People's Democratic Republic of Algeria
Higher Education and Research Scientifique
Echahid Cheikh Larbi Tebessi University - Tebessa
Faculty of Exact Sciences, Natural and Life Sciences
Departement: Mathematics and Computer Sciences

End Of Study Thesis
For Obtaining The MASTER Diploma
Domaine: Mathematics and Computer Sciences
Track: Mathematics
Option: Partial differential equations and application

Theme

An analytic study of an epidemiological model as system of non-linear equations

Presented by:
Mellah Salsabil

Exammination Committee:

Mr. AbdelMalek Salem	*Pr University Larbi Tebessi*	*President*
Mr.Nabti Abderrazak	*MCA University Larbi Tebessi*	*Examiner*
Mr.Bouaziz Khelifa	*MCA University Larbi Tebessi*	*Supervisor*

Defense date : 04/06/2023

Abstract

The objective of this thesis is to introduce the field of epidemiology and its relationship with mathematics, as well as how it is modeled using partial differential equations. We specifically focus on the epidemic reaction-diffusion model for the spread of HIV, with the aim of studying the long-term stability of its solutions. We demonstrate that the model contains two types of equilibrium points for solving the proposed system, which describes the transmission of the infectious disease among individuals. The epidemic model is analyzed using the reproductive number, R_0. We study both local and global stability using the Jacobian matrix and the appropriate Lyapunov function. Finally, we present numerical examples of simulation processes that illustrate the findings discussed throughout the thesis.

Keywords: epidemiological, equilibrium points, reaction-diffusion, reproductive number R0, local stability, global stability.

الملخص

تهدف هذه المذكرة لتقديم مجال علم الاوبئة وعلاقته بالرياضيات، بالإضافة إلى كيفية نمذجته باستخدام المعادلات التفاضلية الجزئية. ونركز بشكل خاص على نموذج التفاعل والانتشار لانتشار فيروس الإيدز. وبهدف دراسة استقرار الحلول على المدى الطويل، تم التوصل الى ان النموذج يحتوي على نقطتي توازن لحل النظام المقترح، الذي يصف بدوره انتقال المرض بالعدوى بين الافراد، وتم تحليل النموذج الوبائي باستخدام معدل الانتاج الانجابي، حيث درسنا كل من الاستقرار المحلي والاستقرار الكلي باستخدام مصفوفة مناسبة ودالة ليابونوف. في النهاية، تم تقديم امثلة عددية لعمليات المحاكاة توضح النتائج المناقشة في جميع انحاء المذكرة.

الكلمات الرئيسية: علم الأوبئة، نقاط التوازن، معادلات تفاعل-انتشار، نسبة التكاثر، الاستقرار المحلي، الاستقرار الكلي.

Résumé

L'objectif de ce mémoire est de présenter le domaine de l'épidémiologie et sa relation avec les mathématiques, ainsi que la manière dont il est modélisé à travers des équations EDO, EDP. Nous abordons le modèle de diffusion de la réaction épidémique du VIH dans le but d'étudier la stabilité à long terme de ses solutions. Nous montrons que le modèle comporte deux types de points d'équilibre pour résoudre le système proposé, qui décrit la transmission de la maladie infectieuse entre les individus. Nous analysons le modèle épidémique en utilisant le nombre de reproduction de base R_0. Nous étudions la stabilité locale et globale en utilisant la matrice jacobienne et la fonction appropriée de Lyapunov. Enfin, nous présentons des exemples numériques de simulations qui clarifient et confirment les résultats de l'étude tout au long de ce mémoire.

Mots-clés: épidémiologique, points d'équilibre, réaction-diffusion, nombre de reproduction R_0, stabilité locale, stabilité globale.

Contents

Dedication

Praise be to Allah, with love, gratitude, and thanks. I could not have done this if it weren't for the grace of Allah. So, praise be to Allah for the beginning and the end.

{وَآخِرُ دَعْوَاهُمْ أَنِ الْحَمْدُ لِلَّهِ رَبِّ الْعَالَمِينَ}

I dedicate this success to everyone who has worked with me to accomplish this journey. You have been a constant support to me. I dedicate this research to my only sanctuary, my eternal joy, the one who paves the way for me to the source of my strength and pride, the one to whom I have always promised this success. Here I am fulfilling my promise and dedicating it to you, **my dear father**, may Allah keep him as a blessing for me.M

To the light of my days and the radiance of my life, to the one whose prayers have always included the noblest wishes, to the one whose heart saw me before her eyes and embraced me in her womb before her hands, to the shelter that I seek at all times, **my beloved mother**.

And to my inspirers, the makers of my strength, the elite of my days, and the solace of my moments, to the apple of my eye, **my sisters and brother**.

To those who spread joy and the spirit of childhood in our hearts, to the beauty of life, **my nieces and nephews.**

To those who walked my paths with me and made the hardships easier, to my refuge in this journey, and every time I stand at the threshold of fear, I find solace in you, **my friends**.

To **my ambitious self**, here you have achieved what was once a dream. Now, my prayer rug that witnessed my tears in the difficult nights bears witness to my joy and happiness.

And in conclusion, as it has been said, it was once a dream, then it became a possibility, and now it has become a reality, not just imagination. Praise be to Allah for the completion.

Acknowledgment

In the name of Allah, the Most Gracious, the Most Merciful, to whom all praise is due.

The research conducted within this thesis has been carried out at the esteemed University of Cheikh Larbi Tebessi in Tebessa, specifically within the Department of Mathematics and Computer Science.

First and foremost, I express my heartfelt gratitude to our esteemed teachers for imparting the necessary knowledge throughout our educational journey.

I would like to express my gratitude to the entire academic community whose pursuit of knowledge and passion for discovery has inspired and motivated me every step of the way. It is through your collective efforts that we continue to push the boundaries of human understanding and advance the frontiers of knowledge.

I extend my deepest appreciation to my supervisor, **Dr. Khelifa BOUAZIZ,** who directed me throughout this challenging yet exhilarating journey of completing my thesis, you have been my guiding , mentor, and pillar of support. Your unwavering dedication, expertise, and unrelenting belief in my abilities have propelled me forward and shaped me into the scholar I am today. Your guidance has extended far beyond the realm of academia. Your constant encouragement, patience, and willingness to listen have provided me with the strength and confidence to overcome obstacles, push boundaries, and strive for excellence. Your unwavering commitment to my personal and professional growth has been nothing short of remarkable, and I am forever indebted to you for the invaluable lessons I have learned under your tutelage.

I am deeply honored by the presence of **Pr. Salem Abdelmalek**, who graciously accepted the role of the jury president, and **Dr. Nabti Abderrazak**, who kindly agreed to review this research.

Additionally, I would like to express my gratitude to my colleagues and friends at the university, as well as all those who hold a special place in my heart. I reserve a special mention for my beloved parents, whose sacrifices, kindness, and unwavering support have been a constant source of strength.

Introduction

Mathematics has been utilized for 350 years in the study and combat of infectious diseases, with recent decades seeing significant advances in the field of mathematical epidemiology. These illnesses now pose fresh difficulties that need for mathematical modeling that combines medical and mathematical viewpoints. Epidemiology, medicine, biology, and mathematics all cross in the field of mathematical modeling, which uses equations to show a condensed version of reality. Pandemics and epidemics have caused tremendous harm to humanity throughout history, frequently leading to profound changes and the probable collapse of civilizations. [3], [13].

In recent years, scientists have worked to create mathematical models that are more and more realistic and answer ever-more-complex issues. The nature of the problems under investigation and the accessibility of more detailed and accurate data are the causes of this complexity. In general, mathematical models provide a condensed understanding of reality by formalizing complicated occurrences and making it easier to examine numerous factors and their relationships. These models contribute to a preliminary understanding of the systems under investigation by producing and testing hypotheses.

Furthermore, mathematical models [12] serve the primary purpose of predicting events across diverse situations, finding particular use in the field of communicable disease epidemiology through various models based on differential equations or probabilistic approaches. Epidemiological models play a vital role in comprehending the spread of infectious diseases and predicting future outcomes. Analytical studies of epidemiological models, involving the analysis of disease transmission dynamics and considering factors such as population size and disease criteria, examine the mathematical behavior and characteristics of epidemics. Non-linear equations, such as reaction-diffusion models, describe the interaction between epidemiological variables like the number of infected, susceptible, exposed, and recovered individuals. Analyzing such systems involves investigating equilibrium points and determining their stability, there by enabling the inference of long-term epidemic behavior. These studies primarily focus on disease control and prevention.

The SIS epidemiology model model has captured the interest of many researchers[2],[4], so in this research, I conducted an analytical study of an SIS epidemiological model specifically targeted at HIV. The study involved a comprehensive analysis of the disease's dynamics within a population, wherein the SIS model represents individuals as susceptible to HIV infection or infected and capable of transmitting the virus. By mathematically analyzing the model's solutions, stability, and other characteristics, the objective was to understand the behavior of HIV epidemics and identify effective disease control strategies. This analytical approach allowed for the exploration of equilibrium points, determination of their stability, and prediction of long-term trends

in HIV transmission. The insights gained from such studies are valuable in designing interventions, prevention programs, and public health policies aimed at mitigating the spread of HIV and reducing its impact on affected communities.
My thesis consists of three chapters:

- The first chapter provides an overview of epidemiology as a field and some of its mathematical models, and the factors R_0 which help to determine whether a disease will spread or disappear.
- The second chapter presents some definitions and concepts that we will used later.
- In the last chapter, firstly, we begin by formulating the model proposed in this study based on the proposed assumptions. Subsequently, we mathematically analyze the model, particularly (focusing on the equilibrium points without disease and the endemic point), then we prove the stability of the system model, then we presents a numerique simulation.

List of Figures

List of Tables

List of Abbreviation and Symbols

- The set of the real numbers, is denoted by $\mathbb{R}$.
- The set of the positive real numbers, is denoted by $\mathbb{R}^+$.
- The set of the real numbers of the n-elements $\mathbb{R}^n$.
- The basic reproductive number, is denoted by $\mathbb{R}_0$.
- The determinant of real and complex matrices, is denoted by $\det(A)$.
- The trace of real and complex matrices, is denoted by $tr(A)$.
- The inverse of real and complex matrices is denoted by A^{-1}.
- The spectral radius of matrix A is denoted by $\rho(A)$.
- Identify matrix denoted by I.
- $||.||$ a norm ecludien.
- The Laplacian operator A, is denoted by ΔA.

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

- Omega an open domain in $\mathbb{R}^n$ where $n > 1$, denoted by Ω.
- The gradient A is denoted by ∇A.

$$\nabla A = \left(\frac{\partial A}{\partial x_1}, \frac{\partial A}{\partial x_2}, ..., \frac{\partial A}{\partial x_n} \right).$$

- (PDE) The partial differential equations.
- (ODE) The ordinary differential equations.
- (DFE) Free-disease equilibrium.
- (EE) Endemic equilibrium.

Chapter 1

An Introduction to Epidemiology

In this chapter we introduce the epidemiology which is a study of how diseases spread and affect populations, aiming to understand patterns, risk factors, and control measures. In mathematics, epidemiology uses models to quantify disease transmission dynamics, analyzing variables such as the reproductive number R_0 which represents the average number of new infections caused by each infected individual. The reproductive number helps predict the potential for outbreaks and guide public health interventions.

1.1 Description of the epidemiology

1.1.1 Epidemic definition

"Epidemic" [6] is a word composed of two Greek words "epi" which means "on" or "among" and "demos" which means "people" or "population", its first use in English was in the late 16^{th} centuary, exactly in 1580, according to the Oxford English Dictionary.
Epidemiology is study of epidemics, this word was uused in 1830 for the first time by the french physician Louis-René Villermé, but it was populazed by another one, named Dr. Pierre Charles Alexandre Louis who use this term at the Parisian School of medcin and it was gradually adopted into English language, when we talk about epidemiology, you think that it is only about medcin, but it is bigger than that.

1.1.2 Epidemiology

is field of study that deals with the determinants occurrence and distribution of deisease in a population. This study aims to understand, control and pevent the spread of diseases and to

adress complex health problems.
There are three main techniques in epidemiology [11] :

Descriptive Epidemiology

It describes the distribution of diseases among individuals and the health phenomenon by time, place and characteristics of the population. It helps to make hypotheses about disease risk factors.

Analytic Epidemiology

It is the second method, it works for analyzes disease determinants. And it contain two methods: The case-control and the cohort one.

Experimental Epidemiology

In which the hypothesis is developed and the factors are addressed in a experimental model to confirm or refute the hypotheses.

1.2 Mathematical Epidemiology

Mathematical epidemiology is a research field and it is considered a powerful tool for managing infectious disease in the population through mathematical modeling and statistical analysis.

1.2.1 Mathematical modeling

Mathematical modeling is process of creating mathematical representation of a matter or system in the real world with the use of mathematical equations to describe the behaviour and relationships of the variables involved in the system.
It can be used in making predictions, testing hypotheses- and it can be applied to a wide range of fields such as Physics, Engeneering, Economics, Biology and Social sciences. It helps researchers understand complex systems, design optimal solutions.
There are serval steps to obtaining mathematical modeling: identifying the problem, collecting data, formulating hypotheses, selecting appropiate mathematical instruments, testing and improving the model, as well as using the model to draw conclusions or make predictions. It is a process of collaboration between mathematicians, scientists and field experts.

1.2.2 Epidemiological model

It is one of the mathematical models used to study the spread of infectious diseases and manage them in the population. It uses mathematical and statistical techniques to learn how infectious diseas is transmitted and developed within a particular population.
These models can be used to predict the future course of the outbreak and identify the main factors affecting the spread of the disease.
Epidemiological models in mathematics typically include diffeeremtial equations and probability theory, and statisistical methods. these are just a few examples of many types of epidemiological models that use mathematical methods to understand and control infectious disease outbreaks.
There many types including compartmental models, stochastic models, spatial models .... and network models.

1.2.3 Compartmental models

Compartmental models are the most commonly used type of epidemiological model, they devide the population into different compartments based on their disease status, such as susceptible, exposed, infected and recovred individuals.[17]

SI, SIS models:

SI model: The SI model is one of the classical models that was created by W. Hamar and developed in 1906. It is a simple one. It devides individuals into two compartments:
-The compartment of susceptible individuals (healthy) who are receptive to the infectious agent but are not contaminated and can catch the disease and become contagious, they become infectious when dealing with ann infectious individual, noted as (S).
-The compartment of infected individuals noted as (I), who are affected and therefore infectious.
Infection spreads through direct contact between susceptibles and infected individuals. In this model, there are no recoveries and is only relevant in incurable diseases or if the phenomenon of acquired immunity can be neglected. An individual changes state that is mean when they infected, they remain infectious for the duration of the disease and do not recover. However, as the number of infected individuals changes over time.
We assume that the population is constant and we model it as follows:

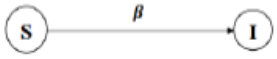

Figure 1: SI Model

we represent the SI model by system of differential equations:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI. \end{cases}$$

with β is the transmission rate of the disease.

SIS model: The acronym SIS stands for "Susceptible-Infectious-Susceptible", the SIS model is similar to the SI one, the difference is that in SIS model there are cases where a susceptible individual becomes infected and the infected ones recover at a certain rate but do not develop immunity and become susceptible again, thus there is no immunity conferred by previous infection, s in the case of tuberculosis.
The scheme of the SIS model:

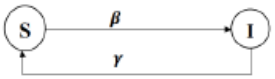

Figure 2: SIS Model

The basic SIS model can be expressed mathemtically as follows:

$$\begin{cases} \frac{dS}{dt} = -\beta SI + \gamma I, \\ \frac{dI}{dt} = \beta SI - \gamma I. \end{cases}$$

γ is the rate at which infected individuals return to the susceptible state due to loss of immunity (The rate at which each infected person recovers).

SIR, SIRS models:

SIR model: The SIR is a model of disease dynamics was proposed in 1927 by Kermack and McKendrick; consisting of three categories of population:

-The healthy people S.
-The infected people I.
-The recovered or cured people R, who have acquired immunity against reinfection or death.
The SIR model can be used to make predictions about the spread of infectious diseases and to evaluate the effectiveness of different intervention strategies, such as vaccination or social distancing measures.
We can model it as follows:

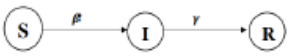

Figure 3: SIR Model

The SIR model consists of three differential equations that describe the changes in the number of individuals in each category over time. The equations are:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI - \gamma I, \\ \frac{dR}{dt} = \gamma I. \end{cases}$$

β is the transmission rate of the disease, which represents the probability that an infected individual will transmit the disease to a susceptible individual.
γ is the recovery rate of the disease, which represents the rate at which infected individuals recover and become immune to the disease.

SIRS model The SIRS model differs from SIR model with a litle thing. The difference is that the SIR assumes that individuals who recover from an infectious disease become immune for life, hile the SIRS model assumes that immunity wanes over time, and the individual does not acquire permanent immunity and lose it and that recovered individuals become susceptible to the disease again and returns to the S compartment at a rate of η , it is often used to study diseases such as measles.
it is represented by the SIRS model as follows:

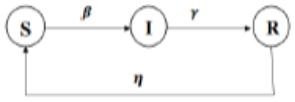

Figure 4: SIRS Model

It can written by:

$$\begin{cases} \frac{dS}{dt} = -\beta SI + \eta R, \\ \frac{dI}{dt} = \beta SI - \gamma I, \\ \frac{dR}{dt} = \gamma I - \eta R. \end{cases}$$

η The rate of loss of immunity (probability of a recovered individual becoming susceptible again per unit time).

SEI, SEIR models:

SEI model: The SEI epidemic model is a compartmental model used to understand the spread of infectious diseases in a population. It divides the population into three compartments:
- S includes individuals who are at risk of contracting the disease.
- E (exposed) icludes individuals that are already infected but not yet contagious (non-infectious), meaning that the susceptible populations move to class I after a period of latency, during which they become infectious. This latency period is called the incubation period.
- I includes individuals who are currently infected and can spread the disease to others.
It can modeled as follows:

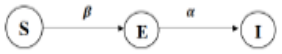

Figure 5: SEI Model

The equation is:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \alpha E, \\ \frac{dI}{dt} = \alpha E. \end{cases}$$

β The transmission rate, which determines the rate at which susceptible individuals become exposed.
α The incubation rate, which determines the rate at which exposed individuals become infectious.

SEIR model: The SEIR model is more comprehensive than the SEI model because it considers the possibility of recovered individuals becoming susceptible again in the future, and it can be used to study the long-term dynamics of infectious diseases.
It represented by:

Figure 6: SEIR Model

and by:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \alpha E, \\ \frac{dI}{dt} = \alpha E - \gamma I, \\ \frac{dR}{dt} = \gamma I. \end{cases}$$

γ is the time between becoming infectious and recovering.

1.3 Basic reproductive number

R_0 is a very important parameter in mathematical epidemiology because it can help predict how quickly a disease will spread and how effective control measures will be in slowing or stopping its spread.. It represents the average number of people who will contract a disease from one infected person in a population where everyone is susceptible to the disease [19].
The value of R_0 assumes NO pre-existing immunity (everyone is susceptible) meaning, no individuals are immunized (naturally or through vaccination) i.e No one has been exposed to the disease before and no one has been vaccinated against the disease.
The value of R_0 indicates whether a disease tends to disappear or persist, depending on its value.
The Values of R_0 are depends on 3 Factors:

1. Infection Period.

2. Contact rate.

3. Mode of transmission.

We don't actually know the true value of R_0 until the outbreak is over.

Values of R_0 :

It has 3 options:

When the R_0 is less than 1, the number of new cases will decline over time, eventually leading to the end of the outbreak without the need for intervention. (One person transmits the infection to less than one person).

Example 1.1 *we put $R_0 = 0.25$ (this model is for illustration only). The number of cases decreases over time.*

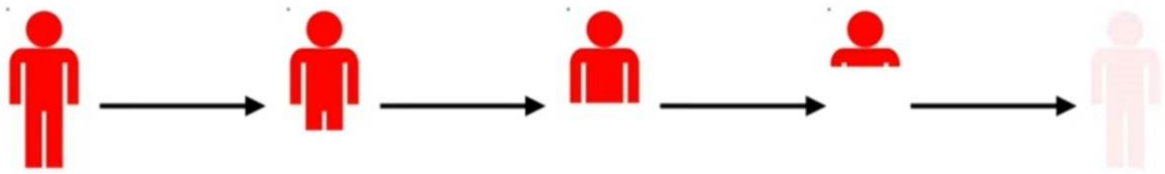

Figure 7: $R_0 = 0.25$

When the R_0 is equal to 1, the number of cases will remain stable, neither increasing nor decreasing. (One person transmits the infection to one person).

Example 1.2 $R_0 = 1$. *The number of cases remains stable over time.*

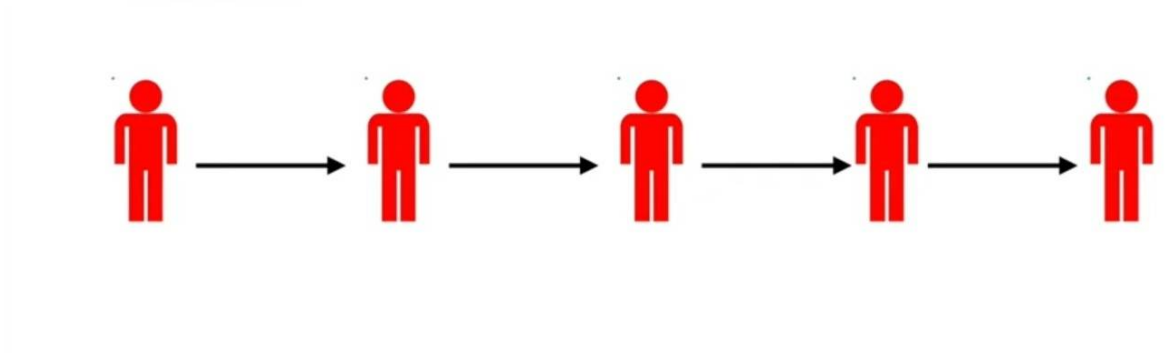

Figure 8: $R_0 = 1$

However, when the R_0 is greater than 1, the outbreak is self-sustaining, meaning that the number of cases will continue to increase unless effective control measures are implemented to reduce transmission. (One person transmits the infection to more than one person).

Example 1.3 $R_0 = 2$. *The number of cases increases over time.*

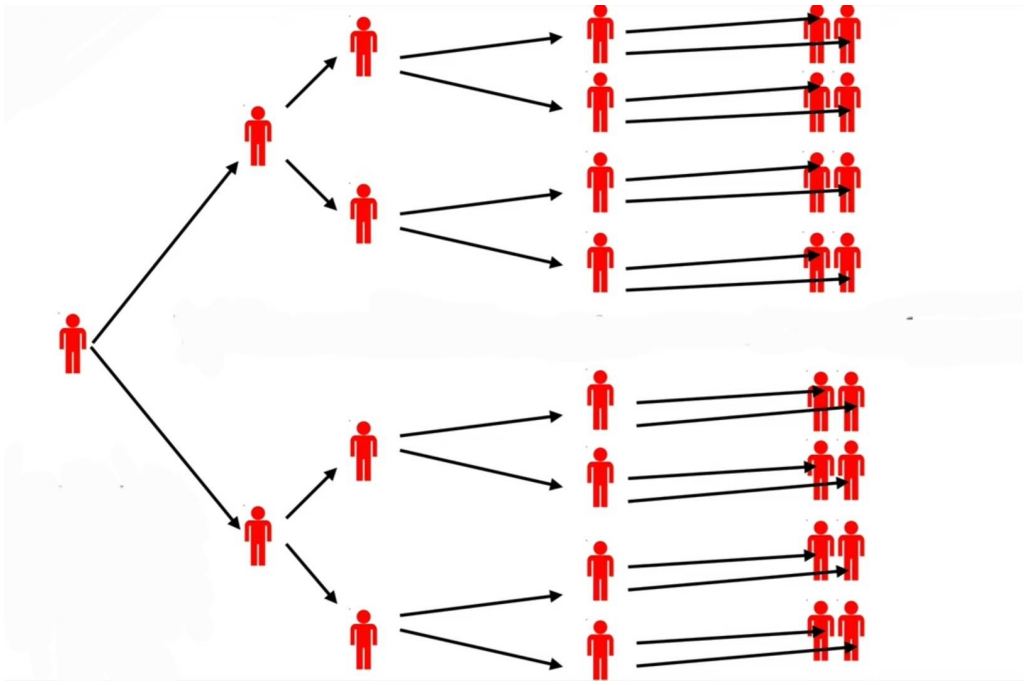

Figure 9: $R_0 = 2$.

We don't actually know the true value of R_0 until the outbreak is over.

1.3.1 Calcul methods

The reproductive number, or R_0, can be calculated using mathematical models that take into account different factors:

The Anderson and May method:

It was developed by R. M. Anderson and R. M. May in the 1980s and is widely used in the field of infectious disease epidemiology.

$$R_0 = \beta \times C \times D,$$

with
β: The probability of disease transmission.
D: The number of contacts.
C: The average duration of the infectious period.

The Van den Driessche and Watmough method:[5]

Either F, V two matrices

$$F = \begin{pmatrix} f_1(S, U_1, U_2) \\ f_2(S, U_1, U_2) \end{pmatrix},$$

$$V = \begin{pmatrix} \upsilon_1(U_1, U_2) \\ \upsilon_2(U_1, U_2) \end{pmatrix}.$$

Derivatives of f_1, f_2 with respect to U_1 and U_2 respectively:

$$F = \begin{pmatrix} \frac{\partial f_1(S,U_1,U_2)}{\partial U_1} & \frac{\partial f_1(S,U_1,U_2)}{\partial U_2} \\ \frac{\partial f_2(S,U_1,U_2)}{\partial U_1} & \frac{\partial f_2(S,U_1,U_2)}{\partial U_2} \end{pmatrix}.$$

Derivatives of υ_1, υ_2 with respect to U_1 and U_2 respectively:

$$V = \begin{pmatrix} \frac{\partial \upsilon_1(U_1,U_2)}{\partial U_1} & \frac{\partial \upsilon_1(U_1,U_2)}{\partial U_2} \\ \frac{\partial \upsilon_2(U_1,U_2)}{\partial U_1} & \frac{\partial \upsilon_2(U_1,U_2)}{\partial U_2} \end{pmatrix}.$$

Then, we calculate the inverse matrix of V, we get

$$V^{-1} = \frac{1}{\frac{\partial \upsilon_1(U_1,U_2)}{\partial U_1}\frac{\partial \upsilon_2(U_1,U_2)}{\partial U_2} - \frac{\partial \upsilon_1(U_1,U_2)}{\partial U_2}\frac{\partial \upsilon_2(U_1,U_2)}{\partial U_1}} \begin{pmatrix} \frac{\partial \upsilon_2(U_1,U_2)}{\partial U_2} & -\frac{\partial \upsilon_1(U_1,U_2)}{\partial U_2} \\ -\frac{\partial \upsilon_2(U_1,U_2)}{\partial U_1} & \frac{\partial \upsilon_1(U_1,U_2)}{\partial U_1} \end{pmatrix}.$$

Then we calculate the matrix (FV^{-1}).The basic reproductive number R_0 is then defined as the spectral radius of the Jacobian matrix. The spectral radius refers to the maximum absolute value of the eigenvalues of the matrix.

$$\begin{aligned} \rho\left(FV^{-1}\right) &= \max\left\{\left|\lambda, \lambda \in \sigma\left(FV^{-1}\right)\right|\right\}, \\ \rho\left(FV^{-1}\right) &= R_0. \end{aligned}$$

In the context of epidemiology, the eigenvalues represent the growth rates of different infection states.

Chapter 2

Reaction-diffusion system and stability in epidemiology

This chapter focus in concepts that we need in later chapter. It gives the meaning of reaction-diffusion models which are used in epidemiology to describe the spread of infectious diseases. They incorporate the interplay between local interactions (reactions) and spatial movement (diffusion) of individuals, then the equilibrium points and for what we use them, and the meaning of stability analysis which helps determine the conditions under which disease-free or endemic equilibria emerge, aiding in understanding disease dynamics and control strategies.

2.1 Reaction-diffusion

Reaction diffusion systems [16] of partial differential equations are highly significant in modeling various real-life applications and have been extensively studied by scientists and academics for decades, particularly in the last twenty years. These mathematical models are used to represent physical phenomena such as changes in the concentration of chemical substances over time and space, the spread of infections among populations and the diffusion of substances across surfaces. These equations arise as models for the densities of substances or organisms that disperse through space by various mechanisms and react to each other and their surroundings. Reaction diffusion models are deterministic, but can be derived from stochastic processes, and are analyzed using methods from the theory of partial differential equations and dynamical systems. These models allow us to translate assumptions about stochastic local movement into deterministic descriptions of global densities.

In ecology, reaction diffusion models are used to study population dynamics and can describe the existence of a minimal patch size needed to sustain a population, the propagation of wave fronts

corresponding to biological invasions, and the formation of spatial patterns in the distributions of populations. These models treat space and time as continuous and are spatially explicit.
Reaction-diffusion models are spatially explicit, describe population densities and treat space and time as continuous. They describe how the concentration of two or more chemical species changes over time and space due to their reactions and diffusion and are used to study pattern formation and morphogenesis in nature, such as the formation of spots and stripes on animal coats or the branching patterns of blood vessels.
In the context of epidemics, reaction-diffusion systems have been widely used to model the spread of epidemics, which involves the diffusion of infectious agents, such as viruses or bacteria, and the interactions between infected and susceptible individuals. In these models, the chemical species represent the infected and susceptible individuals and the diffusion term represents the movement of individuals between neighboring regions. The reaction term describes the transmission of the disease from infected to susceptible individuals and the recovery or death of infected individuals.
One of the most widely used reaction-diffusion models for studying epidemics is the Kermack-McKendrick model, which assumes that the population is divided into three compartments: susceptible individuals, infected individuals and recovered individuals. This model incorporates the effects of the infection rate, the recovery rate and the diffusion of the disease among different regions.
Reaction-diffusion models can be used to explore the effects of various control strategies on the spread of epidemics, such as vaccination, quarantine and social distancing. They can also be used to investigate the impact of environmental factors, such as temperature and humidity, on the transmission of infectious agents.
Overall, reaction-diffusion models provide a valuable tool for understanding the complex dynamics of epidemics and for informing public health policies and strategies for controlling their spread.
The concept of **diffusion** is rooted in the physical sciences, and is defined as the phenomenon where a group of particles spreads according to the erratic motion of each individual particle. This results in a spread that is always directed from regions of higher concentration to regions of lower concentration. The time dependence of the distribution of the particles in space can be mathematically described by the diffusion equation, which is the formulation of the spread dynamics. The diffusion theory aims to explain the spread behavior of a group of particles, rather than just a single particle. This theory can be applied to various fields, such as physics, biology, social sciences, etc. In biology, diffusion is used to describe processes of biodiffusion and population dynamics, as well as the spread of infectious diseases among populations. Similarly, in social sciences, diffusion can be used to describe the spread of ideas, innovations, or lexical

terms. A **reaction** refers to a process that involves a change in the state or configuration of a system. This change can be in the form of a chemical reaction, a nuclear reaction, or a physical reaction. In general, a reaction involves the conversion of one set of substances or particles into another set, which may involve the release or absorption of energy. Examples of reactions in physics include combustion reactions, radioactive decay, and phase transitions.

2.1.1 Reaction diffusion model

Once we have explored various methods for modeling reproduction and dispersion, either for infection, prey, or population in isolation, we then investigate the population dynamic by combining both mechanisms. Our aim is to observe the temporal and spatial behavior of the population size while considering different growth models such as exponential and logistic growth. We focus on diffusion-reaction systems, where the population can grow and disperse simultaneously. We assume Neumann boundary conditions in the [16] form of:

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

In this context, we have a function $U(x,t)$ which represents the population size at any point x and time t, where x is a spatial variable and t is a temporal variable. The operator Δ is the Laplacian operator, which represents the movement of the population from regions of high density to regions of low density. The matrix D is a diagonal matrix of coefficients $(d_1, d_2, ..., d_m)$, and the change in the population size over time at a given location x is described by the diffusion component $D\Delta U(x,t)$, where D is the diffusion coefficient. The growth component is represented by the function $F = (f_0, f_1, ..., f_m)$, which determines the rate of population growth at each location.

One example of a reaction-diffusion system for modeling epidemics is the Kermack-McKendrick model, which assumes that the population is divided into three compartments: susceptible individuals (S), infected individuals (I), and recovered individuals (R). The model's equations are given by:

$$\begin{cases} \frac{\partial S}{\partial t} = d_1\Delta S - \beta SI, \\ \frac{\partial I}{\partial t} = d_2\Delta I + \beta SI - \gamma I, \\ \frac{\partial R}{\partial t} = d_3\Delta R + \gamma I, \end{cases}$$

where β is the infection rate, γ is the recovery rate, and d_1, d_2, and d_3 are the diffusion coefficients of the three compartments. The first equation describes the movement of susceptible individuals, the second describes the movement of infected individuals, and the third describes the movement of recovered individuals.
The Kermack-McKendrick model exhibits a spatial spread of the epidemic, where the disease spreads from infected regions to susceptible regions through the diffusion term. The model can also incorporate other factors such as vaccination, quarantine, and social distancing to study the effectiveness of different control strategies.

2.2 Equilibrium points

An equilibrium point, or a steady state, is a point in a system where the system remains unchanged at the equilibrium point. In other words, the net change in the system's state variables is zero, many stability problems are naturally formulated with respect to equilibrium points [14].

Definition 2.1 *A state x^* is an equilibrium state (or equilibrium point) of the system if once $x(t)$ is equal to x^* , it remains equal to x^* for all future time. This means*

$$f(x^*) = 0,$$

after we solve this nonlinear algebraic equations we can found the equilibrium points. A linear time-invariant system

$$\dot{x} = Ax.$$

If A is nonsingular that is mean the system has a single equilibrium point. If A is singular, it has an infinity of equilibrium point. A nonlinear system can have several (or infinitely many) isolated equilibrium points.

2.2.1 Disease-free equilibria

It is a special state in which the entire population is free from the infectious disease under consideration, meaning that it refers to a state in which the disease is completely eradicated from the population, and there are no infected individuals present. It represents a stable equilibrium point at which the number of infected individuals is zero $(I = 0)$.

2.2.2 Endemic equilibria

The term refers to a stable and persistent state of disease prevalence in a population. It represents the long-term equilibrium point at which the number of infected individuals remains constant

over time, the number of infected individuals is grater than zero $(I > 0)$[15].

2.3 Stability in epidemiology

It indicates the long-term behaviours and equilibrium states of infectious diseases within the population. So to understand if the spread of the disease will continue at a constant level, increase to epidemic proportions, or die out completely.
it is commonly employed in epidemiological models, like compartmental models (SI, SIS, SEI, SIR, SEIR) to study thr equilibrium points in which they represent steady states where the disease prevalence remains constant over time.
The stability properties can be categorized as follows:
Stable: the disease prevalence will remain constant over time. Small perturbations or introductions of infections from outside the system will dampen out, and the system will return to the equilibrium state.
Unstable: Small perturbations in the disease prevalence can have a profound impact over time, leading to significant deviations from the equilibrium. These deviations can manifest as either a rapid escalation in disease transmission, causing an epidemic, or a complete elimination of the disease.
Asymptotic stability: The asymptotic stability of an equilibrium point in epidemiology refers to a state where the system not only returns to the equilibrium state but also approaches it with either an exponential or logarithmic rate. This characteristic implies that the disease prevalence will remain constant and gradually converge towards a specific level over time.
The stability can be local or global, [15] local stability focuses on the behavior of the system in the immediate vicinity of an equilibrium point, while global stability considers the behavior of the system across its entire state space. Local stability analysis relies on linearization and eigenvalue analysis, while global stability analysis often involves more sophisticated techniques like Lyapunov functions. Determining global stability provides stronger guarantees about the long-term behavior of the system under all possible conditions, but it can be more challenging to establish than local stability.

2.3.1 Local stabillity

Jaccobian matrix which is a matrix of first-order partial derivatives. In the realm of differential equations and dynamical systems, the Jacobian matrix assumes a vital role in approximating nonlinear systems around equilibrium points. When dealing with a system of equations that

encompasses multiple variables, the Jacobian matrix characterizes the localized behavior of the system near an equilibrium.
If we have this system of equations:

$$\begin{cases} f_1(x_1, x_2, ..., x_n) = 0, \\ f_2(x_1, x_2, ..., x_n) = 0, \\ \qquad\qquad ... \\ f_n(x_1, x_2, ..., x_n) = 0. \end{cases}$$

Its Jacobian Matrix denoted by J is given by:

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \dots & \frac{\partial f_1}{\partial x_n} \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \dots & \frac{\partial f_2}{\partial x_n} \\ \dots & \dots & \dots & \dots \\ \frac{\partial f_n}{\partial x_1} & \frac{\partial f_n}{\partial x_2} & \dots & \frac{\partial f_n}{\partial x_n} \end{pmatrix}.$$

By evaluating the Jacobian matrix at an equilibrium point, you can obtain valuable information about the stability properties of the system by examining the eigenvalues of the matrix. The system is asymptotic stable if all the real part for all eigenvalues is negative (we use it more detailled in the next chapter).

2.3.2 Global stability

Proving global stability [14] in epidemiology is often more challenging than demonstrating local stability. global stability refers to demonstrating that an equilibrium point in an epidemiological model is not only locally stable but also stable for all possible initial conditions and perturbations. It ensures that the disease prevalence will converge to the equilibrium state from any starting point in the system's state space. It usually involves the use of Lyapunov functions, which are scalar functions that measure the energy or "distance" of the system from the equilibrium. A Lyapunov function is defined such that it decreases over time, reaching a minimum at the equilibrium point.
So to establish global stability, the Lyapunov function must be definite positive (it is positive for all points in the state space except at the equilibrium point where it is zero), and its derivate with respect to time must be negative or zero.
We define this function as follow:

Theorem 2.1 ***(Lyapunov Function).*** *[18] Let x^* be an equilibrium solution of the equation:*

$$x' = f(x(t)).$$

Let Ω be a neighborhood of x^* contained in U, and let $V : \Omega \to \mathbb{R}$ be a C^1 class function such that:

- $V(x^*) = 0$,
- $\forall\, x \in \Omega \backslash \{x^*\}, V(x) > 0$,
- $\forall\, x \in \Omega, V'(x) \leq 0$.

Then, x^* is stable.
V named Lyapunov function.

Theorem 2.2 ***(Strict Lyapunov Function)***. *Let x^* be an equilibrium solution of the equation:*

$$x' = f(x(t)).$$

Let Ω be a neighborhood of x^* included in U, and let $V : \Omega \to \mathbb{R}$ be a C^1 class function such that:

- $V(x^*) = 0$.
- $\forall\, x \in \Omega \backslash \{x^*\}, V(x) > 0$.
- $\forall\, x \in \Omega, V'(x) < 0$.

Then, x^* is asymptotically stable.
V named Strict **Lyapunov function.**

Chapter 3

Modelling and Mathematical analysis for a diffusive epidemic model

In recent years, there has been a growing interest among researchers in the study of infectious diseases, with the aim of improving treatment and reducing mortality rates through predicting the spread of diseases. One type of model that has been proposed and studied extensively is the susceptible-infected-susceptible (SIS) epidemic reaction-diffusion model. Researchers have explored various approaches to modeling disease transmission, including the standard incidence transmission term $\beta SI/N$, which was proposed as an alternative to mass action. Some researchers proposed a frequency-dependent SIS reaction-diffusion model for a population living in a continuous spatial habitat, and some of them discussed the global stability of the endemic equilibrium in some special cases for this model. Over the years, researchers have discovered many important and interesting properties of these models, such as the reproductive number R_0 and the global stability of the disease-free equilibrium.

Our study focuses on the reaction-diffusion system given by the following equations [2]:

$$\begin{cases} \frac{\partial s}{\partial t} - d_1 \Delta s = \Lambda - \beta \frac{s\varphi(i)}{s+i} - \mu s, \text{ in } \mathbb{R}^+ \times \Omega, \\ \frac{\partial i}{\partial t} - d_2 \Delta i = \beta \frac{s\varphi(i)}{s+i} - (\mu + \sigma) i, \text{ in } \mathbb{R}^+ \times \Omega. \end{cases} \tag{3.1}$$

The diffusion coefficients in our model are represented by the positive constants d_1 and d_2, while the Laplacian operator on Ω is denoted by the symbol Δ. Here, $\Omega \subset \mathbb{R}^n$ is a bounded open subset of $\mathbb{R}^n$ with a smooth boundary $\partial\Omega$. Furthermore, we assume that the initial condition:

$$s(x,0) = s_0(x), \qquad i(x,0) = i_0(x), \text{ in } \Omega, \tag{3.2}$$

and homogeneous Neumann boundary conditions:

$$\frac{\partial s}{\partial \eta} = \frac{\partial i}{\partial \eta} = 0, \quad \text{in } \mathbb{R}^+ \times \Omega. \tag{3.3}$$

The nonlinearity φ is assumed to be a nonnegative and continuously differentiable function on $\mathbb{R}^+$ such that:

$$\varphi(0) = 0, \tag{3.4}$$

and

$$0 < i\varphi'(i) \leq \varphi(i) \text{ for all } i > 0. \tag{3.5}$$

Mathematical modeling in biology is an essential tool for improving our understanding of epidemiological patterns and disease control, particularly for infectious diseases which continue to target large populations and remain a leading cause of mortality . One example of a mathematical model is the system proposed to describe the transmission of HIV in a population consisting of susceptible individuals s and infected individuals i. The model parameters include Λ for the flow rate of newly exposed individuals, μ for the death rate, β for the rate of disease prevalence among individuals per unit time, and σ, which is given by $\sigma = \mu + \eta$ and $\frac{1}{\eta}$is the mean period of sexual activity of affected individuals. It is important to note that diseases such as stroke, coronary heart disease, and infectious diseases remain among the leading causes of death worldwide.
The present study has successfully established the system models (3.1-3.3) and corresponding parameter descriptions.

3.1 Properties of the model

The subsequent sections of this paper will introduce the solution to the primary problem, define the basic reproductive number R_0, and establish its connection to the local stability of the system. However, prior to delving into these topics, it is important to introduce a lemma, which we will prove to be valuable in later sections.

Lemma 3.1 *Condition (3.3-3.5) implies*

$$0 < \frac{\varphi(i)}{i} \leq \varphi'(0) \textit{ for all } i > 0. \tag{3.6}$$

Proof. *Starting with the inequality*

$$0 < i\varphi'(i) \leq \varphi(i) \textit{ for all } i > 0,$$

we can divide both sides by i to obtain

$$0 < \varphi\prime(i) \leq \frac{\varphi(i)}{i}.$$

Since $\varphi(i)$ is continuously differentiable with $\varphi(0) = 0$, we know that $\varphi'(0)$ exists. Thus, we can use the mean value theorem to obtain

$$\varphi'(0) = \frac{\varphi(i) - \varphi(0)}{i - 0},$$

so

$$\varphi(i) = \varphi(0) + i\varphi'(0).$$

Dividing both sides by i, we have

$$\frac{\varphi(i)}{i} = \varphi'(0).$$

Now, since $\varphi(i)$ is nonnegative and $\varphi(0) = 0$, we have

$$0 \leq \frac{\varphi(i)}{i} \leq \frac{\varphi(i) - \varphi(0)}{i - 0} = \frac{\varphi(i)}{i},$$

we get

$$0 < \varphi'(i) \leq \frac{\varphi(i)}{i} \leq \varphi'(0).$$

■

3.2 Existence of equilibria

The objective of this section is to demonstrate the presence of steady state solutions for equations (3.1)-(3.3) and compute the fundamental basic reproduction number R_0. The system of equations (3.1-3.3) can be simplified into the following set of ordinary differential equations (ODEs)

$$\begin{cases} \frac{ds}{dt} = \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s, \text{ in } \mathbb{R}^+, \\ \frac{di}{dt} = \beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i \text{ in } \mathbb{R}^+, \end{cases} \tag{3.7}$$

with initial conditions:

$$s_0(x) > 0, \quad i_0(x) \geq 0. \tag{3.8}$$

Proposition 3.1

- *If $R_0 < 1$, the system (3.7) accepts one equilibrium point E_0.*
- *If $R_0 < 1$, the system (3.7) has two equilibrium points E_0 and E^*.*

Proof. The positive equilibria of syste, (3.7) - (3.8) satisfies

$$\begin{cases} \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s = 0, \\ \beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i = 0. \end{cases} \tag{3.9}$$

First, if $i = 0$ it becomes:

$$\begin{cases} \Lambda - \beta\frac{s\varphi(0)}{s+0} - \mu s = 0, \\ \beta\frac{s\varphi(0)}{s+0} - (\mu+\sigma)\times 0 = 0, \end{cases}$$

so

$$s = \frac{\Lambda}{\mu}.$$

We get that the system (3.9) has only equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$. Then, we take $i > 0$ to study endemic steady state conditions, from adding the two equations in the system (3.9) we get

$$\begin{aligned} \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s + \beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i = 0, \\ \Lambda - (\mu+\sigma)i = \mu s \ , \\ s = \frac{\Lambda}{\mu} - \frac{(\mu+\sigma)i}{\mu}. \end{aligned} \tag{3.10}$$

By substituting in the first equation.We start by $s + i$:

$$\begin{aligned} s+i &= \frac{\Lambda-(\mu+\sigma)i}{\mu} + i, \\ &= \frac{\Lambda - \mu i - \sigma i + \mu i}{\mu} \\ &= \frac{\Lambda - \sigma i}{\mu}, \end{aligned}$$

then

$$\begin{aligned} \frac{s}{s+i} &= \frac{\Lambda-(\mu+\sigma)i}{\mu} \times \frac{\mu}{\Lambda-\sigma i} \\ &= \frac{\Lambda-(\mu+\sigma)i}{\Lambda-\sigma i}, \end{aligned}$$

we substite in the first equation:

$$\begin{aligned} \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s &= 0, \\ \Lambda - \beta\varphi(i)\frac{\Lambda-(\mu+\sigma)i}{\Lambda-\sigma i} - \Lambda + (\mu+\sigma)i &= 0, \end{aligned}$$

we get

$$\begin{aligned}
\frac{\beta\varphi(i)}{(\mu+\sigma)i} &= \frac{\Lambda-\sigma i}{\Lambda-(\mu+\sigma)i},\\
\frac{\beta\varphi(i)}{(\mu+\sigma)i}[\Lambda-(\mu+\sigma)i] &= \Lambda-\sigma i,\\
\frac{\Lambda\beta\varphi(i)}{(\mu+\sigma)i}-\beta\varphi(i) &= \Lambda-\sigma i,\\
\frac{\beta\varphi(i)}{(\mu+\sigma)i} &= \frac{\Lambda-\sigma i+\beta\varphi(i)}{\Lambda}.
\end{aligned}$$

$$h(i)=\frac{\beta\varphi(i)}{(\mu+\sigma)i}-[1-\frac{\sigma i}{\Lambda}+\frac{\beta\varphi(i)}{\Lambda}],$$
$$h(i)=0 \text{ for any } i>0, \tag{3.11}$$

$h(i)$ is continuous for any $i>0$. By using the intermediate value theorem, there exists a real $i^*\in(0,\frac{\Lambda}{\sigma})$ such that (3.11) holds.

$$\begin{aligned}
\lim_{i\to 0}h(i) &= \lim_{i\to 0}\frac{\beta\varphi(i)}{(\mu+\sigma)i}-[1-\frac{\sigma i}{\Lambda}+\frac{\beta\varphi(i)}{\Lambda}],\\
&= \frac{\beta\varphi'(0)}{(\mu+\sigma)}-[1-\frac{\sigma(0)}{\Lambda}+\frac{\beta\varphi(0)}{\Lambda}],\\
&= \frac{\beta\varphi'(0)}{(\mu+\sigma)}-1>0,\\
&= R_0-1>0,
\end{aligned}$$

and

$$\begin{aligned}
\lim_{i\to\frac{\Lambda}{\sigma}} h(i) &= \lim_{i\to\frac{\Lambda}{\sigma}} \frac{\beta\varphi(i)}{(\mu+\sigma)i} - [1 - \frac{\sigma i}{\Lambda} + \frac{\beta\varphi(i)}{\Lambda}], \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{(\mu+\sigma)\frac{\Lambda}{\sigma}} - [1 - \frac{\sigma(\frac{\Lambda}{\sigma})}{\Lambda} + \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda}], \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{(\mu+\sigma)\frac{\Lambda}{\sigma}} - \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda} \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\mu(\frac{\Lambda}{\sigma})+\Lambda} - \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda} \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda(1+\frac{\mu}{\sigma})} - \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda} \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma}) - \beta\varphi(\frac{\Lambda}{\sigma})(1+\frac{\mu}{\sigma})}{\Lambda(1+\frac{\mu}{\sigma})} \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda}(\frac{1-1-\frac{\mu}{\sigma}}{1+\frac{\mu}{\sigma}}) \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda}(\frac{-\frac{\mu}{\sigma}}{\frac{\sigma+\mu}{\sigma}}) \\
&= \frac{\beta\varphi(\frac{\Lambda}{\sigma})}{\Lambda}(\frac{-\mu}{\sigma+\mu}) < 0.
\end{aligned}$$

If the derivative of h is negative for all values of i greater than zero, then the function is monotonically decreasing in that interval using (3.4)-(3.6).

$$\begin{aligned}
\frac{dh(i)}{di} &= \frac{\beta\varphi'(i)(\mu+\sigma)i - (\mu+\sigma)\beta\varphi(i)}{((\mu+\sigma)i)^2} - (\frac{-\sigma}{\Lambda} + \frac{\beta\varphi(i)}{\Lambda}) \\
&= \frac{\beta i\varphi'(i) - \beta\varphi(i)}{(\mu+\sigma)i^2} + \frac{1}{\Lambda}(\sigma - \beta\varphi'(i)) \\
&= \frac{\beta(i\varphi'(i) - \varphi(i))}{(\mu+\sigma)i^2} + \frac{1}{\Lambda}(\sigma - \beta\varphi'(i)) < 0.
\end{aligned}$$

Consequently, there exists a unique real i^* within the interval $(0, \frac{\Lambda}{\sigma})$ for which $h(i^*) = 0$. This condition implies the existence of a unique $s^* = \frac{\Lambda}{\mu} - \frac{(\mu+\sigma)i^*}{\mu}$. As a result, we can conclude that the proof is complete. ■

3.3 Basic reproductive number R_0 of the model

The basic reproductive number R_0 can be defined as the spectral radius of the matrix FV^{-1} [9]-[7]

So, we can write the systems (3.7)-(3.8) in the following vector form

$$\begin{pmatrix} i_t \\ s_t \end{pmatrix} = \begin{pmatrix} \beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i \\ \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s \end{pmatrix} = \begin{pmatrix} \beta\frac{s\varphi(i)}{s+i} \\ 0 \end{pmatrix} - \begin{pmatrix} (\mu+\sigma)i \\ -\Lambda + \beta\frac{s\varphi(i)}{s+i} + \mu s \end{pmatrix}.$$

The Jacobian matrices associated with the vectors $\begin{pmatrix} \beta\frac{s\varphi(i)}{s+i} \\ 0 \end{pmatrix}$ and $\begin{pmatrix} (\mu+\sigma)i \\ -\Lambda + \beta\frac{s\varphi(i)}{s+i} + \mu s \end{pmatrix}$ at the disease-free equilibrium $E_0 = \left(\frac{\Lambda}{\mu}, 0\right)$ are provided as follows

$$J_1(s,i) = \begin{pmatrix} \frac{[\beta s\varphi'(i)](s+i)-s\varphi(i)}{(s+i)^2} & \frac{[\beta\varphi(i)](s+i)-s\varphi(i)}{(s+i)^2} \\ 0 & 0 \end{pmatrix}.$$

Then, we have

$$J_1(E_0) = \begin{pmatrix} \beta\varphi'(0) & 0 \\ 0 & 0 \end{pmatrix} = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix},$$

Moreover, we have

$$J_2(s,i) = \begin{pmatrix} \mu+\sigma & 0 \\ \frac{[\beta s\varphi'(i)](s+i)-s\varphi(i)}{(s+i)^2} & \frac{[\beta\varphi(i)](s+i)-s\varphi(i)}{(s+i)^2} + \mu \end{pmatrix}.$$

$$J_2(E_0) = \begin{pmatrix} \mu+\sigma & 0 \\ \beta\varphi'(0) & \mu \end{pmatrix}, = \begin{pmatrix} V & 0 \\ V_1 & V_2 \end{pmatrix}.$$

Calculating V^{-1}

$$V^{-1} = \frac{1}{VV_2}\begin{pmatrix} V_2 & 0 \\ -V_1 & V \end{pmatrix} = \begin{pmatrix} \frac{1}{V} & 0 \\ \frac{-V_1}{VV_2} & \frac{1}{V_2} \end{pmatrix}.$$

Then FV^{-1}

$$\begin{aligned} FV^{-1} &= \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{V} & 0 \\ \frac{-V_1}{VV_2} & \frac{1}{V_2} \end{pmatrix} \\ &= \begin{pmatrix} \frac{F}{V} & 0 \\ 0 & 0 \end{pmatrix}. \end{aligned}$$

R_0 is the spectral radius of a matrix is the maximum absolute value among all the eigenvalues of the matrix.

To determine the spectral radius of a this matrix, we need to calculate its eigenvalues and then identify the one with the highest absolute value,

$$det(FV^{-1} - \lambda I) = 0.$$

$$\begin{pmatrix} \frac{F}{V} - \lambda & 0 \\ 0 & -\lambda \end{pmatrix} = 0.$$

$$\begin{aligned} \left(\frac{F}{V} - \lambda\right)(0 - \lambda) - 0 &= 0 \\ \lambda(\lambda - \frac{F}{V}) &= 0. \end{aligned}$$

The eigenvalues are the values of λ that satisfy this equation which are $\lambda_1 = 0$ and $\lambda_2 = \frac{F}{V}$ the hightest value is λ_2 so the reproductive number R_0 is given by:

$$\begin{aligned} R_0 &= \rho(FV^{-1}) \\ &= \frac{F}{V}. \end{aligned}$$

$$R_0 = \frac{\beta\varphi'(0)}{\mu + \sigma}. \tag{3.12}$$

3.4 Positivity of solutions

When the initial data (3.2) satisfy the following conditions:

$$s(x,0) = s_0(x) > 0, \quad i(x,0) = i_0(x) \geq 0, \text{ in } \Omega. \tag{3.13}$$

By applying the maximum principle to system (3.1)-(3.3), it can be deduced that the functions $s(t,x)$ and $i(t,x)$ satisfy certain conditions

$$s(t,x) > 0, \quad i(t,x) \geq 0, \quad \forall (t,x) \in (0, T_{\max}) \times \Omega. \tag{3.14}$$

3.5 Existence of solutions

The proposition below establishes that solutions of system (3.1)-(3.3) exists globally over time span, and these solutions remain bounded by parameter-dependent constant. This proposition holds under the assumption that the function satisfies conditions (3.4) and (3.5).

Proposition 3.2 *Assume that the initial data $s_0, i_0 \in C\left(\bar{\Omega}\right)$ and fulfill (3.13), and also φ satisfies (3.4) and (3.5). Then the solution (s, i) of system (3.1)-(3.3) exists uniquely and globally in time. Moreover, (3.14) holds for $T_{\max} = +\infty$, as well as ther exists a constant $A\left(s_0, i_0, \Lambda, \beta, \mu, \sigma\right) > 0$, such that*

$$\left\|s\left(\cdot, t\right)\right\|_{L^\infty(\Omega)} + \left\|i\left(\cdot, t\right)\right\|_{L^\infty(\Omega)} \leq A, \quad \textit{for all } t > 0. \tag{3.15}$$

Furthermore, there exists a positive constant $\tilde{A}\left(\Lambda, \beta, \mu, \sigma\right)$ such that for a large $T > 0$,

$$\left\|s\left(\cdot, t\right)\right\|_{L^\infty(\Omega)} + \left\|i\left(\cdot, t\right)\right\|_{L^\infty(\Omega)} \leq \tilde{A}, \ \textit{for all } t > T. \tag{3.16}$$

Proof. Let $s\left(t, x\right) \in \left(0, T_{\max}\right) \times \Omega$, we can formulate the first part of the local solution for system (3.1) which given by:

$$\begin{cases} \frac{\partial s}{\partial t} - d_1 \Delta s = \Lambda - \beta \frac{s\varphi(i)}{s+i} - \mu s, \text{in } \left(0, T_{\max}\right) \times \Omega\,, \\ s\left(0, x\right) = s_0\left(x\right), \qquad\qquad \text{on } \Omega\,, \\ \frac{\partial s}{\partial \nu} = 0, \qquad\qquad \text{on } \left(0, T_{\max}\right) \times \partial\Omega. \end{cases} \tag{3.17}$$

For any positive function $i\left(t, x\right) \in \left(0, T_{\max}\right) \times \Omega$, there is an upper solution exists for (3.17) which is provided by:

$$C_1 = \max\left\{\frac{\Lambda}{\mu}, \left\|s_0\right\|_{C(\bar{\Omega})}\right\}.$$

By using the comparison principle, $s\left(t, x\right) \leq C_1$ in $\left[0, T_{\max}\right) \times \bar{\Omega}$, which is uniformly bounded . We consider

$$\tilde{\chi} = \int_\Omega \left(s\left(x, t\right) + i\left(x, t\right)\right) dx.$$

Using (3.1)-(3.3):

$$\begin{aligned} \frac{d}{dt}\tilde{\chi}\left(t\right) &= \Lambda\left|\Omega\right| - \int_\Omega \left(\mu s\left(x, t\right) + \left(\mu + \sigma\right) i\left(x, t\right)\right) dx \\ &\leq \Lambda\left|\Omega\right| - \mu\tilde{\chi}\left(t\right). \end{aligned} \tag{3.18}$$

Using Gronwall's inequality, and for $t \in \left(0, T_{\max}\right)$, we have

$$\tilde{\chi}\left(t\right) \leq C_2, \tag{3.19}$$

which is greater than 0.

$$i(t,\cdot) \in L^1(\Omega). \tag{3.20}$$

Using the second equation of (3.1), there exists $C_3 > 0$, depends on C_2 such that $i(t,x) \leq C_3$ in $[0, T_{\max}) \times \bar{\Omega}$, also by using the standard theory of semilinear parabolic systems, we deduce $T_{\max} = \infty$.
and when $T_{\max} = +\infty$, problem (3.17) becomes

$$\begin{cases} \frac{\partial s}{\partial t} - d_1 \Delta s = \Lambda - \beta \frac{s\varphi(i)}{s+i} - \mu s \leq \Lambda - \mu s, & \text{in } (0,+\infty) \times \Omega, \\ s(0,x) = s_0(x) \leq \|s_0\|_{C(\bar{\Omega})}, & \text{on } \Omega, \\ \frac{\partial s}{\partial \nu} = 0, & \text{on } (0,\infty) \times \partial\Omega. \end{cases} \tag{3.21}$$

We use the the comparison principle to find that $s(t,x) \leq \omega(t)$ where $\omega(t) = \|s_0\|_{C(\bar{\Omega})} e^{-\mu t} + \left(\frac{\Lambda}{\mu}\right)(1 - e^{-\mu t})$ which is the unique solution of the initial value problem

$$\begin{cases} \frac{d\omega}{dt} = \Lambda - \mu\omega, \ t > 0, \\ \omega(0) = \|s_0\|_{C(\bar{\Omega})}. \end{cases} \tag{3.22}$$

Then for $x \in \bar{\Omega}$, we have

$$s(t,x) \leq \omega(t) \longrightarrow_{t\to\infty} \frac{\Lambda}{\mu}.$$

Thus, we have an upper bound for $\|s(t,\cdot)\|_{L^\infty(\Omega)}$ independent of the initial data for a given sufficiently large t, and we found that $\|i(t,\cdot)\|_{L^\infty(\Omega)}$ also bounded by a positive constant independent of the initial data for a large enough t. ■

3.6 The local stability of ODE

In the case of ODE we study the system in the absence of diffusion.
We now turn our attention to analyzing the local asymptotic stability of two equilibrium points: the disease-free equilibrium point E_0 and the endemic equilibrium point E^*, as outlined in the following proposition.

Proposition 3.3 1. *If $R_0 < 1$ the disease-free equilibrium point E_0 is locally asymptotically stable.*

2. *If $R_0 > 1$, E_0 is unstable and the endemic equilibrium point $E^*$is locally asymptotically stable.*

Proof. In order to establish the local asymptotic stability, we utilize the Jacobian matrix, which given as follows.

$$J(s,i) = \begin{pmatrix} -\mu - \beta\frac{i\varphi(i)}{(s+i)^2} & -\beta\frac{s\varphi'(i)(s+i)-s\varphi(i)}{(s+i)^2} \\ \beta\frac{i\varphi(i)}{(s+i)^2} & \beta\frac{s\varphi'(i)(s+i)-s\varphi(i)}{(s+i)^2} - (\mu+\sigma) \end{pmatrix}. \tag{3.23}$$

First of all, at E_0, we have

$$\begin{aligned} J(E_0) &= \begin{pmatrix} -\mu - \beta\frac{(0)\varphi(0)}{(\frac{\Lambda}{\mu}+0)^2} & -\beta\frac{(\frac{\Lambda}{\mu})\varphi'(0)(\frac{\Lambda}{\mu}+0)-(\frac{\Lambda}{\mu})\varphi(0)}{(\frac{\Lambda}{\mu}+0)^2} \\ \beta\frac{(0)\varphi(0)}{(\frac{\Lambda}{\mu}+0)^2} & \beta\frac{(\frac{\Lambda}{\mu})\varphi'(0)(\frac{\Lambda}{\mu}+0)-(\frac{\Lambda}{\mu})\varphi(0)}{(\frac{\Lambda}{\mu}+0)^2} - (\mu+\sigma) \end{pmatrix}, \\ &= \begin{pmatrix} -\mu & -\beta\frac{(\frac{\Lambda}{\mu})^2\varphi'(0)}{(\frac{\Lambda}{\mu})^2} \\ 0 & \beta\frac{(\frac{\Lambda}{\mu})^2\varphi'(0)}{(\frac{\Lambda}{\mu}+0)^2} - (\mu+\sigma) \end{pmatrix}, \\ &= \begin{pmatrix} -\mu & -\beta\varphi'(0) \\ 0 & \beta\varphi'(0) - (\mu+\sigma) \end{pmatrix}, \end{aligned}$$

then, we can calculate the eigenvalues of $J(E^*)$ as follows

$$\det(J(E_0) - \lambda I) = 0,$$

$$\det\begin{pmatrix} -\mu-\lambda & -\beta\varphi'(0) \\ 0 & \beta\varphi'(0) - (\mu+\sigma) - \lambda \end{pmatrix} = 0,$$

we get

$$(-\mu-\lambda)\left[\beta\varphi'(0) - (\mu+\sigma) - \lambda\right] = 0,$$

so we can show that the eigenvalues are

$$\begin{aligned} \lambda_1 &= -\mu < 0 \text{ because } \mu > 0, \\ \lambda_2 &= \beta\varphi'(0) - (\mu+\sigma),\ \lambda_2 < 0 \text{ only when } R_0 < 1, \end{aligned} \tag{3.24}$$

and it leqds to asymptotic stability result.

In the second case it is clearly that the equilibrium E_0 is unstable when $R_0 > 1$, but the system possesses an equilibrium point E^*.

Evaluating the matrix (3.23) at E^*, we obtain

$$J(E^*) = \begin{pmatrix} -\mu - \beta\frac{i^*\varphi(i^*)}{(s^*+i^*)^2} & -\beta\frac{s^*\varphi'(i^*)(s^*+i^*)-s^*\varphi(i^*)}{(s^*+i^*)^2} \\ \beta\frac{i^*\varphi(i^*)}{(s^*+i^*)^2} & \beta\frac{s^*\varphi'(i^*)(s^*+i^*)-s^*\varphi(i^*)}{(s^*+i^*)^2} - (\mu+\sigma) \end{pmatrix}. \tag{3.25}$$

We put

$$X = \beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2}, \tag{3.26}$$

and

$$Y = \beta \frac{s^* \varphi'(i^*)(s^* + i^*) - s^* \varphi(i^*)}{(s^* + i^*)^2}. \tag{3.27}$$

So, we get

$$J(E^*) = \begin{pmatrix} -\mu - X & -Y \\ X & Y - (\mu + \sigma) \end{pmatrix}. \tag{3.28}$$

The equilibrium E^* is locally asymptotically stable if $tr\left(J(E^*)\right) < 0$ and $\det\left(J(E^*)\right) > 0$.
We start by calculating the $tr\left(J(E^*)\right)$

$$tr\left(J(E^*)\right) = -\mu - X + Y - (\mu + \sigma).$$

We have

$$\begin{aligned} \beta \frac{s^* \varphi(i^*)}{s^* + i^*} - (\mu + \sigma) i^* &= 0, \\ \beta \frac{s^* \varphi(i^*)}{s^* + i^*} &= (\mu + \sigma) i^* \\ (\mu + \sigma) &= \beta \frac{s^* \varphi(i^*)}{(s^* + i^*) i^*}, \end{aligned}$$

and from (3.5), we have

$$\begin{aligned} 0 &< i\varphi'(i) \leq \varphi(i) \\ \varphi'(i) &\leq \frac{\varphi(i)}{i}, \end{aligned}$$

So, we use them as follows

$$\begin{aligned} -X + Y - (\mu + \sigma) &= -\beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2} + \beta \frac{s^* \varphi'(i^*)(s^* + i^*) - s^* \varphi(i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*) i^*}, \\ &= -\beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2} + \beta \frac{s^* \varphi'(i^*)(s^* + i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*) i^*} \\ &= -\beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2} + \beta \frac{s^* \varphi'(i^*)}{(s^* + i^*)} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*) i^*} \\ &\leq -\beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2} + \beta \frac{s^* \varphi(i^*)}{i^* (s^* + i^*)} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*) i^*} \\ &\leq -\beta \frac{i^* \varphi(i^*)}{(s^* + i^*)^2} - \beta \frac{s^* \varphi(i^*)}{(s^* + i^*)^2} \\ &\leq -\beta \frac{\varphi(i^*)}{(s^* + i^*)^2} (s^* + i^*), \\ &\leq -\beta \frac{\varphi(i^*)}{(s^* + i^*)} < 0, \end{aligned} \tag{3.29}$$

so we find that :

$$tr\left(J(E^*)\right) = -\mu - X + Y - (\mu + \sigma) < 0,$$

we move to the determinant of the Jacobian which is given by:

$$\begin{aligned}
\det\left(J(E^*)\right) &= (-\mu - X)\left(Y - (\mu + \sigma)\right) - (-YX) \\
&= (-\mu - X)\left(Y - (\mu + \sigma)\right) + YX \\
&= -\mu Y + \mu(\mu + \sigma) - XY + X(\mu + \sigma) + YX \\
&= \mu\left(X - Y + \sigma + \mu\right) + \sigma X.
\end{aligned}$$

We can see easily (3.29) that the $\det\left(J(E^*)\right) > 0$, so the endemic equilibrium E^* is locally asymptotically stable. ■

3.7 The local stability of PDE

In the case of PDE, we study the system in the presence of diffusion.
So we investigate the local stability of more general partial differential equations (PDE) cases (3.1)-(3.3).

Theorem 3.1 *For system (3.1)*

1. *If $R_0 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable.*
2. *If $R_0 > 1$, the endemic equilibrium $E^*$is locally asymptotically stable.*

Proof. We have the system in the PDE case which satisfaies the equilibrim points which given by:

$$\begin{cases} d_1\Delta s + \Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s = 0, \text{ in } \mathbb{R}^+ \times \Omega, \\ d_2\Delta i + \beta\frac{s\varphi(i)}{s+i} - (\mu + \sigma)i = 0, \text{ in } \mathbb{R}^+ \times \Omega. \end{cases} \tag{3.30}$$

subject to the homogeneous Neumann boundary condition $\frac{\partial i}{\partial \nu} = \frac{\partial s}{\partial \nu} = 0$, in $\mathbb{R}^+ \times \Omega$[1].
Let $0 = \lambda_0 < \lambda_1 \leq \lambda_2 \leq \ldots$. be the sequence of eigenvalues for the elliptic operator $(-\Delta)$ subject to the homogeneous Neumann boundary condition on Ω, where each λ_j has multiplicity $m_j \geq 1$. Also let Φ_{jh} , $1 \leq h \leq m_j$, ($\Phi_0 = const$ and $\lambda_j \to \infty$ at $j \to \infty$) be the normalized eigenfunctions corresponding to λ_j. That is, Φ_{jh} and λ_j satisfy $-\Delta\Phi_{jh} = \lambda_j\Phi_{jh}$ in Ω, with $\partial\Phi_{jh}\partial\nu = 0$ in $\partial\Omega$, and $\int_\Omega \Phi_{jh}^2(x)dx = 1$.

we start by the first equilibrium point E_0.
Its Jacobian matrix given by:

$$L\left(E_0\right)=\begin{pmatrix} d_1\Delta-\mu & -\beta\varphi'(0) \\ 0 & d_2\Delta+\beta\varphi'(0)-(\mu+\sigma) \end{pmatrix}.$$

Similar to the ODE case, the asymptotic stability can be determined by examining the eigenvalues of the operator L if they have negative real parts. we suppose $(\Upsilon(x),\Psi(x))$ is an eigenfunction of L corresponding to an eigenvalue ξ,
we have

$$L(\Upsilon(x),\Psi(x))^t=\xi(\Upsilon(x),\Psi(x))^t.$$

Give us

$$(L-\xi I)\begin{pmatrix}\Upsilon\\ \Psi\end{pmatrix}=\begin{pmatrix}0\\0\end{pmatrix}.$$

leading to:

$$\sum_{0\leq j\leq\infty,1\leq h\leq m_j}(J_j-\xi I)\begin{pmatrix}a_{jh}\\ b_{jh}\end{pmatrix}\Phi_{jh}=\begin{pmatrix}0\\0\end{pmatrix}.$$

where:

$$\Upsilon=\sum_{0\leq j\leq\infty,1\leq h\leq m_j}a_{jh}\Phi_{jh},\qquad \Psi=\sum_{0\leq j\leq\infty,1\leq h\leq m_j}b_{jh}\Phi_{jh},$$

and

$$J_j\left(E_0\right)=\begin{pmatrix} -d_1\lambda_j-\mu & -\beta\varphi'(0) \\ 0 & -d_2\lambda_j+\beta\varphi'(0)-(\mu+\sigma) \end{pmatrix},\ \text{for all } j. \tag{3.31}$$

We can easily shown that the eigenvalues of the matrix are given for all $i\geq 0$ by:

$$\begin{cases} k_{1j}=-d_1\lambda_j-\mu, \\ k_{2j}=-d_2\lambda_j+\beta\varphi'(0)-(\mu+\sigma). \end{cases}$$

Given that the Laplacian eigenvalues are positive and arranged in ascending order, it is evident that both k_{1j} and k_{2j} possess negative real parts when $R_0<1$. As a result, it is lead to the local stablility of E_0.
Then we move to the second equilibrium E^* satisfies (3.3)-(3.30). Its corresponding linearization operator is

$$L\left(E^*\right)=\begin{pmatrix} d_1\Delta-\mu-X & -Y \\ X & d_2\Delta+Y-(\mu+\sigma) \end{pmatrix}.$$

The values of X and Y are taken from (3.26) and (3.27), the stability of E* rests on the negativity of the real parts of the eigenvalues of matrice:

$$J_j\left(E^*\right) = \begin{pmatrix} -d_1\lambda_j - \mu - X & -Y \\ X & -d_2\lambda_j + Y - (\mu+\sigma) \end{pmatrix},$$

the trace is as follows

$$\begin{aligned} tr\left(J_j\left(E^*\right)\right) &= -d_1\lambda_j - \mu - X - d_2\lambda_j + Y - (\mu+\sigma) \\ &= -\lambda_j\left(d_1+d_2\right) - \mu + (-X + Y - (\mu+\sigma) < 0. \end{aligned}$$

So $tr\left(J_j\left(E^*\right)\right) < 0$.
The determinant of the Jacobian is given by:

$$\begin{aligned} \det\left(J_j\left(E^*\right)\right) &= \left[-d_1\lambda_j - \mu - X\right]\left[-d_2\lambda_j + Y - (\mu+\sigma)\right] + XY, \\ &= d_1d_2\lambda_j^2 - d_1\lambda_j + (\mu+\sigma)d_1\lambda_j + \mu d_2\lambda_j - \mu Y + \mu(\mu+\sigma) + Xd_2\lambda_j - XY \\ &\quad +(\mu+\sigma)X + XY, \\ &= d_1d_2\lambda_j^2 + [-d_1Y + (\mu+\sigma)d_1 + \mu d_2 + Xd_2]\lambda_j + \mu[-Y + \mu + \sigma + X] + \sigma X \\ &= d_1d_2\lambda_j^2 + [-d_1Y + (\mu+\sigma)d_1 + \mu d_2 + Xd_2]\lambda_j + \det J(E^*). \end{aligned}$$

We put that

$$\begin{aligned} H_0 &= -d_1Y + (\mu+\sigma)d_1 + \mu d_2 + Xd_2, \\ &= d_1(-Y + (\mu+\sigma)) + d_2(\mu + X) \\ &= d_1\left(-\beta\frac{s^*\varphi'(i^*)(s^*+i^*) - s^*\varphi(i^*)}{(s^*+i^*)^2} + (\mu+\sigma)\right) + d_2(\mu + \beta\frac{i^*\varphi(i^*)}{(s^*+i^*)^2}\Big). \end{aligned}$$

Keeping in mind that (s^*, i^*) are solutions of system (3.1), so we get:

$$\begin{cases} \Lambda = \beta\frac{s^*\varphi(i^*)}{s+i^*} + \mu s^* = (\mu+\sigma)i^* + \mu s^*, \\ (\mu+\sigma)i^* = \beta\frac{s^*\varphi(i^*)}{s^*+i^*}, \end{cases} \tag{3.32}$$

and from (3.5) and (3.32), we get

$$\begin{aligned} H_0 &\geq d_1(-\beta\frac{s^*\varphi(i^*)(s^*+i^*)}{i^*(s^*+i^*)^2} + \beta\frac{s^*\varphi(i^*)}{(s^*+i^*)^2} + \beta\frac{s^*\varphi(i^*)}{i^*(s^*+i^*)}) + d_2(\mu + \beta\frac{i^*\varphi(i^*)}{(s^*+i^*)^2}). \\ &\geq d_1\beta\frac{s^*\varphi(i^*)}{(s^*+i^*)} + d_2(\mu + \beta\frac{i^*\varphi(i^*)}{(s^*+i^*)^2}) > 0, \end{aligned}$$

which is leading to $\det\left(J_j\left(E^*\right)\right) > 0$. Hence, E^* is locally asymptotically stable. ■

3.8 Global stability

We have chosen to analyze $R_0 < 1$ and $R_0 > 1$ separatefy in order to examine the global stability that is depends on the reproductive number R_0.

3.8.1 Global asymptotic stability for $R_0 < 1$

Theorem 3.2 *If $R_0 < 1$, E_0 is a globally asymptotically stable for system (3.1), whith*

$$\varphi'(0) \leq \frac{\Lambda}{\beta\left(\theta\frac{\Lambda}{\mu} + \frac{2\Lambda}{\mu+\sigma}\right)}, \tag{3.33}$$

and

$$\theta > \frac{(d_1+d_2)^2}{4d_1d_2}. \tag{3.34}$$

Proof. Let:

$$F_\theta(t) = \int_\Omega \left[is + \frac{\theta}{2}\left(s - \frac{\Lambda}{\mu}\right)^2 + \frac{1}{2}i^2 + 2\frac{\Lambda}{\mu+\sigma}i\right]dx,$$

we have to show that $F_\theta(t)$ is a Lyapunov function.
At $E_0 = \left(\frac{\Lambda}{\mu}, 0\right), F_\theta(t) = 0$.
When $\frac{\Lambda}{\mu} \neq 0$ we will prove that $F_\theta(t) > 0$.
Calculating the derivate

$$\begin{aligned}
\dot{F}_\theta(t) &= \int_\Omega (\frac{\partial s}{\partial t}i + \frac{\partial i}{\partial t}s)dx + \theta\int_\Omega \frac{1}{2}\frac{\partial s}{\partial t}\left(s - \frac{\Lambda}{\mu}\right) + \frac{\partial s}{\partial t}\left(s - \frac{\Lambda}{\mu}\right) + \int_\Omega \frac{1}{2}\frac{\partial i}{\partial t}idx + \frac{1}{2}\frac{\partial i}{\partial t}idx \\
&\quad +2\frac{\Lambda}{\mu+\sigma}\int_\Omega \frac{\partial i}{\partial t}dx. \\
&= \int_\Omega (\frac{\partial s}{\partial t}i + \frac{\partial i}{\partial t}s)dx + \theta\int_\Omega \frac{1}{2}\times 2\frac{\partial s}{\partial t}\left(s - \frac{\Lambda}{\mu}\right) + \int_\Omega \frac{1}{2}\times 2\frac{\partial i}{\partial t}idx + 2\frac{\Lambda}{\mu+\sigma}\int_\Omega \frac{\partial i}{\partial t}dx \\
&= \int_\Omega (\frac{\partial s}{\partial t}i + \frac{\partial i}{\partial t}s)dx + \theta\int_\Omega \frac{\partial s}{\partial t}\left(s - \frac{\Lambda}{\mu}\right) + \int_\Omega \frac{\partial i}{\partial t}idx + 2\frac{\Lambda}{\mu+\sigma}\int_\Omega \frac{\partial i}{\partial t}dx.
\end{aligned}$$

By replacing the values of the partial derivatives $\frac{\partial s}{\partial t}$ and $\frac{\partial i}{\partial t}$ with their corresponding values from equation (3.1), we can obtain the following expression:

$$\begin{aligned}
\dot{F}_\theta(t) &= \int_\Omega \frac{\partial s}{\partial t} i dx + \int_\Omega \frac{\partial i}{\partial t} s dx + \theta \int_\Omega \frac{\partial s}{\partial t}\left(s - \frac{\Lambda}{\mu}\right) dx + \int_\Omega \frac{\partial i}{\partial t} i dx \\
&\quad + 2\frac{\Lambda}{\mu+\sigma}\int_\Omega \frac{\partial i}{\partial t} dx \\
&= \int_\Omega \left[i + \theta\left(s - \frac{\Lambda}{\mu}\right)\right] \frac{\partial s}{\partial t} dx + \int_\Omega \left[s + i + 2\frac{\Lambda}{\mu+\sigma}\right] \frac{\partial i}{\partial t} dx \\
&= \int_\Omega \left[i + \theta\left(s - \frac{\Lambda}{\mu}\right)\right] \left(d_1 \Delta s + \Lambda - \beta \frac{s\varphi(i)}{s+i} - \mu s\right) dx \\
&\quad + \int_\Omega \left[s + i + 2\frac{\Lambda}{\mu+\sigma}\right] \left(d_2 \Delta i + \beta \frac{s\varphi(i)}{s+i} - (\mu+\sigma) i\right) dx \\
&= I_1 + I_2.
\end{aligned} \tag{3.35}$$

We can write I_1 as $I_{11} + I_{12}$, and we use the Neumann boundary conditions in (3.3) qnd Green's formula we get

$$\begin{aligned}
I_{11} &= \int_\Omega \left[i + \theta\left(s - \frac{\Lambda}{\mu}\right)\right] d_1 \Delta s dx \\
&= d_1 \int_\Omega \left(i + \theta s - \theta\frac{\Lambda}{\mu}\right) \Delta s dx \\
&= -d_1 \int_\Omega (\nabla i + \theta \nabla s) \nabla s dx \\
&= -d_1 \int_\Omega \nabla i \nabla s dx - \theta d_1 \int_\Omega |\nabla s|^2 dx,
\end{aligned}$$

and

$$\begin{aligned}
I_{12} &= \int_\Omega \left[s + i + 2\frac{\Lambda}{\mu+\sigma}\right] d_2 \Delta i dx \\
&= d_2 \int_\Omega \left(s + i + 2\frac{\Lambda}{\mu+\sigma}\right) \Delta i dx \\
&= -d_2 \int_\Omega (\nabla s + \nabla i) \nabla i dx \\
&= -d_2 \int_\Omega \nabla s \nabla i dx - d_2 \int_\Omega |\nabla i|^2 dx,
\end{aligned}$$

so we can write this term I_1 as follows

$$\begin{aligned}
I_1 &= -d_1 \int_\Omega \nabla i \nabla s dx - \theta d_1 \int_\Omega |\nabla s|^2 dx - d_2 \int_\Omega \nabla s \nabla i dx - d_2 \int_\Omega |\nabla i|^2 dx \\
&= -\int_\Omega \left(d_1 (\nabla i \nabla s) + d_2 (\nabla i \nabla s) + \theta d_1 |\nabla s|^2 + d_2 |\nabla i|^2\right) dx \\
&= -\int_\Omega \left(\theta d_1 |\nabla s|^2 + (d_1 + d_2) \nabla i \nabla s + d_2 |\nabla i|^2\right) dx \\
&= -\int_\Omega Q(\nabla s, \nabla i) dx,
\end{aligned}$$

where $Q(\nabla s, \nabla i)$ is a quadratic form.
As we know Q positive because θ, d_1 and d_2 are satisfying the conditions $\theta d_1 > 0$ and $\theta > \frac{(d_1+d_2)^2}{4d_1d_2}$, which gives us

$$I_1 \leq 0. \tag{3.36}$$

Now, we move to the other part I_2 which is given by the rest of the expression

$$\begin{aligned}
I_2 &= \int_\Omega \left[\left(i+\theta\left(s-\frac{\Lambda}{\mu}\right)\right)\left(\Lambda-\beta\frac{s\varphi(i)}{s+i}-\mu s\right)\right]dx \\
&\quad+\int_\Omega\left[\left(s+i+2\frac{\Lambda}{\mu+\sigma}\right)\left(\beta\frac{s\varphi(i)}{s+i}-(\mu+\sigma)i\right)\right]dx \\
&= \Lambda\int_\Omega idx-\beta\int_\Omega\frac{is\varphi(i)}{s+i}dx-\mu\int_\Omega isdx+\theta\int_\Omega\Lambda\left(s-\frac{\Lambda}{\mu}\right)dx-\theta\beta\int_\Omega\left(s-\frac{\Lambda}{\mu}\right)\frac{s\varphi(i)}{s+i}dx \\
&\quad-\theta\mu\int_\Omega s\left(s-\frac{\Lambda}{\mu}\right)dx+\beta\int_\Omega\frac{s^2\varphi(i)}{s+i}dx+\beta\int_\Omega\frac{is\varphi(i)}{s+i}dx+\frac{2\beta\Lambda}{\mu+\sigma}\int_\Omega\frac{s\varphi(i)}{s+i}dx \\
&\quad-(\mu+\sigma)\int_\Omega sidx\ -(\mu+\sigma)\int_\Omega i^2dx\ -2\Lambda\int_\Omega idx \\
&= -\mu\int_\Omega isdx+\Lambda\int_\Omega idx-\beta\int_\Omega\frac{is\varphi(i)}{s+i}dx-\theta\mu\int_\Omega\left(s-\frac{\Lambda}{\mu}\right)^2dx-\theta\beta\int_\Omega\frac{s^2\varphi(i)}{s+i}dx \\
&\quad+\theta\beta\frac{\Lambda}{\mu}\int_\Omega\frac{s\varphi(i)}{s+i}dx+\beta\int_\Omega\frac{s^2\varphi(i)}{s+i}dx-(\mu+\sigma)\int_\Omega sidx\ +\beta\int_\Omega\frac{is\varphi(i)}{s+i}dx \\
&\quad-(\mu+\sigma)\int_\Omega i^2dx\ +2\frac{\beta\Lambda}{\mu+\sigma}\int_\Omega\frac{s\varphi(i)}{s+i}dx-\Lambda\int_\Omega idx, \\
&= I_{21}+I_{22}+I_{23}+I_{24},
\end{aligned}$$

which are:

$$I_{21} = \beta\int_\Omega\frac{s^2\varphi(i)}{s+i}dx-\theta\beta\int_\Omega\frac{s^2\varphi(i)}{s+i}dx-\beta\int_\Omega\frac{is\varphi(i)}{s+i}dx, \tag{3.37}$$

$$I_{22} = \beta\int_\Omega\frac{is\varphi(i)}{s+i}dx, \tag{3.38}$$

$$\begin{aligned}
I_{23} &= -\Lambda\int_\Omega idx-(\mu+\sigma)\int_\Omega sidx \\
&\leq -\Lambda\int_\Omega idx,
\end{aligned} \tag{3.39}$$

$$I_{24} = \theta\beta\frac{\Lambda}{\mu}\int_\Omega\frac{s\varphi(i)}{s+i}dx+2\frac{\beta\Lambda}{\mu+\sigma}\int_\Omega\frac{s\varphi(i)}{s+i}dx-\theta\mu\int_\Omega\left(s-\frac{\Lambda}{\mu}\right)^2dx-(\mu+\sigma)\int_\Omega i^2dx\,.$$

By using

$$\frac{s}{s+i}\leq 1, \tag{3.40}$$

we get that

$$I_{24} \leq \left(\theta\beta\frac{\Lambda}{\mu} + 2\frac{\beta\Lambda}{\mu+\sigma}\right)\int_\Omega \varphi(i)dx - \theta\mu\int_\Omega \left(s - \frac{\Lambda}{\mu}\right)^2 dx - (\mu+\sigma)\int_\Omega i^2 dx\,. \tag{3.41}$$

so by using (3.37), (3.38), (3.41) and (3.39), we can easily show that

$$\begin{aligned} I_2 &= I_{21} + I_{22} + I_{23} + I_{24} \\ &\leq \beta\int_\Omega \frac{s^2\varphi(i)}{s+i}dx - \theta\beta\int_\Omega \frac{s^2\varphi(i)}{s+i}dx - \beta\int_\Omega \frac{is\varphi(i)}{s+i}dx + \beta\int_\Omega \frac{is\varphi(i)}{s+i}dx \\ &\quad + \left[\left(\theta\beta\frac{\Lambda}{\mu} + 2\frac{\beta\Lambda}{\mu+\sigma}\right)\varphi'(0) - \Lambda\right]\int_\Omega i dx - \theta\mu\int_\Omega \left(s - \frac{\Lambda}{\mu}\right)^2 dx - (\mu+\sigma)\int_\Omega i^2 dx \\ &\leq \beta\int_\Omega \frac{s^2\varphi(i)}{s+i}dx - \theta\beta\int_\Omega \frac{s^2\varphi(i)}{s+i}dx \\ &\quad + \left[\left(\theta\beta\frac{\Lambda}{\mu} + 2\frac{\beta\Lambda}{\mu+\sigma}\right)\varphi'(0) - \Lambda\right]\int_\Omega i dx - \theta\mu\int_\Omega \left(s - \frac{\Lambda}{\mu}\right)^2 dx - (\mu+\sigma)\int_\Omega i^2 dx, \end{aligned}$$

from (3.34):

$$I_2 \leq \beta(1-\theta)\int_\Omega \frac{s^2\varphi(i)}{s+i}dx - \theta\mu\int_\Omega \left(s - \frac{\Lambda}{\mu}\right)^2 dx - (\mu+\sigma)\int_\Omega i^2 dx. \tag{3.42}$$

Finally we get fron (3.36) and (3.42):

$$\begin{aligned} \dot{F}_\theta(t) &\leq -\theta d_1\int_\Omega |\nabla s|^2 dx - d_2\int_\Omega |\nabla i|^2 dx - \theta\mu\int_\Omega \left(s - \frac{\Lambda}{\mu}\right)^2 dx - (\mu+\sigma)\int_\Omega i^2 dx \\ &\leq 0. \end{aligned} \tag{3.43}$$

.

Finally, by Lyapunov direct method E_0 is globally asymptotically stable. ■

3.8.2 Global asymptotic stability with $\mathbf{R}_0 > 1$

First of all we have to prove a lemma extract from [8] , then we will appropriate Lyapunov function.

We take :

$$V(x) = x - 1 - \ln x, \quad \text{for all } x > 0. \tag{3.44}$$

Lemma 3.2 *Given that φ satisfies criteria (3.12) and (3.44) . The inequality*

$$V\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right) \leq V\left(\frac{i}{i^*}\right), \tag{3.45}$$

holds, where (s^*, i^*) is the endemic equilibrium point E, of system.

Proof. from (3.12) we note that φ is nondeacreasing function for all $i > 0$. 2[8]-[10] we put

$$g(i) = \frac{\varphi(i)}{s+i},$$

and

$$m(i) = \frac{g(i)}{i}, \text{ also } m(i^*) = \frac{g(i^*)}{i^*},$$

At thr first case, we suppose that $i \geq i^*$,

$$\begin{aligned} m'(i) &= \frac{g'(i)i - g(i)}{i^2} \\ &\leq \frac{g(i) - g(i)}{i^2} = 0. \end{aligned}$$

Thus, m is deacreasing function,

$$\begin{aligned} m(i) &\leq m(i^*) \\ \frac{g(i)}{i} &\leq \frac{g(i^*)}{i^*}, \end{aligned}$$

since

$$\frac{g(i)}{g(i^*)} \leq \frac{i}{i^*},$$

from (3.12) we get that g increasing , so we have:

$$1 \leq \frac{g(i)}{g(i^*)} \leq \frac{i}{i^*},$$

note that:

$$V'(x) = 1 - \frac{1}{x}.$$

Thus, V is increasing for $x > 1$.
Hence

$$\begin{aligned} V\left(\frac{g(i)}{g(i^*)}\right) &\leq V\left(\frac{i}{i^*}\right) \\ V\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right) &\leq V\left(\frac{i}{i^*}\right), \end{aligned}$$

holds.
In the second case, for $0 < i < i^*$, and note that $m(i)$ is decreasing function,

$$\begin{aligned} m(i) &> m(i^*), \\ \frac{g(i)}{i} &> \frac{g(i^*)}{i^*}, \\ \frac{g(i)}{g(i^*)} &> \frac{i}{i^*}. \end{aligned}$$

$g(i)$: nondecreasing so

$$g(i) < g(i^*)$$

leading to

$$1 > \frac{g(i)}{g(i^*)},$$

so

$$1 > \frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}} > \frac{i}{i^*} > 0.$$

V is decreasing for $0 < x < 1$.

Hence

$$V\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right) \leq V\left(\frac{i}{i^*}\right),$$

holds. ■

Theorem 3.3 *If $R_0 > 1, E^*$ is a globally asymptotically stable, endemic steady state for system (3.1)*

Proof. For this prove, we consider the condidate Lyapunov function

$$W(t) = \int_\Omega \left[s^* V\left(\frac{s}{s^*}\right) + i^* V\left(\frac{i}{i^*}\right) \right] dx.$$

The derivate of $W(t)$ as follows

$$\begin{aligned} \dot{W}(t) &= \int_\Omega \left[\frac{ds}{dt}\left(1 - \frac{s^*}{s}\right) + \frac{di}{dt}\left(1 - \frac{i^*}{i}\right) \right] dx \qquad (3.46) \\ &= \int_\Omega \left(1 - \frac{s^*}{s}\right) \left[d_1 \Delta s + \Lambda - \beta \frac{s\varphi(i)}{s+i} - \mu s \right] dx \\ &\quad + \int_\Omega \left(1 - \frac{i^*}{i}\right) \left[d_2 \Delta i + \beta \frac{s\varphi(i)}{s+i} - (\mu + \sigma) i \right] dx, \qquad (3.47) \end{aligned}$$

we have to use Green's formula and Neuman boundry conditions to get

$$\begin{aligned}
\dot{W}(t) &= -d_1 \int_\Omega \nabla\left(1-\frac{s^*}{s}\right)\nabla s dx + \int_\Omega \left(1-\frac{s^*}{s}\right)\left(\Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s\right)dx - d_2\int_\Omega \nabla\left(1-\frac{i^*}{i}\right)\nabla i dx \\
&\quad + \int_\Omega \left(1-\frac{i^*}{i}\right)\left(\beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i\right)dx \\
&= -d_1 \int_\Omega \frac{s^*}{s^2}|\nabla s|^2 dx + \int_\Omega \left(1-\frac{s^*}{s}\right)\left(\Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s\right)dx - d_2\int_\Omega \frac{i^*}{i^2}|\nabla i|^2 dx \\
&\quad + \int_\Omega \left(1-\frac{i^*}{i}\right)\left(\beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i\right)dx \\
&= M + N,
\end{aligned}$$

we put that

$$M = -d_1 \int_\Omega \frac{s^*}{s}|\nabla s|^2 dx - d_2\int_\Omega \frac{i^*}{i}|\nabla i|^2 dx, \tag{3.48}$$

it is clear that

$$M \leq 0.$$

We move to

$$N = \int_\Omega \left(1-\frac{s^*}{s}\right)\left(\Lambda - \beta\frac{s\varphi(i)}{s+i} - \mu s\right)dx + \int_\Omega \left(1-\frac{i^*}{i}\right)\left(\beta\frac{s\varphi(i)}{s+i} - (\mu+\sigma)i\right)dx, \tag{3.49}$$

we substitute (3.32) in (3.49) we get

$$
\begin{aligned}
N &= \int_\Omega \left(1-\frac{s^*}{s}\right)\left(\beta\frac{s^*\varphi(i^*)}{s^*+i^*}+\mu s^*-\beta\frac{s\varphi(i)}{s+i}-\mu s\right)dx, && (3.50)\\
&+\int_\Omega \left(1-\frac{i^*}{i}\right)\left(\beta\frac{s\varphi(i)}{s+i}-\beta\frac{s^*i\varphi(i^*)}{i^*\left(s^*+i^*\right)}\right)dx, && (3.51)\\
&= \int_\Omega \left[\left(1-\frac{s^*}{s}\right)(\mu s^*-\mu s)+\left(1-\frac{s^*}{s}\right)\left(\beta\frac{s^*\varphi(i^*)}{s^*+i^*}-\beta\frac{s\varphi(i)}{s+i}\right)\right]dx,\\
&+\int_\Omega \left(1-\frac{i^*}{i}\right)\left[\beta\frac{s\varphi(i)}{s+i}-\beta\frac{s^*i\varphi(i^*)}{i^*\left(s^*+i^*\right)}\right]dx\\
&= \int_\Omega [\mu s^*-\mu s-\frac{s^*}{s}\mu s^*+\frac{s^*}{s}\mu s+\beta\frac{s^*\varphi(i^*)}{s^*+i^*}-\beta\frac{s\varphi(i)}{s+i}-\frac{s^*}{s}\times\beta\frac{s^*\varphi(i^*)}{s^*+i^*}+\frac{s^*}{s}\times\beta\frac{s\varphi(i)}{s+i}\\
&+\beta\frac{s\varphi(i)}{s+i}-\beta\frac{s^*i\varphi(i^*)}{i^*\left(s^*+i^*\right)}-\frac{i^*}{i}\times\beta\frac{s\varphi(i)}{s+i}+\frac{i^*}{i}\times\beta\frac{s^*i\varphi(i^*)}{i^*\left(s^*+i^*\right)}]dx\\
&= \int_\Omega \mu s^*\left(1-\frac{s}{s^*}-\frac{s^*}{s}+1\right)dx+\beta\frac{s^*\varphi(i^*)}{s^*+i^*}\int_\Omega(1-\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}-\frac{s^*}{s}+\frac{s^*}{s}\times\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}+\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}\\
&-\frac{i}{i^*}-\frac{i^*}{i}\times\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}+1)dx\\
&= \int_\Omega \mu s^*\left(1-\frac{s^*}{s}\right)\left(1-\frac{s}{s^*}\right)dx\\
&+\beta\frac{s^*\varphi(i^*)}{s^*+i^*}\int_\Omega\left[\left(1-\frac{s^*}{s}\right)\left(1-\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}\right)+\left(1-\frac{i^*}{i}\right)\left(\frac{\frac{s\varphi(i)}{s+i}}{\frac{s^*\varphi(i^*)}{s^*+i^*}}-\frac{i}{i^*}\right)\right]dx && (3.52)\\
&= \mu s^*\int_\Omega\left(1-\frac{s^*}{s}\right)\left(1-\frac{s}{s^*}\right)dx+\beta\frac{s^*\varphi(i^*)}{s^*+i^*}\int_\Omega\left[1-\frac{s^*}{s}+\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}+1-\frac{i}{i^*}-\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}}\right]dx.
\end{aligned}
$$

with some algebric manipulations, we obtain

$$
\begin{aligned}
\left(1-\frac{s^*}{s}\right)\left(1-\frac{s}{s^*}\right) &= -V\left(\frac{s^*}{s}\right)-V\left(\frac{s}{s^*}\right),\\
1-\frac{s^*}{s} &= -V\left(\frac{s^*}{s}\right)-\ln\left(\frac{s^*}{s}\right),\\
1-\frac{i}{i^*} &= -V\left(\frac{i}{i^*}\right)-\ln\left(\frac{i}{i^*}\right),\\
-1+\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}} &= V\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right)+\ln\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right),\\
1-\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}} &= -V\left(\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}}\right)-\ln\left(\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}}\right).
\end{aligned}
$$

Then, we have

$$-\ln\left(\frac{s^*}{s}\right)-\ln\left(\frac{i}{i^*}\right)+\ln\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right)-\ln\left(\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}}\right)=0.$$

and thanks to (3.45) we find

$$\begin{aligned}N &= -\mu s^*\int_\Omega\left[V\left(\frac{s^*}{s}\right)+V\left(\frac{s}{s^*}\right)\right]dx\\ &\quad+\beta\frac{s^*\varphi(i^*)}{s^*+i^*}\int_\Omega\left[-V\left(\frac{s^*}{s}\right)-V\left(\frac{i}{i^*}\right)+V\left(\frac{\frac{\varphi(i)}{s+i}}{\frac{\varphi(i^*)}{s^*+i^*}}\right)-V\left(\frac{\frac{i^*s\varphi(i)}{s+i}}{\frac{is^*\varphi(i^*)}{s^*+i^*}}\right)\right]dx.\end{aligned}$$

which is less than or equal to 0.

So we have that $M\leq 0$ and $N\leq 0$, leading to $\dot{W}(t)\leq 0$. Hence, the global asymptotic stability of E^* foloozs fro, Lyqpunov function direct method ■

3.9 Numerical example

we consider the function $\varphi(i)=\frac{i}{1+i}$, and we obtain

$$\begin{cases}\frac{\partial s}{\partial t}-d_1\Delta s=\Lambda-\beta\frac{si}{(s+i)(1+i)}-\mu s & \text{in } (0,+\infty)\times\Omega,\\ \frac{\partial i}{\partial t}-d_2\Delta i=\beta\frac{si}{(s+i)(1+i)}-(\mu+\sigma)i & \text{in } (0,+\infty)\times\Omega,\\ s_0(x)=s(x,0), i_0(x)=i(x,0) & \text{in } \Omega,\\ \frac{\partial s}{\partial\nu}=\frac{\partial i}{\partial\nu}=0 & \text{in } (0,+\infty)\times\Omega.\end{cases}\tag{3.53}$$

The imposed conditions may be verified as follows:

$$\begin{cases}\varphi(0)=0,\varphi'(0)=1,\\ i\varphi'(i)=\frac{i}{(1+i)^2}\leq\frac{i}{1+i}=\varphi(i).\end{cases}$$

The steady states of system (3.53) are given by $E_0=\left(\frac{\Lambda}{\mu},0\right)$ and $E^*=\left(\frac{\Lambda(1+i^*)}{\beta-\sigma(1+i^*)},\Lambda\frac{(1+i^*)(\sigma+\mu)-\beta}{(\sigma+\mu)(\sigma(1+i^*)-\beta}\right)$ with the reproductive number $R_0=\frac{\beta}{\sigma+\mu}$. In the table bollow, we use different sets of parameters to obtain numerical solutions in the ODE and PDE. For the second example, we assume a single spatial dimension with $\Omega=(0,10)$ throughout the PDE simulations.

Table: Simulation parameters for the Example:

Set	s_0	i_0	d_1	d_2	Λ	β	μ	σ
ODE set 1	2.5	7	-	-	0.8	0.3	0.4	0.2
ODE set 2	6	2.5	-	-	0.8	0.6	0.1	0.3
PDE set 1	$2+\frac{\cos x}{7}$	$3+\frac{\sin x}{8}$	0.02	0.01	0.8	$\frac{3}{10}$	$\frac{2}{5}$	$\frac{1}{5}$
PDE set 2	$1.8+\frac{\cos x}{5}$	$3+\frac{\cos x}{6}$	2	0.5	0.9	$\frac{2}{7}$	$\frac{1}{10}$	$\frac{6}{100}$

The following is a description of the results:

Figure 10 : shows the solutions in the ODE case subject to set 1, with $R_0 = 0.5$. In this case, as $R_0 < 1, E_0 = (2, 0)$ is globally asymptotically stable.

Figure 11 : shows the solutions in the ODE case subject to set 2, with $R_0 = 1.5$. In this case, as $R_0 > 1, E^* = (6.2223, 0.826)$ is globally asymptotically stable.

Figure 12, 13 : depicts the solution in the PDE case subject to parameter set 2, where $R_0 = 1.7857$, which by Theorem (3-4) means that $E^* = (7.9586, 0.65072)$ is globally asymptotically stable.

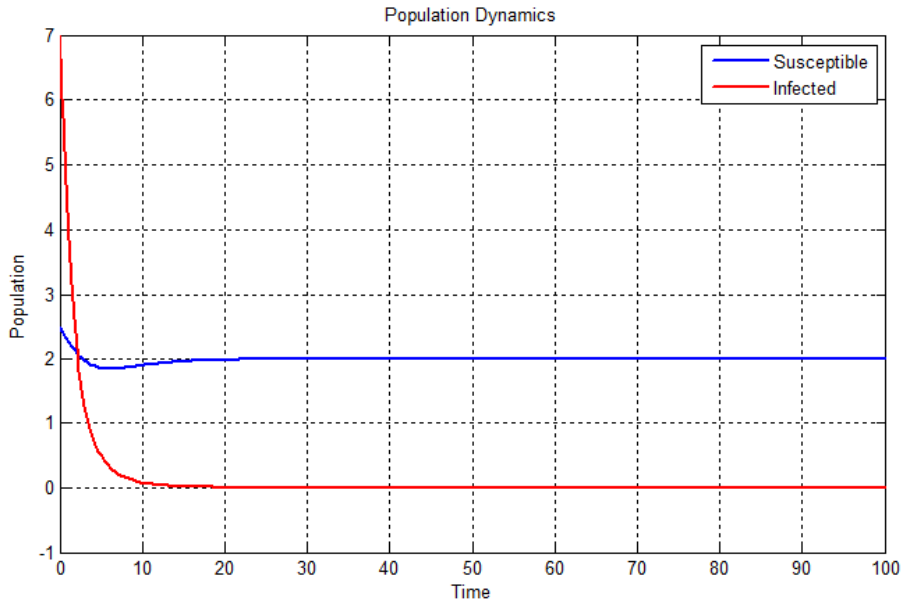

Figure 10: Numerical solutions of system (3.53) (ODE case) subject to the first set of parameters.

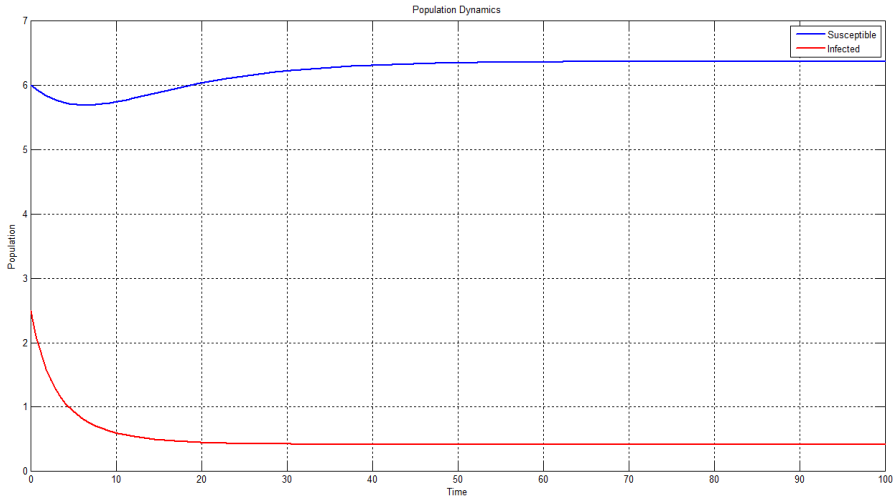

Figure 11: Numerical solutions of system (3.53) (ODE case) subject to the second set of parameters.

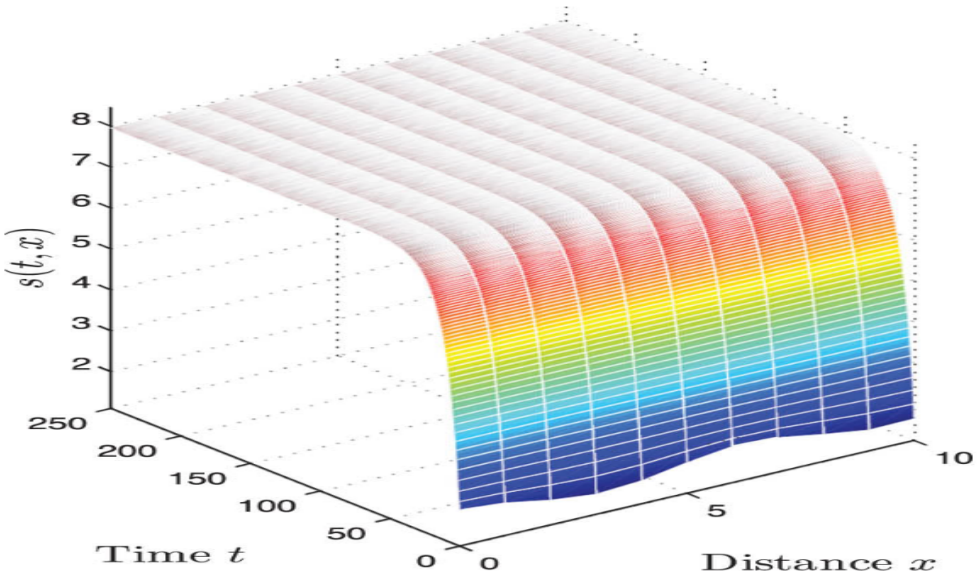

Figure 12: Numerical solutions of system (3.53) susceptible population (PDE case) subject to the second set of parameters.

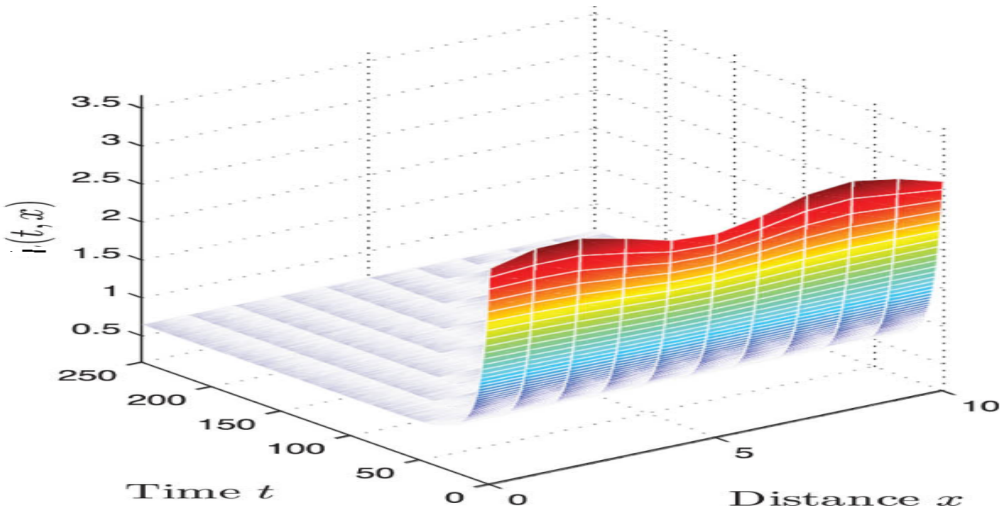

Figure 13: Numerical solutions of system (3.53) infected population (PDE case) subject to the second set of parameters.

Conclusion

We studied an epidemiological model that describes the spread of an infectious disease among people. By analyzing non-linear equations, many important results emerge, the model reveals the existence of equilibrium points, these points can be stable or unstable, indicating the long-term behavior of the disease within the population. The disease dies out or persist within the population depending on the reproductive number. The concept of basic reproduction number (R_0), which represents the average number of secondary infections caused by a single infected individual in a susceptible population. In this work we focus on analyzing the stability of equilibrium points in the proposed model using the Lyapunov function. The results demonstrate that the disease-free equilibrium and the endemic equilibrium are both locally and globally asymptotically stable under specific conditions. Specifically, the disease-free equilibrium is stable when the reproduction number (R_0) is less than 1, while the endemic equilibrium is stable when R_0 is greater than 1. These findings provide crucial insights into the dynamics and long-term behavior of the model, emphasizing the importance of the reproduction number in determining the stability of disease equilibria. In future, we look forward to new research studying the case of $R_0 = 0,$ it is interesting to know if the dosease-free and endemic equilibrium will be stable.

Bibliography

[1] Abdelmalek, S., & Bendoukha, S. Global asymptotic stability for a SEI reaction–diffusion model of infectious diseases with immigration. International Journal of Biomathematics, 11(03), (2018).

[2] Bouaziz, K., Douaifia, R., & Abdelmalek, S. Asymptotic stability of solutions for a diffusive epidemic model. Demonstratio Mathematica, 55(1), (2022), 553-573.

[3] Gayer, M., Legros, D., Formenty, P., & Connolly, M. A. Conflict and emerging infectious diseases. Emerging infectious diseases, 13(11), (2007).

[4] Huang, W., Han, M., & Liu, K. Dynamics of an SIS reaction-diffusion epidemic model for disease transmission. Math. Biosci. Eng, 7(1), (2010), 51-66.

[5] James Watmough P. van den Driessche. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Biosciences, 180, (2002).

[6] Martin, P. M., & Martin-Granel, E. 2,500-year evolution of the term epidemic. Emerging infectious diseases, 12(6),(2006), 976.

[7] P. Driessche and J. Watmough, Reproduction number and sub-threshold endemic equilibria for compartmental models for disease transmission, Math. Biosci. 180 (2002), no. 1–2, 29–48.

[8] R. P. Sigdel and C. C. McCluskey, Global stability for an SEI model of infectious disease with immigration, Appl. Math. Comput. 243 (2014), 684–689.

[9] S. Al-Sheikh and F. Musali, Stability analysis of an HIV/AIDS epidemic model with screening, Int. Math. Forum 66 (2011), 3251–3273.

[10] S. Henshaw and C. C. McCluskey, Global stability of a vaccination model with immigration, Electron. J. Differ. Equ. (2015), no. 92, 1–10.

[11] Brachman PS. Epidemiology. In: Baron S, editor. Medical Microbiology. 4th edition. Galveston (TX): University of Texas Medical Branch at Galveston; Chapter 9, (1996)

[12] Brauer, F., Van den Driessche, P., & Allen, L. J. Mathematical epidemiology 1945 , (2008), 3-17.

[13] Martcheva, M. An introduction to mathematical epidemiology 61, (2015), 9-31.

[14] Slotine, J. J. E., & Li, W. Applied nonlinear control 199, 1, 705. Englewood Cliffs, NJ: Prentice hall. (1991).

[15] Alshakhoury, N. S. Mathematical modeling and control of MERS-CoV epidemics (Doctoral dissertation). (2017).

[16] Chaima, B., & Amel, H. The Volterra-Lyapunov for global stability analysis of a model of Reaction-Diffusion system (Doctoral dissertation, Larbi Tbessi University–Tebessa). (2020).

[17] G.Sallet , Modélisation et simulation en épidémiologie , (2010).

[18] KOUTOU, O. Modélisation mathématique et controle optimal de la dynamique de transmission du paludisme. DEA de l'Université Polytechnique de Bobo-Dioulasso. (2017).

[19] https://www.healthline.com/health/r-naught-reproduction-number#conditions-it-measures