned} I_1 &= 2 \int_{\Omega} m_1 a \Delta S (S - S^*) dx, \\ &= -2m_1 a \int_{\Omega} \nabla S \nabla (S - S^*) dx, \\ &= -2m_1 a \int_{\Omega} |\nabla S|^2 dx. \end{aligned} \quad (3.35)$$

$$\begin{aligned} I_2 &= 2 \int_{\Omega} m_2 b \Delta I_1 (I_1 - I_1^*) dx, \\ &= -2m_2 b \int_{\Omega} \nabla I_1 \nabla (I_1 - I_1^*) dx, \\ &= -2m_2 b \int_{\Omega} |\nabla I_1|^2 dx. \end{aligned} \quad (3.36)$$

and

$$\begin{aligned}
 I_3 &= 2 \int_{\Omega} m_3 c \Delta I_2 (I_2 - I_2^*) dx \\
 &= -2m_3 c \int_{\Omega} \nabla I_2 \nabla (I_2 - I_2^*) dx \\
 &= -2m_3 c \int_{\Omega} |\nabla I_2|^2 dx.
 \end{aligned} \tag{3.37}$$

$$\begin{aligned}
 I_4 &= 2 \int_{\Omega} m_4 d \Delta A (A - A^*) dx \\
 &= -2m_4 d \int_{\Omega} \nabla A \nabla (A - A^*) dx \\
 &= -2m_4 d \int_{\Omega} |\nabla A|^2 dx.
 \end{aligned} \tag{3.38}$$

Therefor, by (3.29), we have

$$I = -2m_1 a \int_{\Omega} |\nabla S|^2 dx - 2m_2 b \int_{\Omega} |\nabla I_1|^2 dx - 2m_3 c \int_{\Omega} |\nabla I_2|^2 dx - 2m_4 d \int_{\Omega} |\nabla A|^2 dx,$$

$$I = - \int_{\Omega} [2m_1 a |\nabla S|^2 + 2m_2 b |\nabla I_1|^2 + 2m_3 c |\nabla I_2|^2 + 2m_4 d |\nabla A|^2] dx < 0. \tag{3.39}$$

It is clear that $I \leq 0$ and $J \leq 0$ which leads to $\frac{dV}{dt} < 0$. Therefore, by Lyapunov's direct method, the endemic equilibrium (S^*, I_1^*, I_2^*, A^*) is globally asymptotically stable.

Conclusion

In this work, we built the HIV / AIDS model in PDE, we studied the overall asymptotic stability of endemic equilibria for HIV / AIDS. Use of the method of Lyapunov functions combined with the theory of stable matrices of **Volterra –Lyapunov**. Although the method of Lyapunov functions has been widely applied to a variety of dynamical systems, most of our analysis is based on the lesser-known results of stable **Volterra – Lyapunov** matrices.

BIBLIOGRAPHY

- [1] C. C. Chavez. Z. Feng. W. Huang, On the computation of R_0 and its role on global stability, in mathematical approaches for emerging and re-emerging infectious diseases: An introduction, IMA. Vol. 125. Springer, (2002).
- [2] K.O. Okosun. R. Ouifki. N. Marcus, Optimal control analysis of a malaria disease transmission model that includes treatment and vaccination with waning immunity. *Bio systems* 106 (2011).
- [3] J. Wang. S. Liao, A generalized cholera model and epidemic–endemic analysis, *J. Biol. Dynam*, 6 (2012).
- [4] A. Lajmanovich. J. Yorke, A deterministic model for gonorrhoea in a non-homogeneous population. *Biosci. Engrg*, 28 (1976).
- [5] F. Rinaldi, Global stability results for epidemic models with latent period, *IMA J. Appl. Med. Biol.* 7 (1990).
- [6] A. Tripathi. R. Naresh. D. Sharma, modelling the effect of screening of unaware infectives on the spread of HIV infection. *Comput*, 184 (2007).
- [7] R. Naresh. A. Tripathi. D. Sharma, modelling and analysis of the spread of AIDS epidemic with immigration of HIV infectives. *Comput. Model.* 49 (2009).
- [8] K. O. Okosun. O. D. Makinde. I. Takaidza, Impact of optimal control on the treatment of HIV/AIDS and screening of unaware infectives. *Model*, 37 (2013).
- [9] M. S. Zahedi. N. S. Kargar, The Volterra–Lyapunov matrix theory for global stability analysis of a model of the HIV/AIDS. P. O. Box. Tehran, iran.

- [10] R. Seroul, Stable polynomials, in programming for mathematicians. Berlin. Springer-Verlag, (2000).
- [11] S. Abdelmalek, S. Bendoukha, Global asymptotic stability of a diffusive SVIR epidemic model with immigration of individuals, (2016).
- [12] A. Turing, The Chemical basis of morphogenesis, philosophical transactions of the royal society of london. Series b. Biological sciences, Vol. 237, No. 641. Aug, 14 (1952).
- [13] V.P. Driessche. J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Biosci, 180 (2002).
- [14] R. Redheffer. J. SIAM.Volterra multipliers I Algebr. Discrete meth, 6 (1985).
- [15] R. Redheffer. J. SIAM.Volterra multipliers II Algebr. Discrete meth, 6 (1985).
- [16] A. Kandler, Roman unger population dispersal via diffusion-reaction equations.
- [17] Kh. Hattaf . N. Yousfi, Global stability for reaction–diffusion equations in biology, November (2013).
- [18] R. Casten. G. Charles. J. holland, Stability properties of solutions to systems of reaction-diffusion equations. System, J. Siam. Vol. 33. No, 2 september (1977).
- [19] C. Paul, Fife mathematical aspects of reacting and diffusing systems mathematics. Springer-Verlag berlin heidelberg. New York (1979).
- [20] M. Smith. L. Wang, Global dynamics of an SEIR epidemic model with vertical transmission. J. Siam. Anal, 62 (2001).
- [21] J. Yorke. A. Lajmanovich, A deterministic model for gonorrhoea in a non-homogeneous population. Biosci. Engrg, 28 (1976).
- [22] A. Kandler. R. Unger. J. Steele, Language shift, bilingualism and the future of britain’s celtic languages. Phil. Trans. R. Soc, B, (2010).
- [23] J. L. Lions, Equations differentielles operationnelles, Springer-Verlag, New York, (1961).
- [24] H. D. Arazoza. R. Lounes, A nonlinear model for a sexually transmitted disease with contact tracing, IMA J. Appl. Med. Biol, 19 (2002).

- [25] S. Busenberg. K. Cooke. H. Ying-Hen. A model for HIV in Asia. *Biosci. Engrg*, 128 (1995).
- [26] M. Doyle. D. Greenhalgh. S. Blythe, Equilibrium analysis of a mathematical model for the spread of AIDS in a two-sex population with mixing constraints, *J. Biol. Syst*, 6 (1998).
- [27] Marsudi, Sensitivity analysis of effect of screening and HIV therapy on the dynamics of spread of HIV. Vol, 8 (2014).
- [28] T. Kuniya. J. Wang, Lyapunov functions and global stability for a spatially diffusive SIR epidemic model, (2016).
- [29] S. Abdelmalek. S. Bendoukha, The Lengyel–Epstein reaction diffusion system, chapter. May (2019).
- [30] S. Abdelmalek, These of global existence of reaction -diffusion solution system via functional methods.
- [31] Y. Belbachir, Epidemiological model with the age of vaccination b. 12 juin (2019).
- [32] A. Tripathi. R. Naresh. D. Sharma, Modelling the effect of screening of unaware infectives on the spread of HIV infection. *Comput.* 184 (2007).