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Continuous and discrete mathematical modelling of the epidemic propagation

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Abstract

This thesis investigates the continuous and discrete mathematical modeling of epidemic propagation. It examines various epidemiological models, including Susceptible-Infected-Recovered (SIR), Susceptible-Infected-Recovered-Vaccinated (SIRV), and vector-borne disease models like the Ross-Macdonald model. Key analyses involve determining equilibrium points and conducting stability analyses (local and global) for these continuous models, often using the basic reproduction number (R_0) to understand conditions for disease persistence or eradication.

The research further extends to the discretization of these models using finite difference methods, exploring the qualitative properties such as nonnegativity and monotonicity of the resulting discrete systems. Stability conditions for these discrete models, particularly for the discretized Ross-Macdonald model, are also investigated, supported by numerical simulations. The study aims to provide a comprehensive understanding of the mathematical tools and frameworks used to model and analyze the spread of infectious diseases under different scenarios, including the impact of vaccination and vector transmission.

Key words:

Susceptible-Infected-Recovered (SIR) model, Epidemic propagation, Vector-borne disease models, Ross-Macdonald model, The basic reproduction number, Nonnegativity, Monotonicity, Stability.

الملـــــــــخص

تتناول هذه الأطروحة النمذجة الرياضية المستمرة والمتقطعة لانتشار الأوبئة. تدرس نماذج وبائية مختلفة، بما في ذلك نموذج SIR (المعرضون للإصابة - المصابون - المتعافون)، و نموذج $SIRV$ (المعرضون للإصابة - المصابون - المتعافون - الملقحون)، ونماذج الأمراض المنقولة بالنواقل مثل نموذج روس ماكدونالد. تتضمن التحليلات الرئيسية تحديد نقاط التوازن وإجراء تحليلات الاستقرار (المحلي والشامل) لهذه النماذج المستمرة، وغالبا ما يستخدم رقم التكاثر الأساسي (R_0) لفهم ظروف استمرار المرض أو القضاء عليه.
يمتد البحث أيضا إلى تحويل هذه النماذج المستمرة إلى نماذج متقطعة باستخدام طرق الفروق المحدودة، واستكشاف الخصائص النوعية مثل عدم السلبية والرتابة للأنظمة المتقطعة الناتجة. كما يتم بحث شروط الاستقرار لهذه النماذج المتقطعة، لا سيما لنموذج روس ماكدونالد المتقطع، مدعوما بمحاكاة عددية. تهدف الدراسة إلى تقديم فهم شامل للأدوات والأطر الرياضية المستخدمة في نمذجة وتحليل انتشار الأمراض المعدية في ظل سيناريوهات مختلفة، بما في ذلك تأثير التطعيم وانتقال العدوى عن طريق النواقل.

الكلمات المفتاحية:

نموذج SIR (المعرضون للإصابة - المصابون - المتعافون)، انتشارالأوبئة، نماذج الأمراض المنقولة بالنواقل، نموذج روس ماكدونالد، رقم التكاثر الأساسي، عدم السلبية، الرتابة، الاستقرار.

Résumé

Cette thèse étudie la modélisation mathématique continue et discrète de la propagation des épidémies. Elle examine divers modèles épidémiologiques, y compris les modèles Susceptible-Infecté-Rétabli (SIR), Susceptible-Infecté-Rétabli-Vacciné (SIRV), et les modèles de maladies à transmission vectorielle comme le modèle de Ross-Macdonald. Les analyses clés comprennent la détermination des points d'équilibre et la conduite d'analyses de stabilité (locale et globale) pour ces modèles continus, utilisant souvent le nombre de reproduction de base (R_0) pour comprendre les conditions de persistance ou d'éradication de la maladie. La recherche s'étend en outre à la discrétisation de ces modèles par des méthodes de différences finies, explorant les propriétés qualitatives telles que la non-négativité et la monotonie des systèmes discrets résultants. Les conditions de stabilité pour ces modèles discrets, en particulier pour le modèle de Ross-Macdonald discrétisé, sont également étudiées, étayées par des simulations numériques. L'étude vise à fournir une compréhension globale des outils et cadres mathématiques utilisés pour modéliser et analyser la propagation des maladies infectieuses dans différents scénarios, y compris l'impact de la vaccination et de la transmission vectorielle.

Mots clés:

Les modèles Susceptible-Infecté-Rétabli (SIR), la propagation des épidémies, Les modèles de maladies à transmission vectorielle, Le modèle de Ross-Macdonald, Le nombre de reproduction de base, La non-négativité, La monotonie, stabilité.

DEDICATION

It is with the help of Almighty **GOD** that this modest work could be carried out, **GOD** who gave me time, reason and lucidity.

Thank you a thousand times and that **«Allah»** will keep and protect.

My dear parents ♡ **"Ghemam Amara Mohammed Khazzani "** ♡ and ♡ **" Ghemam Hamed Salha "** ♡ for their love, their patience and their encouragement which never failed to suit me During my student years,

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INTRODUCTION

The SIR model, which includes the compartments of Susceptible, Infectious, and Recovered, is one of the most important frameworks in mathematics for modeling the spread of infectious diseases. This model traces its roots back to the remarkable contributions of two scholars, William Ogilvy Kermack and Anderson Gray McKendrick, in their 1927 paper " A Contribution to the Mathematical Theory of Epidemics" (Kermack & McKendrick, 1927)[10]. This epitomized work greatly contributed toward the development of structed mathematical reasoning to infectious diseases, which catapulted modern epidemiology.

Prior the implementation of SIR model, disease modeling was almost entirely observational and highly statistical without any coherent mathematical framework. At the dawn of epidemic modeling, one of the earliest attempts can be attributed to Daniel Bernoulli in 1760, wherein he created a basic mathematical model to determine the effects of smallpox inoculation (Bernoulli, 1760) [2]. It was seen in his study that vaccination could prevent a considerable number of deaths, setting the basis for studying disease control through a quantitative lens. Ronald Ross also contributed significantly in 1908 when he introduced mathematical frameworks for modeling the spread of malaria (Ross, 1911)[16].

Ross's work pioneered the idea of thresholds for disease persistence, which would later assist in epidemic modeling.

The SIR model proposed by Kermack and McKendrick in 1927 was a milestone in epidemic modeling[10]. It provided a system of flow with three compartments: a chain of progression

comprising of a Susceptible (S) group of people prone to be infected with the disease, an Infectious (I) group who have the disease and can spread it to other people, and a Recovered (R) group who used to have the disease but are cured and are immune to it). This categorized formation offered the possibility of writing, for the first time in history, equations governing the flows of movements between compartments, and thus provide, for the first time in history, mathematically formalize insight on how an epidemic spreads.

One of the most important insights gained from their research was the concept of epidemic threshold. They proved that an epidemic could die out regardless of the fact that a fraction of the population is still susceptible, as long as a substantial portion of the population is recovering and gaining immunity. This insight is the basis of understanding herd immunity and stating a fundamental need to control the surge of wide spread infections (Kermack & McKendrick 1927).

The SIR model has undergone sophisticated enhancements aimed at addressing new challenges of the epidemiological paradigm since foundational steps were set.

The inclusion of an Exposed (E) compartment in the SEIR model accounts for incubation periods, which is a chronological consideration. Likewise, the SIS model considers immunity that is captured only temporarily and the SIRS model accounts for fading immunity over a period of time. These modifications have enabled the SIR model to analyze an array of infectious diseases like influenza, measles, and, more recently, COVID-19 (Hethcote, 2000; Anderson & May, 1991). Furthermore, the model has been useful in estimating the impact of vaccination policies, quarantine policies, and overall public health policies.

The SIR model continues to be one of the most popular mathematical models in epidemiology because of its innovative approach to offering a new assessment and control strategies for diseases. It incorporates both theoretical reasoning and practical application of controlling a disease. Their work on these models has exposed epidemiologists to new realms of thinking which continues, even to this day, to enable them to tackle an infectious disease.

This thesis explores the continuous and discrete mathematical modeling of epidemic propagation. The work is systematically organized into four main chapters, each dedicated to specific aspects of epidemiological modeling and analysis, providing a cohesive progression from foundational concepts to advanced applications.

The structure of the thesis is as follows:

In the chapter 1: It introduces essential mathematical notation, preliminary results, key theorems (like the Routh-Hurwitz criterion), and important lemmas (such as those related to Lyapunov functions and invariant principles) that form the theoretical bedrock for the subsequent analyses presented in the thesis.

In the chapter 2: Continuous Mathematical Models of the Epidemic Propagation The second chapter delves into the formulation and analysis of continuous mathematical models that describe how epidemics spread through a population. It begins with an examination of SIR-type models, including the foundational Kermack-McKendrick model, a detailed exploration of a specific SIR model (referred to as Model 1) focusing on its stability properties, and an extension to an SIRV model which incorporates the effects of vaccination programs. The chapter further expands to cover vector-borne epidemic models, with a particular focus on the Ross-Macdonald model for malaria transmission, and includes a thorough stability analysis of the equilibrium points for these continuous systems.

In the chapter 3: Discretization by Finite Difference Method of SIR-Type Processes This chapter bridges the gap between continuous theoretical models and their computational counterparts by applying finite difference methods to discretize the SIR-type processes discussed previously. It outlines the discrete formulations for the first SIR model and the SIRV model, employing the forward Euler method for this transformation. A key focus of this chapter is the analysis of the qualitative properties of these newly derived discrete models, specifically addressing the conditions necessary to ensure the nonnegativity of solutions and examining their monotonicity

. **In the chapter 4:** Discretization by Standard Different Finite Difference Techniques The final technical chapter continues the investigation of discrete models, with a particular emphasis on vector-type propagation and more detailed stability considerations. It introduces a discrete version of the Malaria model, building upon the Ross-Macdonald framework. Following this, the chapter provides a stability analysis for the discrete equilibrium points of this model, which involves eigenvalue analysis of the corresponding Jacobian matrix. The chapter concludes by presenting numerical simulations to visually represent and analyze the behavior of these discrete epidemic models.

GENERAL NOTATIONS

Certain notations will be used throughout this memoir which we list below:

$\mathbb{R}$	Set of real numbers.
$\mathbb{R}^+$	Set of positive or zero real numbers.
$\mathbb{R}^n$	Real vector space of dimension n built on the field of the reals.
$\mathbb{N}$	Set of natural numbers.
$\frac{\partial F}{\partial X}$	The jacobian matrix.
λ_i	Proper value.
$\dot{X}$	Derivable from X.

CHAPTER 1

PRELIMINARIES

1.1 Some mathematical preliminaries

In this part we formulate those notions and theorems which are used in our thesis.

1.1.1 Equilibrium point

Definition 1.1.1. *(The equilibrium point)*
An equilibrium point of a dynamical system generated by an autonomous system of ordinary differential equations (ODEs) is a solution that does not change with time.
More precisely, the ODE $x' = f(x)$ *has an equilibrium solution* $x(t) = x_e$, *if* $f(x_e) = 0$.

Definition 1.1.2. *[14](The disease-free equilibrium point)*
The disease-free equilibrium is defined as the point at which no disease is present in the population.

Definition 1.1.3. *[9](The endemic equilibrium)*
The endemic equilibrium state is the state where the disease cannot be totally eradicated but remains in the population. For the disease to persist in the population, the Immunized Class, the Latently Infected Class, the Infectious class and the Recovered Class must not be zero at equilibrium state.

1.1.2 Routh-Hurwitz

Theorem 1.1.1. *(Routh-Hurwitz)*
Consider the characteristic equation

$$|\lambda \boldsymbol{I} - \boldsymbol{B}| = \lambda^n + b_1\lambda^{n-1} + \cdots + b_{n-1}\lambda + b_n = 0,$$

determining the n eigenvalues λ of the real $n \times n$ matrix $\boldsymbol{B}$. Then all the eigenvalues λ of B have negative real parts if

$$\Delta_1 > 0,\ \Delta_2 > 0,\ \ldots,\ \Delta_n > 0,$$

where

$$\Delta_k = \begin{vmatrix} b_1 & 1 & 0 & 0 & 0 & 0 & \cdots & 0 \\ b_3 & b_2 & b_1 & 1 & 0 & 0 & \cdots & 0 \\ b_5 & b_4 & b_3 & b_2 & b_1 & 0 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & & & \\ b_{2k-1} & b_{2k-2} & b_{2k-3} & b_{2k-4} & b_{2k-5} & b_{2k-6} & \cdots & b_k \end{vmatrix}.$$

1.1.3 Lyapunov function and invariant principle

Definition 1.1.4. *(Asymptotic stability)*
An equilibrium point $x^* = 0$ is asymptotically stable at $t = t_0$ if

1. *$x^* = 0$ is stable,*
2. *$x^* = 0$ is locally attractive; i.e., there exists $\delta(t_0)$ such that*

$$\| x(t_0) \| < \delta \Rightarrow \lim_{t\to\infty} x(t) = 0.$$

Definition 1.1.5. *(Global asymptotic stability)*
The equilibrium state $x^* = 0$ is globally asymptotically stable, if it is asymptotically stable for any $\delta' > 0$.

Definition 1.1.6. *(Lyapunov function)*
Let $V : A \to \mathbb{R}$ be a continuous function defined in some neighborhood A of the equilibrium point $x = \bar{x}$ such that:

1. $V(\bar{x}) = 0$ *and* $V(x) > 0$ *if* $x \neq \bar{x}$.

2. $\dot{V}(x) \leqslant 0$ *in* $A \setminus \{\bar{x}\}$.

Definition 1.1.7. *(Lyapunov stability point)*
If there exists a Lyapunov function of the system $\dot{x} = f(x(t))$, *then* $x = 0$ *is a stable equilibrium point i.s.L. If in addition,* $\dot{V}(x) < 0$, *for* $0 < \|x\| < r$ *for some* r, *then* $x = 0$ *is asymptotically stable (locally in the region* $0 < \|x\| < r$*).*
Global asymptotic stability (G.A.S.) of the origin may be concluded if there exists a $V(x)$ *that is positive definite and* $\dot{V}(x)$ *that is negative definite in the entire state space. In addition,* V *must be redially unbounded, that is,* $V(x) \to \infty$ *as* $\| x \| \to \infty$.

Definition 1.1.8. *(Invariant set)*
Let $F : X \to X$ *some given map. We say that the subset* B *in* X *is an invariant subset for* F *when every trajectory* $x(t)$ *which starts from a point in* B *remains in* B *for all time.*

Theorem 1.1.2. *(Lasalle's Invariant Principle)[15]*
Let $V : \mathbb{R}^n \to \mathbb{R}$ *be a locally positive definite function. Suppose that within the compact region* $A_c = \{x \in \mathbb{R}^n : V(x) \leqslant c\}$ *the function satisfies* $\dot{V}(x) \leqslant 0$. *Define the set*

$$S = \{x \in A_c : \dot{V}(x) = 0\}.$$

As $t \to \infty$, *any trajectory will approach the largest invariant subset within* S; *specifically, the* ω*-limit set of the trajectory will be contained in this maximal invariant subset.*
Moreover, if S *contains no invariant sets other than* $x = 0$, *then the equilibrium point* $x = 0$ *is asymptotically stable.*

CHAPTER 2

CONTINUOUS MATHEMATICAL MODELS OF THE EPIDEMIC PROPAGATION

2.1 SIR-type models

2.1.1 The basic SIR model (Kermack-McKendrick model)

One of the early achievements in mathematical epidemiology is the Kermak-McKendrick Model.

The following system of ordinary differential equations provides this simple model, which is developed for a population that is separated into three discrete sub-populations or compartments- susceptible class S, infective class I, and recovered class R.

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -aIS, \tag{2.1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = aIS - bI, \tag{2.2}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = bI. \tag{2.3}$$

In (2.1)-(2.3) we have: a is the infection rate, b is the recovery rate, $S(t)$ is the number of susceptible individuals, $I(t)$ is the number of infected individuals, $R(t)$ is the number of

recovered individuals.

In a closed population, the Kermack-McKendrick model is a SIR model for the number of cases of a contagious illness over time. It was put up to explain the sharp increases and decreases in infected patients seen during epidemics like the plague.

Combining the infection rate and the rate of recovery, we determine the model's "epidemiological threshold" attribute.

$$R_0 = \frac{aS(t)}{b}, \tag{2.4}$$

which is called basic reproduction number. When $R_0 < 1$, there is no outbreak of the disease in the sense that the population of the infectious class $I(t)$ decreases monotonically to 0. In the sense that $I(t)$ first climbs monotonically to a maximum value and then declines monotonically to zero, there will only be one breakout of the disease when $R_0 > 1$.

2.1.2 Model 1 and its stability

The SIR Model is used in epidemiology to compute the amount of susceptible, infected, recovered people in a population.

This model is suitable for use under the following assumptions:

- The population is fixed.
- There is no effect on the risk of infection by age, gender, marital status and ethnicity.
- The only way a person can leave the susceptible group is to become infected.
- The only way a person can leave the infected group is to recover from the disease.
- The death rate is equal among all three categories, and we assume that the birth and death rates are equal so that the total population remains constant.
- There is no effect of genetic immunity.

Notation of the model:

- $S(t)$ is the number of susceptible individuals at time t.
- $I(t)$ is the number of infected individuals at time t.

- $R(t)$ is the number of recovered individuals at time t.
- N is the total population size.

We can define this model with these differential equations [3]:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = bN - \frac{aI(t)S(t)}{N} - bS(t), \tag{2.5}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{aI(t)S(t)}{N} - cI(t) - bI(t), \tag{2.6}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = cI(t) - bR(t), \tag{2.7}$$

where $S(t)$ is the number of susceptible at time t, $I(t)$ is the number of infected people at time t, $R(t)$ is the number of recovered at time t, a is the transmission of disease from an infected person in a time period, b is the death and birth rate which are assumed to be equal, c is the recovery rate and the population size is constant.
Here $N = S(t) + I(t) + R(t)$ and hence

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \frac{\mathrm{d}S}{\mathrm{d}t} + \frac{\mathrm{d}I}{\mathrm{d}t} + \frac{\mathrm{d}R}{\mathrm{d}t} = 0. \tag{2.8}$$

Differential equations can be simplified by using the notations
$s = \frac{S}{N}$, $i = \frac{I}{N}$ and $r = \frac{R}{N}$.
We get:

$$\begin{aligned}\frac{\mathrm{d}s}{\mathrm{d}t} = \frac{\mathrm{d}}{\mathrm{d}t}\left(\frac{S}{N}\right) = \frac{1}{N}\left(\frac{\mathrm{d}S}{\mathrm{d}t}\right) = \frac{1}{N}\left(bN - \frac{aIS}{N} - bS\right) &= b - a\left(\frac{I}{N}\right)\left(\frac{S}{N}\right) - \frac{bS}{N} \\ &= b - ais - bs,\end{aligned} \tag{2.9}$$

so (2.5) takes the form

$$\frac{\mathrm{d}s}{\mathrm{d}t} = b - ais - bs.$$

We did the same for (2.6) and (2.7), and as a result, we obtain:

$$\frac{\mathrm{d}i}{\mathrm{d}t} = ais - ci - bi, \tag{2.10}$$

$$\frac{\mathrm{d}r}{\mathrm{d}t} = ci - br. \tag{2.11}$$

The initial conditions are given as follows
$s(0) > 0,\ i(0) > 0,\ r(0) > 0.$

Here b is the recruitment and natural death rate, a is the effective contact rate between susceptible and infected individuals and c is the recovery rate of infected individuals.
By considering the total population density, we have $s(t) + i(t) + r(t) = 1$, therefore $r(t) = 1 - s(t) - i(t)$.
Since the first two equations in the model are independent on $r(t)$, we can consider their system as a model.

$$\frac{\mathrm{d}s}{\mathrm{d}t} = b - ais - bs, \tag{2.12}$$

$$\frac{\mathrm{d}i}{\mathrm{d}t} = ais - ci - bi. \tag{2.13}$$

First, we show an important qualitative property of the model, which is quite natural based on the physical meaning.

Lemma 2.1.1. *If* $s(0) > 0,\ i(0) > 0,\ r(0) > 0$, *the solutions* $s(t),\ i(t)$, *and* $r(t)$ *of the system are positive for all* $t \geqslant 0$.

Proof. We assume a contradiction: The contradiction means the following. The statement is not true for one of the components $s(t),\ i(t)$, and $r(t)$. This yields that there exist the time-level t_i $(i = 1, 2, 3)$, t_1 for the component S, t_2 for the component I and t_3 for the component R, such that:

1. There exists a first time t_1 such that
   $s(t_1) = 0,\ s'(t_1) \leqslant 0,\ i(t_1) \geqslant 0,\ r(t_1) \geqslant 0,\ i(t_1) + r(t_1) > 0,\ i(t) > 0,\ r(t) > 0,\ t \in (0, t_1)$.

2. There exists a t_2, such that $i(t_2) = 0,\ i'(t_2) \leqslant 0,\ s(t_2) \geqslant 0,\ r(t_2) \geqslant 0,\ s(t_2) + r(t_2) > 0,\ s(t) > 0,\ r(t) > 0,\ t \in (0, t_2)$.

3. There exists a t_3, such that $r(t_3) = 0,\ r'(t_3) \leqslant 0,\ s(t_3) \geqslant 0,\ i(t_3) \geqslant 0,\ s(t_3) + i(t_3) > 0,\ s(t) > 0,\ i(t) > 0, t \in (0, t_3)$.

In the first case, we have $s'(t_1) = b > 0$ which is a contradiction meaning that $s(t) > 0,\ t \geqslant 0$, in the second case, we have $i'(t_2) = 0$ which is a contradiction meaning that $i(t) > 0,\ t \geqslant 0$, in the third case, we have $r'(t_3) = ci > 0$ which is a contradiction meaning that $r(t) > 0,\ t \geqslant 0$. Thus, the solutions $s(t),\ i(t)$, and $r(t)$ of the system remain positive for all $t > 0$. □

2.1.2.1 The equilibrium point

Let $\Omega = \left\{(s(t),\ i(t)) \in \mathbb{R}^2_+,\ s(t) + i(t) \leqslant 1\right\}$ be the set for the above system of equation (2.11),(2.12) and (2.13).

In the following we analyze the equilibrium points of this model.

For this model there exist two equilibrium points which we define in sequel.

From the above system equation, we get the condition for the equilibrium :

$$\begin{cases} b - ais - bs = 0, & (2.14) \\ ais - ci - bi = 0. & (2.15) \end{cases}$$

Here

$$\begin{cases} b - ais - bs = 0, & (2.16) \\ i(as - c - b) = 0, & (2.17) \end{cases}$$

and hence from (2.17), we get: $i = 0$ or $s = \dfrac{b+c}{a}$.

For $i = 0$, we have $b - bs = 0$ this means $s = 1$.

For $s = \dfrac{b+c}{a}$, we have

$$b - ai\left(\frac{b+c}{a}\right) - b\left(\frac{b+c}{a}\right) = b - i(b+c) - b\left(\frac{b+c}{a}\right) = 0.$$

Therefore, $i = \dfrac{b(a-b-c)}{a(b+c)}$.

Hence, as a final result, we obtain: the system has two equilibrium points namely

1. Disease-free equilibrium point $A_0(s = 1,\ i = 0)$.
2. Endemic equilibrium point $A^*\left(s = \dfrac{c+b}{a},\ i = \dfrac{b(a-c-b)}{a(c+b)}\right)$.

Corollary 2.1.1. *The endemic equilibrium point A^* exists only when the infection rate is greater than the death rate of the infected individuals, that means $a > c + b$.*

Proof. Due to the relation we have

$$\begin{aligned} s^* + i^* &= \frac{c+b}{a} + \frac{b(a-c-b)}{a(c+b)} = \frac{c+b}{a} + \frac{ba}{a(c+b)} - \frac{b(c+b)}{a(c+b)} = \frac{c+b}{a} + \frac{b}{c+b} - \frac{b}{a} \\ &= \frac{c}{a} + \frac{b}{b+c} = \frac{c(b+c)+ba}{a(b+c)}. \end{aligned}$$

Since, $s^* + i^* \leqslant 1$, we have the relation $\frac{c(b+c)+ba}{a(b+c)} \leqslant 1$. Therefore,

$$c(b+c)+ba \leqslant a(b+c),$$

than,

$$c(b+c) \leqslant a(b+c)-ba,$$

than,

$$b+c \leqslant a,$$

hence,

$$\frac{a}{b+c} \geqslant 1 \ / \frac{a}{b+c} = R.$$

□

2.1.2.2 The linear stability of the equilibrium points

Theorem 2.1.1. *(Linearisation method)*
Let c_0 is an equilibrium point and let $J(x)$ be the Jacobian matrix of the system (2.5)-(2.7).

- *If all eigenvalues of $J(x)$ at the point $x = c_0$ has negative real part, then the equilibrium point c_0 is asymptotically stable.*
- *If some eigenvalue of $J(x)$ has positive (negative) real part, then equilibrium c_0 is unstable.*

The Jacobian matrix for the above system is:

$$\mathrm{J}(s,i) = \begin{bmatrix} -ai-b & -as \\ ai & as-c-b \end{bmatrix}.$$

For the disease-free equilibrium point:

$$J(1,0) = \begin{bmatrix} -b & -a \\ 0 & a-c-b \end{bmatrix},$$

since $J(1,0)$ is an upper triangular matrix, and hence its eigenvalues are the diagonal elements. Therefore,

$\lambda_1 = -b$ and $\lambda_2 = a - b - c$.

Hence, clearly we have $\lambda_1 < 0$. Lets study λ_2. If λ_2 is negative that means the condition $a - b - c < 0$.

For this case the disease-free equilibrium point is locally asymptotically stable as both the eigenvalues are negative.

Consequently, for the case $a < b + c$ the disease-free equilibrium point of the system is locally asymptotically stable. This indicates that the disease will be overcome. On the other hand, the infected group exists only if $a > b + c$.

The disease-free equilibrium point is unstable if $\lambda_2 > 0$, then, each infected individual will result in the entire infection period coming into contact with an susceptible individual, which will then lead to disease invasion of the susceptible population, and the disease-free equilibrium point will become unstable.

For Endemic equilibrium point:

$$\mathrm{J}\left(\frac{c+b}{a}, \frac{b(a-c-b)}{a(c+b)}\right) = \begin{bmatrix} -\dfrac{ba}{c+b} & -(c+b) \\ \dfrac{ba}{c+b} - b & 0 \end{bmatrix}.$$

Now, we calculate the characteristic equation of the previous matrix as follows:

$$\begin{vmatrix} -\dfrac{ba}{c+b} - \lambda & -(c+b) \\ \frac{ba}{c+b} - b & -\lambda \end{vmatrix} = 0.$$

We get this equation

$$\left(-\frac{ba}{c+b} - \lambda\right)(-\lambda) + b(a-c-b) = 0.$$

We get the characteristic equation:

$$\lambda^2 + \frac{ab}{c+b}\lambda + b(a-c-b) = 0.$$

The coefficients $\dfrac{ab}{c+b}$ and $b(a-c-b)$ are positive (since $a > b + c$).

Hence the eigenvalues are:

$$\lambda_1 = \frac{1}{2}\left[-\frac{ab}{c+b} \pm \sqrt{b^2a^2/(b+c)^2 - 4b(a-c-b)}\right],$$

and

$$\lambda_2 = \frac{1}{2}\left[-bR \pm \sqrt{b^2R^2 - 4b(a-c-b)}\right].$$

If the quantity under the square root is negative than the eigenvalues are complex with real part.

That means the endemic equilibrium is stable since the real parts of both eigenvalues are negative.

Observation:

The linear stability of the equilibrium points that the disease-free and endemic equilibrium point cannot exist concurrently. If $R < 1$, then the disease-free equilibrium point is stable, and if $R > 1$, then the endemic equilibrium point is stable.

Theorem 2.1.2. *If $R \leqslant 1$, then the disease free equilibrium point of the system is globally asymptotically stable on Ω.*

Proof. Let the following Lyapunov function $V : \Omega \to \mathbb{R} \quad V(s,i) = i(t)$. Then

$$\dot{V}(s,i) = i(\dot{t}) = ais - (c+b)i = (c+b)i\Big[\frac{a}{c+b}s - 1\Big] = (c+b)i\,[Rs-1]. \tag{2.18}$$

For $R < 1$ then $\dot{V}(s,i) = 0$. Moreover $\dot{V}(s,i) = 0$ if $i(t) = 0$ or $s(t) = 1$ and $R = 1$, hence by Lasalle invariance principle, the disease-free equilibrium point is globally asymptotically stable in Ω. □

Theorem 2.1.3. *The endemic equilibrium point $A^*(s^*,\ i^*)$ of the system is globally asymptotically stable on Ω.*

Proof. We have to define the suitable Lyapunov function $L : \Omega_+ \to \mathbb{R}$.

We us the notation $\Omega_+ = \{s(t), i(t) \in \Omega | s(t) > 0,\ i(t) > 0\}$. We define the Lyapunov function L as follows

$L(s,i) = \alpha\Big[s - s^* ln(\frac{s}{s^*})\Big] + \beta\Big[i - i^* ln(\frac{i}{i^*})\Big]$,

where α and β are positive constants will be chosen later. Then

$$\frac{\mathrm{d}L}{\mathrm{d}t} = \alpha(\dot{s} - \frac{s^*\dot{s}}{s}) + \beta(i - \frac{i^*\dot{i}}{i}) = \alpha(s-s^*)(\frac{b}{s} - b - ai) + \beta(i-i^*)(as - (b+c),$$

giving consideration to the equilibrium point we get,

$$\frac{b}{s} - b - ai = 0,$$

then $b = \frac{b}{s^*} - ai^*$ and $as - (b + c) = 0$ then $b + c = as^*$. After substituting we get,

$$\frac{\mathrm{d}L}{\mathrm{d}t} = a(s - s^*)(i - i^*)(\beta - \alpha) - b\alpha\frac{(s - s^*)^2}{ss^*},$$

so, for $\alpha = \beta = 1$, the derivative of the Lyapunov function is negative. Either if $s = s^*$ then $\frac{\mathrm{d}L}{\mathrm{d}t} = 0$, then by Lasalle invariance principle, the endemic equilibrium point is globally asymptotically stable. □

Summary: In this model is used for computing the amount of susceptible, infected, recovered people in a population. It uses some specific assumption.
If the initial conditions are positive, then the solutions of the system are positive for all $t > 0$.
This model has two equilibrium points which are:

$$A_0(s = 1,\ i = 0)\ and\ A^*\left(s = \frac{c + b}{a},\ i = \frac{b(a - c - b)}{a(c + b)}\right).$$

We use the linearisation method to know if the equilibrium points are stable or no, we found that the disease-free equilibrium point is locally asymptotically stable and if $R < 1$ it's globally asymptotically stable on Ω, the endemic equilibrium point is locally stable and it's globally asymptotically stable on Ω_+ by using Lyapunov stability point.

2.1.3 SIRV Model and its stability

The second model is a model in which we have also induced vaccination[3]:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = (1 - p)b - aIS - bS, \tag{2.19}$$
$$\frac{\mathrm{d}I}{\mathrm{d}t} = aIS - cI - bI, \tag{2.20}$$
$$\frac{\mathrm{d}R}{\mathrm{d}t} = cI - bR, \tag{2.21}$$
$$\frac{\mathrm{d}V}{\mathrm{d}t} = pb - bV. \tag{2.22}$$

Notation of the model:

- V is the group to which vaccination is given.

- p is the vaccination rate.

Since $S + I + R + V = 1$ then by giving consideration to the total population density we get

$$R(t) = 1 - S(t) - I(t) - V(t).$$

Then we can consider the following system

$$\frac{\mathrm{d}S}{\mathrm{d}t} = (1-p)b - aIS - bS. \tag{2.23}$$

The differential equation (2.22) for V can be solved directly, independently of the others, since it is linear differential equation. Its solution is $V(t) = V(0) + p(1 - e^{-bt})$.

$$\frac{\mathrm{d}I}{\mathrm{d}t} = aIS - cI - bI, \tag{2.24}$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} = pb - bV. \tag{2.25}$$

The region into the system is $\Omega_1 = \left\{(S(t),\ I(t),\ V(t)) \in \mathbb{R}^3_+,\ S(t) + I(t) + V(t) \leqslant 1\right\}$.

2.1.3.1 The equilibrium point

There are two equilibrium points for this model we get it as same in the Model 1:

1. Disease-free equilibrium point $A_0(S = 1 - p,\ I = 0,\ V = p)$.
2. Endemic equilibrium point $A_e\left(S = \frac{b+c}{a},\ I = \frac{b(a(1-p)-c-b)}{a(c+b)},\ V = p\right)$.

2.1.3.2 The linear stability of the equilibrium points

The Jacobian matrix for the above system is

$$\mathrm{J}(S, I, V) = \begin{bmatrix} -aI - b & -aS & 0 \\ aI & aS - c - b & 0 \\ 0 & 0 & -b \end{bmatrix}.$$

1. **For disease-free equilibrium point:**

$$\mathrm{J}(1-p, 0, p) = \begin{bmatrix} -b & -a(1-p) & 0 \\ 0 & a(1-p) - c - b & 0 \\ 0 & 0 & -b \end{bmatrix}.$$

The equation that corresponds to the disease-free equilibrium points is

$$\begin{vmatrix} -b-\lambda & -a(1-p) & 0 \\ 0 & a(1-p)-c-b-\lambda & 0 \\ 0 & 0 & -b-\lambda \end{vmatrix} = 0.$$

It gives two eigenvalues

$$\lambda_1 = -b,\ \lambda_2 = -b \text{ and } \lambda_3 = a(1-p)-c-b.$$

Here, λ_1 and λ_2 are negative.

There is a condition for λ_3:

- If $\lambda_3 > 0$ that is mean $p < \dfrac{a-c-b}{a}$, then the disease-free equilibrium point is not locally asymptotically stable.
- If $\lambda_3 < 0$ that is mean $p > \dfrac{a-c-b}{a}$.

  Therefore, the model is linearly stable because the eigenvalues are negative.

2. **For endemic equilibrium point:**

$$\mathrm{J}\left(\frac{b+c}{a}, \frac{b(a(1-p)-c-b)}{a(c+b)}, p\right) = \begin{bmatrix} -\frac{ba(1-p)}{c+b} & -(c+b) & 0 \\ \frac{b[a(1-p)-c-b]}{c+b} & 0 & 0 \\ 0 & 0 & -b \end{bmatrix}.$$

Now, we calculate the characteristic equation of the previous matrix as follows:

$$\begin{vmatrix} -\frac{ba(1-p)}{c+b}-\lambda & -(c+b) & 0 \\ \frac{b[a(1-p)-c-b]}{c+b} & -\lambda & 0 \\ 0 & 0 & -b\lambda \end{vmatrix} = 0.$$

We get characteristic equation

$$(b+\lambda)\left\{\lambda^2 + \frac{ba(1-p)}{c+b}\lambda + b[a(1-p)-c-b]\right\} = 0.$$

Clearly $\dfrac{ba(1-p)}{c+b}$ and $b[a(1-p)-c-b]$ are positive. Since $p < \dfrac{a-b-c}{a}$.

So, the eigenvalues are

$$\lambda_1 = -b,$$

$$\lambda_2 = -\frac{ba(1-p)}{c+b} \pm \sqrt{b^2a^2(1-p)^2/(c+b)^2 - 4b[a(1-p)-c-b]}.$$

λ_1 is negative, now we studying λ_2.

- If the quantity under the square root is greater than $\left(-\frac{ba(1-p)}{c+b}\right)^2$, then the eigenvalue is complex with negative real part.
- If the quantity under the square root is smaller than $\left(-\frac{ba(1-p)}{c+b}\right)^2$, then the eigenvalue still has negative real part.

  Hence, the endemic equilibrium is locally stable because the both eigenvalues are negative.

Theorem 2.1.4. *The disease-free equilibrium point of the system is globally asymptotically stable on* Ω_1.

Proof. Let use define the following Lyapunov function $T : \Omega_1 \to \mathbb{R}$ $T(S, I, V) = S(t)+I(t)$. Then

$$\begin{aligned} \dot{T}(S, I, V) &= \dot{S}(t) + \dot{I}(t), \\ \dot{T}(S, I, V) &= (1-p)b - bS - (c+b)I, \\ \dot{T}(S, I, V) &= (c+b)\left[\frac{(1-p)b}{c+b} - \frac{bS}{c+b} - I\right]. \end{aligned} \tag{2.26}$$

If $\frac{b(1-p)}{a+c} < 1$ then, $\dot{T}(S, I, V) < 0$. moreover at $(1-p, 0, V)$, $\dot{T}(S, I, V) = 0$.
Hence, by LaSalle's Invariance Principle [11], the disease-free equilibrium point is globally asymptotically stable. □

Theorem 2.1.5. *The endemic equilibrium point* $A_e(S^*,\ I^*,\ V^*)$ *of the system is globally asymptotically stable on* Ω_1.

Proof. Where $\Omega_+ = \{(S(t), I(t)) \in \Omega,\ S(t) > 0,\ I(t) > 0,\ V(t) > 0\}$ given by

$$L_1(S, I, V) = \alpha\left[S - S^* \ln\left(\frac{S}{S^*}\right)\right] + \beta\left[I - I^* \ln\left(\frac{I}{I^*}\right)\right] + \gamma\left[V - V^* \ln\left(\frac{V}{V^*}\right)\right].$$

Here α and $\beta,\ \gamma$ are positive constants will choose later. Then

$$\begin{aligned} \frac{\mathrm{d}L_1}{\mathrm{d}t} &= \alpha(S - S^*)\left[\frac{(1-p)b}{S} - aI - b\right] + \beta(I - I^*)\left[aS - c - b\right] \\ &\quad + \gamma(V - V^*)\left[\frac{pb}{V} - b\right]. \end{aligned}$$

Giving consideration to the equilibrium point we get,

$$\frac{(1-p)b}{S} - aI - b = 0,$$

then

$$b = \frac{(1-p)b}{S^*} - aI^* \text{ and } aS - c - b = 0,$$

then

$$aS^* = c + b, \ \frac{pb}{V} - b = 0,$$

then $V^* = b$. After substituting we get

$$\frac{\mathrm{d}L_1}{\mathrm{d}t} = -b\alpha(1-p)\frac{(S-S^*)^2}{SS^*} + a(\beta-\alpha)(I-I^*)(S-S^*) - \gamma b(V-V^*)^2.$$

So, for $\alpha = \beta = \gamma = 1$, the derivative of the Lyapunov function is negative. Either, if $S = S^*$ and $V = V^*$ then $\frac{\mathrm{d}L}{\mathrm{d}t} = 0$, then by Lasalle invariance principle, the endemic equilibrium point is globally asymptotically stable. □

2.2 Vector-born epidemic

2.2.1 The Ross-Macdonald model

The total number of population at time t is given by $N = S_h(t) + I_h(t) + R_h(t)$ and $M = S_m(t) + I_m(t)$. The structure of model is shown in Figure. The transfer diagram leads to the following system of ordinary differential equations:

$$\frac{dS_h(t)}{dt} = aN - \frac{cS_hI_m}{N} + gR_h - aS_h, \tag{2.27}$$

$$\frac{dI_h(t)}{dt} = \frac{cS_hI_m}{N} + hR_h - (f+a)I_h, \tag{2.28}$$

$$\frac{dR_h(t)}{dt} = fI_h - (g+h+a)\,R_h, \tag{2.29}$$

$$\frac{dS_m(t)}{dt} = bM - \frac{eS_mI_h}{N} - \frac{dS_mR_h}{N} - bS_m, \tag{2.30}$$

$$\frac{dI_m(t)}{dt} = \frac{eS_mI_h}{N} + \frac{dS_mR_h}{N} - bI_m. \tag{2.31}$$

Notation:

- S_h is the number of susceptible humans.
- I_h is the number of infectious humans.
- R_h is the number of recovered humans.

- S_m is the number of susceptible mosquitoes.
- I_m is the number of infectious mosquitoes.
- N is the total size of the human population (constant).
- M is the total size of the mosquitoes population (constant).
- a is the natural birth and death rate of humans.
- b is the natural birth and death rate of mosquitoes.
- c is from an infectious mosquito to a susceptible human transmission rate in humans.
- d is the recovered human to a susceptible mosquito transmission rate in mosquitoes.
- e is the infectious human to a susceptible mosquito transmission rate in mosquitoes.
- f is treatment rate.
- g is recovery rate.
- h is relapse rate.
- j is the number of mosquitoes per individual.

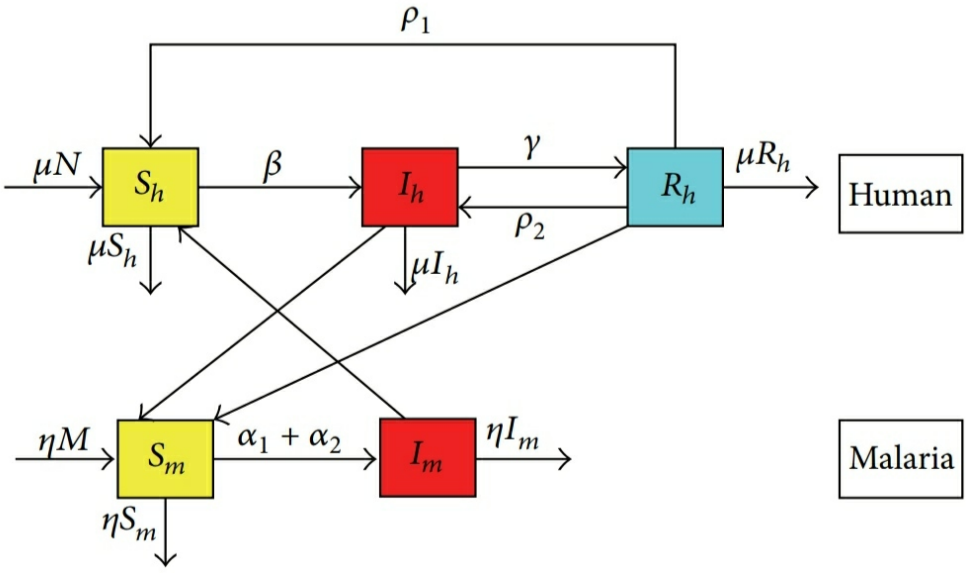

Figure 2.1: The mechanism of the Ross-Macdonald model.

Let: $s_h = \frac{S_h}{N}$, $z_1 = \frac{I_h}{N}$, $z_2 = \frac{R_h}{N}$, $s_m = \frac{S_m}{M}$, $w = \frac{I_m}{M}$, with $s_h + z_1 + z_2 = 1$, $s_m + w = 1$. Then the system (2.27)-(2.31) becomes

$$\frac{dz_1(t)}{dt} = jc\,(1 - z_1 - z_2)\,w + hz_2 - (f + a)z_1, \tag{2.32}$$

$$\frac{dz_2(t)}{dt} = fz_1 - (g + h + a)\,z_2, \tag{2.33}$$

$$\frac{dw(t)}{dt} = e(1 - w)z_1 + d(1 - w)z_2 - bw. \tag{2.34}$$

For this model there are two equilibrium points:

1. Disease-free Equilibrium Point $A = (0, 0, 0)$.

2. Endemic equilibrium point.

2.2.2 Stability of the equilibrium points :

1. **For disease -free equilibrium point:**

   The basic reproduction number of system (2.32)-(2.34) will be obtained by the next generation matrix method formulated in [19].

   The new infection and transition matrices are:

$$F = \begin{bmatrix} -dcw & dcw & jc(1 - z_1 - z_2) \\ 0 & 0 & 0 \\ e(1 - w) & d(1 - w) & 0 \end{bmatrix}.$$

   and

$$V = \begin{bmatrix} f + a & -h & 0 \\ -f & g + h + a & 0 \\ 0 & 0 & b \end{bmatrix}.$$

   The Jacobian matrices F and V of the system at A_0 are :

$$F_{A_0} = \begin{bmatrix} 0 & 0 & jc \\ 0 & 0 & 0 \\ e & d & 0 \end{bmatrix}.$$

and

$$V_{A_0} = \begin{bmatrix} f+a & -h & 0 \\ -f & g+h+a & 0 \\ 0 & 0 & b \end{bmatrix},$$

respectively. The basic reproduction number of the above system is

$$R = \sqrt{\frac{jc(e(g+h+a)+df)}{b(f+a)(g+h+a)-fh)}}.$$

In this case, R is involved to disease transmission by infected humans as well as infection of susceptible humans by infected mosquitos. Malaria infection can be acquired by susceptible mosquitos from infected humans in two ways: infected or recoveries. Susceptible humans become infected after making direct contact with infected mosquitos.

Theorem 2.2.1. *The disease-free equilibrium A_0 is locally asymptotically stable if $R < 1$ and unstable if $R > 1$.*
Moreover, A_0 is globally asymptotically stable if $R \leqslant 1$.

Proof. First, we calculation the characteristic equation of the linearised system is given by:

$$\begin{vmatrix} -(f+a)-\lambda & h & jc \\ f & -(g+h+a)-\lambda & 0 \\ e & d & -b-\lambda \end{vmatrix} = 0.$$

We get this equation

$$jc(fd+e(g+h+a+\lambda))-(b+\lambda)((f+a+\lambda)(g+h+a+\lambda)-fh)=0.$$

The equation thus becomes

$$jc(fd+e(g+h+a))+ejc\lambda+bfh-(b+\lambda)(f+a+\lambda)(g+h+a+\lambda)+\lambda fh=0,$$

$$jc(fd+e(g+h+a))+ejc\lambda+bfh-(b+\lambda)(\lambda^2+\lambda(g+h+2a+f)+(f+a)(g+h+a))+\lambda fh=0.$$

So, the characteristic equation of the linearised system is

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0,$$

with

$$\begin{aligned} A_1 &= g + h + 2a + f + b, \\ A_2 &= b(g + h + 2a + f) + (f + a)(g + h + a) - fh - ejc, \\ A_3 &= -jc(fd + e(g + h + a)) - bfh + b(f + a)(g + h + a). \end{aligned}$$

□

We use the **Routh-Hurwitz** Theorem (1.1.1) to prove that all roots has negative real part.

We have $a_0 = 1 > 0$ and $\Delta_1 = A_1 = f + g + h + b + 2a > 0$.

Now we should prove $\Delta_2 = \begin{vmatrix} A_1 & 1 \\ A_3 & A_2 \end{vmatrix} = A_1A_2 - A_3 > 0$.

$A_1A_2 - A_3 = (g + h + 2a + f + b)(b(g + h + 2a + f) + (f + a)(g + h + a) - fh - ejc) + jc(fd + e(g + h + a)) + bfh - b(f + a)(g + h + a)$.

We introduce $x = f + a$ and $y = g + h + a$, so that

$$\begin{aligned} A_1A_2 - A_3 &= (x + y + b)(b(x + y) + xy - fh - ejc) + jc(fd + ey) + bfh - bxy \\ &= (x + y)(xy - fh) + b(xy - fh) + (x + y)^2b + b^2(x + y) - (x + y)ejc - bejc \\ &\quad + jc(fd + ey) - b(xy - fh) \\ &= (x + y)(xy - fh) - b(xy - fh) + (x + y)^2b + b^2(x + y) - (x + y)ejc - bejc \\ &\quad + b(xy - fh)(1 - R^2) \\ &= (x + y)(xy - fh) + (x + y + b)(b(x + y) - ejc) + b(xy - fh)R^2 > 0. \end{aligned}$$

Therefore, all roots has negative real parts, then the disease-free equilibrium point is locally asymptotically stable if $R < 1$.

Theorem 2.2.2. *For system (2.32)-(2.34), the disease-free equilibrium A_0 is globally asymptotically stable if $R \leqslant 1$.*

Proof. Let use define the following Lyapunov function [12, 13]

$$L = \alpha z_1 + \beta z_2 + \gamma w, \tag{2.35}$$

where $\alpha = e(g+h+a)+df$, $\beta = d(f+a)+eh$, and $\gamma = (f+a)(g+h+a)-fh$. It is easy to see that α, β, and γ are all positive. The derivative of L is given by

$$\begin{aligned}\dot{L} &= \alpha\dot{z}_1 + \beta\dot{z}_2 + \gamma\dot{w}\\ &= \alpha[jc(1-z_1-z_2)w + hz_2 - (f+a)z_1] + \beta[fz_1 - (g+h+a)z_2]\\ &+ \gamma[e(1-w)z_1 + d(1-w)z_2 - bw]\\ &= (\alpha jc - \gamma b)w - [\alpha(f+a) - \beta f - \gamma e]z_1 + [\alpha h - \beta(g+h+a)\\ &+ \gamma d]z_2 - (\alpha jc + \gamma e)z_1 w - (\alpha jc + \gamma d)z_2 w\\ &= -(\alpha jc + \gamma e)z_1 w - (\alpha jc + \gamma d)z_2 + \frac{(R^2-1)w}{[(f+a)(g+h+a)-fh]b}.\end{aligned} \tag{2.36}$$

If $R \leqslant 1$, then $(R^2-1)/[(f+a)(g+h+a)-fh]b \leqslant 0$.

As we know that $-(\alpha jc + \gamma e) < 0$ and $-(\alpha jc + \gamma d) < 0$, so we obtain $\dot{L} \leqslant 0$. Furthermore, $\dot{L} = 0$ only if $w = 0$ or $R = 1$. The maximum invariant set in $\{(z_1,\ z_2,\ w) : \dot{L} = 0\}$ is the singleton A_0. By LaSalle's Invariance Principle [11], A_0 is globally asymptotically stable in Ω. □

2. **For existence of the Endemic Equilibrium :**

Theorem 2.2.3. *If $R > 1$, system (2.32)-(2.34) has a unique endemic equilibrium $A^* = (z_1^*,\ z_2^*,\ w^*)$, where*

$$\begin{aligned}z_1^* &= ((g+h+a)[(f+a)(g+h+a)-fh] \times b(R^2-1)) \times ([(f+a)(g+h+a)-fh]\\ &\times [e(g+h+a)+df] + (g+h+a+f))^{-1},\end{aligned} \tag{2.37}$$

$$\begin{aligned}z_2^* &= \frac{f}{g+h+a}z_1,\\ w^* &= \frac{e(g+h+a)+df]z_1}{[e(g+h+a)+df]z_1 + b(g+h+a)}.\end{aligned}$$

Proof. It follows from system (2.32)-(2.34) that

$$\begin{aligned}jc(1-z_1^*-z_2^*)w^* + hz_2^* - (f+a)z_1^* &= 0,\\ fz_1^* - (g+h+a)z_2^* &= 0,\\ e(1-w^*)z_1^* + d(1-w^*)z_2^* - bw &= 0.\end{aligned} \tag{2.38}$$

From the second equation of (2.38), we obtain

$$z_2^* = \frac{f}{g+h+a} z_1^*. \tag{2.39}$$

Substituting z_2^* into the third equation of (2.38), we have

$$w^* = \frac{[e(g+h+a)+df]z_1^*}{[e(g+h+a)+df]z_1^* + b(g+h+a)}. \tag{2.40}$$

Then substituting (2.39) and (2.40) into first equation of (2.38), we get

$$\begin{aligned} z_1^* = {} & ((g+h+a)[(f+a)(g+h+a)-fh] \times b(R^2-1)) \times ([(f+a)(g+h+a)-fh] \\ & \times [e(g+h+a)+df] + (g+h+a+f))^{-1}. \end{aligned} \tag{2.41}$$

Hence, if $R \leqslant 1$, there is no positive root of (2.41), while if $R > 1$ there is one positive root. □

Summary: Ross-Macdonald models are best defined by a consensus set of assumptions. The mathematical model is just one part of a theory for the dynamics and control of mosquito-transmitted pathogens that also includes epidemiological and entomological concepts and metrics for measuring transmission. It has two equilibrium points:

- Disease-free Equilibrium Point $A = (0, 0, 0)$.
- Endemic equilibrium point.

The next generation matrix method proposed in [19] will be used to determine the fundamental reproduction number of the system (2.32)-(2.34).

The disease-free equilibrium point is locally asymptotically stable if $R < 1$ and globally if $R \leqslant 1$. The system has a unique endemic equilibrium point if $R > 1$.

MODEL 1:

In this model is used for computing the amount of susceptible, infected, recovered people in a population. It uses some specific assumption.

If the initial conditions are positives, then the solutions of the system are positives for all $t > 0$.

This model has two equilibrium points which are:

$$A_0(s = 1,\ i = 0) \text{ and } A^*\left(s = \frac{c+b}{a},\ i = \frac{b(a-c-b)}{a(c+b)}\right).$$

We use the Linearisation method to know if the equilibrium points are stable or no, we found that the disease-free equilibrium point is locally asymptotically stable and if $r < 1$ it's globally asymptotically stable on omega, the Endemic equilibrium point is locally stable and it's globally asymptotically stable on Ω_1 by using Lyapunov stability point.

MODEL 2:

In the SIRV model we used the same previous assumption with adding the vaccinations. The differential equations for v can be solved directly, independently of the others, and we get $V(t) = V(0) + p(1 - e - bt)$.

- Disease-free equilibrium point $A_0(S = 1 - p, I = 0, V = p)$.
- Endemic equilibrium point $A_e\left(S = \frac{b+c}{a},\ I = \frac{b(a(1-p)-c-b)}{a(c+b)},\ V = p\right)$.

We found that the disease-free equilibrium point is stable if λ_3 is negative and it's globally asymptotically stable on Ω_1 by using Lasalle invariance principle.

MODEL 3:

Ross-Macdonald models are best defined by a consensus set of assumptions. The mathematical model is just one part of a theory for the dynamics and control of mosquito-transmitted pathogens that also includes epidemiological and entomological concepts and metrics for measuring transmission. It has two equilibrium points:

- Disease-free Equilibrium Point $A = (0, 0, 0)$.
- Endemic equilibrium point.

The next generation matrix method proposed in [19] will be used to determine the fundamental reproduction number of the system (2.32)-(2.34).
The disease-free equilibrium point is locally asymptotically stable if $R < 1$ and globally if $R \leqslant 1$. The system has a unique endemic equilibrium point if $R > 1$.

CHAPTER 3

DISCRETIZATION BY FINITE DIFFERENCE METHOD OF SIR-TYPE PROCESSES

3.1 Finite difference approximation

3.1.1 Discrete formulation for First model

We consider the system:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = bN - \frac{aI(t)S(t)}{N} - bS(t), \tag{3.1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{aI(t)S(t)}{N} - cI(t) - bI(t), \tag{3.2}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = cI(t) - bR(t). \tag{3.3}$$

To discretize the given system of ordinary differential equations (ODEs) using the forward Euler method with a uniform grid, we will replace the continuous derivatives with their discrete approximations $\frac{dX}{dt} \simeq \frac{X_{n+1}-X_n}{\Delta t}$, where Δt is the uniform time step and X_n represents

the value of variable X at time $t_n = n\Delta t$.

$$\frac{S_{n+1} - S_n}{\Delta t} = bN - \frac{aI_nS_n}{N} - bS_n, \tag{3.4}$$

$$\frac{I_{n+1} - I_n}{\Delta t} = \frac{aI_nS_n}{N} - cI_n - bI_n, \tag{3.5}$$

$$\frac{R_{n+1} - R_n}{\Delta t} = cI_n - bR_n. \tag{3.6}$$

To study the nonnegativity of the given discrete SIR model, we need to ensure that all variables $S_n,\ I_n,\ R_n$ remain nonnegative for all n, given that the initial values are nonnegative. Here are the update equations again:

$$S_{n+1} = S_n + \Delta t\left(bN - \frac{aI_nS_n}{N} - bS_n\right), \tag{3.7}$$

$$I_{n+1} = I_n + \Delta t\left(\frac{aI_nS_n}{N} - cI_n - bI_n\right), \tag{3.8}$$

$$R_{n+1} = R_n + \Delta t\left(cI_n - bR_n\right). \tag{3.9}$$

3.1.2 Discrete SIRV model

We consider the system of the SIRV model:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = bN - \frac{aI(t)S(t)}{N} - bS(t), \tag{3.10}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \frac{aI(t)S(t)}{N} - cI(t) - bI(t), \tag{3.11}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = cI(t) - bR(t) \tag{3.12}$$

$$\frac{\mathrm{d}V}{\mathrm{d}t} = pb - bV_n. \tag{3.13}$$

To discretize the given system of ordinary differential equations (ODEs) using the forward Euler method with a uniform grid, we will replace the continuous derivatives with their discrete approximations $\frac{dX}{dt} \simeq \frac{X_{n+1}-X_n}{\Delta t}$, where Δt is the uniform time step and X_n represents the value of variable X at time $t_n = n\Delta t$.

$$\frac{S_{n+1} - S_n}{\Delta t} = (1-p)b - aI_nS_n - bS_n, \tag{3.14}$$

$$\frac{I_{n+1} - I_n}{\Delta t} = aI_nS_n - cI_n - bI_n, \tag{3.15}$$

$$\frac{R_{n+1} - R_n}{\Delta t} = cI_n - bR_n, \tag{3.16}$$

$$\frac{V_{n+1} - V_n}{\Delta t} = pb - bV_n. \tag{3.17}$$

To study the nonnegativity of the given discrete SIR model, we need to ensure that all variables $S_n,\ I_n,\ R_n,\ V_n$ remain nonnegative for all n, given that the initial values are nonnegative.

Here are the update equations again:

$$S_{n+1} = S_n + \Delta t\left((1-p)b - aI_nS_n - bS_n\right), \tag{3.18}$$

$$I_{n+1} = I_n + \Delta t\left(aI_nS_n - cI_n - bI_n\right), \tag{3.19}$$

$$R_{n+1} = R_n + \Delta t\left(cI_n - bR_n\right), \tag{3.20}$$

$$V_{n+1} = V_n + \Delta t\left(pb - bV_n\right). \tag{3.21}$$

3.2 Qualitative properties of the discrete First model (nonnegativity, monotonicity)

3.2.1 Nonnegativity analysis of the discrete First model

1. **Non-negativity of S_{n+1}**

$$\begin{aligned} S_{n+1} &= S_n + \Delta t\left(bN - \frac{aI_nS_n}{N} - bS_n\right) \\ &= S_n\left(1 - \Delta t\left(\frac{aI_n}{N} + b\right)\right) + \Delta tbN. \end{aligned}$$

We have $S_n \geqslant 0$, and $bN \geqslant 0$, so for nonnegativity of S_{n+1}, we just need the term $1 - \Delta t\left(\frac{aI_n}{N} + b\right) \geqslant 0$, so:

$$1 - \Delta t\left(\frac{aI_n}{N} + b\right) \geqslant 0 \implies \Delta t\left(\frac{aI_n}{N} + b\right) \leqslant 1 \implies \Delta t \leqslant \frac{1}{\frac{aI_n}{N} + b}.$$

2. **Non-negativity of I_{n+1}**

$$\begin{aligned} I_{n+1} &= I_n + \Delta t\left(\frac{aI_nS_n}{N} - cI_n - bI_n\right) \\ &= I_n\left(1 + \Delta t\left(\frac{aS_n}{N} - c - b\right)\right). \end{aligned}$$

from the hypothesis we have $I_n \geqslant 0$, than it is enough to study the nonnegativity of $1 + \Delta t\left(\frac{aS_n}{N} - (c+b)\right) \geqslant 0$ for ensuring that $I_{n+1} \geqslant 0$, so:

$$1 + \Delta t\left(\frac{aS_n}{N} - (c+b)\right) \geqslant 0.$$

- If $\frac{aS_n}{N} - (c+b) \geqslant 0$, then $1 + \Delta t\left(\frac{aS_n}{N} - (c+b)\right) \geqslant 1$, this gives us $I_{n+1} \geq I_n \geqslant 0$.
- If $\frac{aS_n}{N} - (c+b) < 0$, then $(c+b) - \frac{aS_n}{N} > 0$. Then, the condition becomes $1 \geqslant \Delta t\left((c+b) - \frac{aS_n}{N}\right)$, therefore $\Delta t \leqslant \frac{1}{(c+b)-\frac{aS_n}{N}}$.

3. **Non-negativity of R_{n+1}**

$$\begin{aligned} R_{n+1} &= R_n + \Delta t\,(cI_n - bR_n) \\ &= R_n\,(1 - \Delta t b) + \Delta t c I_n. \end{aligned}$$

Here, $R_n,\ I_n \geqslant 0$, so for $R_{n+1} \geqslant 0$, we obtain:

$$1 - \Delta t b \geqslant 0 \Rightarrow \Delta t \leqslant \frac{1}{b}.$$

Corollary 3.2.1. *To preserve nonnegativity of the system, time step Δt must achive:*

$$\Delta t \leqslant \min\left\{\frac{1}{\frac{aI_n}{N} + b},\ \frac{1}{c + b - \frac{aS_n}{N}},\ \frac{1}{b}\right\}.$$

3.2.2 Monotonicity analysis of the discrete First model

We investigate whether each sequence is increasing, decreasing, or constant (its monotony) by examining the sign of $X_{n+1} - X_n$.
We have $\Delta t,\ S_n,\ I_n,\ R_n,\ a,\ b$ and c. They are positive.

1. **Monotony of S_n :**

$$S_{n+1} - S_n = \Delta t\left(bN - \frac{aI_nS_n}{N} - bS_n\right).$$

The monotony of S_n depends on the sign of the term $\left(bN - \frac{aI_nS_n}{N} - bS_n\right)$ so:

- S_n is increasing if $bN - \frac{aI_nS_n}{N} - bS_n > 0 \Rightarrow\ bN > \frac{aI_nS_n}{N} + bS_n$.
- S_n is decreasing if $bN - \frac{aI_nS_n}{N} - bS_n < 0 \Rightarrow\ bN < \frac{aI_nS_n}{N} + bS_n$.
- S_n is constant if $bN - \frac{aI_nS_n}{N} - bS_n = 0 \Rightarrow\ bN = \frac{aI_nS_n}{N} + bS_n$.

2. **Monotony of I_n :**

$$I_{n+1} - I_n = \Delta t\left(\frac{aI_nS_n}{N} - cI_n - bI_n\right) = \Delta t I_n\left(\frac{aS_n}{N} - c - b\right).$$

Then The monotony of I_n is related to the sign of $\left(\frac{aS_n}{N} - c - b\right)$:

- If $I_n = 0$, then $I_{n+1} - I_n = 0$, so I_n is constant.
- If $I_n > 0$:
  - I_n is increasing if $\frac{aS_n}{N} - c - b > 0 \Rightarrow \ S_n > \frac{N(c+b)}{a}$.
  - I_n is decreasing if $\frac{aS_n}{N} - c - b < 0 \Rightarrow \ S_n < \frac{N(c+b)}{a}$.
  - I_n is constant if $\frac{aS_n}{N} - c - b = 0 \Rightarrow \ S_n = \frac{N(c+b)}{a}$.

3. **Monotony of R_n** :

$$R_{n+1} - R_n = \Delta t(cI_n - bR_n).$$

The sign of $(cI_n - bR_n)$ determines the monotonic behavior of R_n, then:

- R_n is increasing if $cI_n - bR_n > 0 \Rightarrow \ cI_n > bR_n$.
- R_n is decreasing if $cI_n - bR_n < 0 \Rightarrow \ cI_n < bR_n$.
- R_n is constant if $cI_n - bR_n = 0$, or $cI_n = bR_n$.

3.2.3 Nonnegativity analysis of the discrete SIRV model

Let's consider each one individually.

Assumptions

We assume:

- $S_n,\ I_n,\ R_n,\ V_n \geqslant 0$.
- Parameters $a,\ b,\ c,\ p,\ \Delta t \geqslant 0$.
- $0 \leqslant p \leqslant 1$.

1. **Nonnegativity of S_{n+1}**

$$S_{n+1} = S_n + \Delta t\left((1-p)b - aI_nS_n - bS_n\right).$$

$$S_{n+1} = S_n(1 - \Delta t(aI_n + b)) + \Delta t(1-p)b.$$

Since $S_n \geqslant 0$ by the inductive hypothesis, and $(1-p)b \geqslant 0$ (as $0 \leqslant p \leqslant 1$ and $b \geqslant 0$), for $S_{n+1} \geqslant 0$, so a sufficient condition is:

the term $S_n(1 - \Delta t(aI_n + b)) \geqslant 0$.

Since $S_n \geqslant 0$ than:

$$1 - \Delta t(aI_n + b) \geqslant 0 \implies \Delta t(aI_n + b) \leqslant 1 \implies \Delta t \leqslant \frac{1}{aI_n + b}.$$

If $S_n = 0$, then $S_{n+1} = \Delta t(1 - p)b \geqslant 0$. So to guarantee nonnegativity, we need:

$$\Delta t \leqslant \frac{1}{aI_n + b}. \tag{3.22}$$

2. **Non-negativity of I_{n+1}**

$$\begin{aligned} I_{n+1} &= I_n + \Delta t(aI_nS_n - cI_n - bI_n) \\ &= I_n\left(1 + \Delta t(aS_n - c - b)\right). \end{aligned}$$

Since $I_n \geqslant 0$ by the inductive hypothesis, for $I_{n+1} \geqslant 0$, we need:

$$1 + \Delta t(aS_n - c - b) \geqslant 0.$$

- If $aS_n - c - b \geqslant 0$, then $1 + \Delta t(aS_n - c - b) \geqslant 1$, so $I_{n+1} \geqslant I_n \geqslant 0$. Non-negativity is maintained.
- If $aS_n - c - b < 0$, then $c + b - aS_n > 0$. The condition becomes $1 \geqslant \Delta t(c + b - aS_n)$, so $\Delta t \leqslant \frac{1}{c+b-aS_n}$.

So to guarantee nonnegativity, should:

$$\Delta t \leqslant \frac{1}{c + b - aS_n}.$$

3. **Non-negativity of R_{n+1}**

$$\begin{aligned} R_{n+1} &= R_n + \Delta t(cI_n - bR_n) \\ &= R_n(1 - \Delta tb) + \Delta tcI_n. \end{aligned}$$

We have $R_n \geqslant 0$ and $I_n \geqslant 0$ (from the hypothesis), and $c \geqslant 0$, the term $\Delta tcI_n \geqslant 0$. So for $R_{n+1} \geqslant 0$, a sufficient condition is:

$$1 - \Delta tb \geqslant 0 \implies \Delta tb \leqslant 1 \implies \Delta t \leqslant \frac{1}{b} \ (\ \textit{if}\ b > 0).$$

This is always nonnegative if:

$$\Delta t \leqslant \frac{1}{b} \ (\ \textit{if}\ b > 0).$$

4. **Non-negativity of V_{n+1}**

$$\begin{aligned} V_{n+1} &= V_n + \Delta t(pb - bV_n) \\ &= V_n(1 - \Delta tb) + \Delta tpb. \end{aligned}$$

To ensure $V_{n+1} \geqslant 0$, a sufficient condition is:

$$1 - \Delta tb \geqslant 0 \implies \Delta tb \leqslant 1 \implies \Delta t \leqslant \frac{1}{b} \ (\ \textit{if}\ b > 0).$$

This is also always nonnegative if:

$$\Delta t \leqslant \frac{1}{b} \ (\ \textit{if}\ b > 0).$$

Corollary 3.2.2. *To ensure nonnegativity of the system, the time step Δt must satisfy:*

$$\Delta t \leqslant \min\left\{\frac{1}{aI_n + b}, \frac{1}{c + b - aS_n}, \frac{1}{b}\right\}.$$

3.2.4 Monotonicity analysis of the discrete SIRV model

1. **Monotony of S_n :**

$$S_{n+1} - S_n = \Delta t((1-p)b - aI_nS_n - bS_n).$$

The monotony of S_n depends on the sign of the term $((1-p)b - aI_nS_n - bS_n)$ **:**

- S_n is increasing if $(1-p)b - aI_nS_n - bS_n > 0 \Rightarrow\ (1-p)b > S_n(aI_n + b)$.
- S_n is decreasing if $(1-p)b - aI_nS_n - bS_n < 0 \Rightarrow\ (1-p)b < S_n(aI_n + b)$.
- S_n is constant if $(1-p)b - aI_nS_n - bS_n = 0 \Rightarrow\ (1-p)b = S_n(aI_n + b)$.

2. **Monotony of I_n :**

$$I_{n+1} - I_n = \Delta t(aI_nS_n - cI_n - bI_n) = \Delta tI_n(aS_n - c - b).$$

The sign of $I_n(aS_n - c - b)$ determines the monotonic behavior of I_n, then:

- If $I_n = 0$, then I_n remains constant.
- If $I_n > 0$ **:**

- I_n is increasing if $aS_n - c - b > 0 \Rightarrow\ S_n > \frac{c+b}{a}$.
- I_n is decreasing if $aS_n - c - b < 0 \Rightarrow\ S_n < \frac{c+b}{a}$.
- I_n is constant if $aS_n - c - b = 0 \Rightarrow\ S_n = \frac{c+b}{a}$.

3. **Monotony of** R_n :

$$R_{n+1} - R_n = \Delta t(cI_n - bR_n).$$

Then The monotony of R_n is related to the sign of $(cI_n - bR_n)$ **:**

- R_n is increasing if $cI_n - bR_n > 0 \Rightarrow\ cI_n > bR_n$.
- R_n is decreasing if $cI_n - bR_n < 0 \Rightarrow\ cI_n < bR_n$.
- R_n is constant if $cI_n - bR_n = 0 \Rightarrow\ cI_n = bR_n$.

4. **Monotony of** V_n :

$$V_{n+1} - V_n = \Delta t(pb - bV_n) = \Delta tb(p - V_n).$$

The sign of $(p - V_n)$ determines the monotonic behavior of V_n, then:

- V_n is increasing if $p - V_n > 0 \Rightarrow\ V_n < p$.
- V_n is decreasing if $p - V_n < 0 \Rightarrow\ V_n > p$.
- V_n is constant if $p - V_n = 0 \Rightarrow\ V_n = p$.

3.3 Numerical simulations

3.3.1 Numerical simulations of the SIRV model

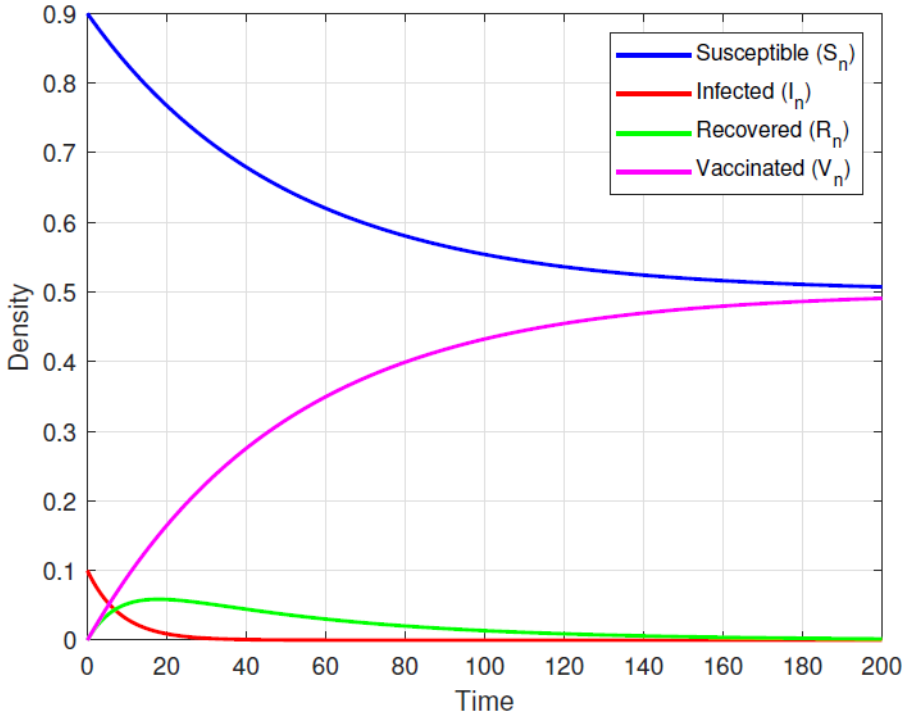

Figure 3.1: SIRV model simulation: Densities of compartments over time.

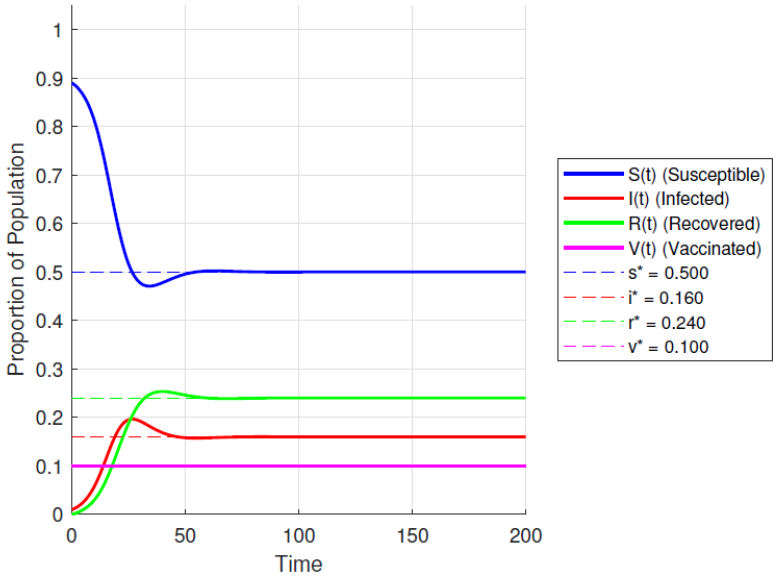

Figure 3.2: SIRV model with Vaccination at endemic equilibrium point with (a = 0.5, b = 0.1, c = 0.15, p = 0.1, Δt = 0.1).

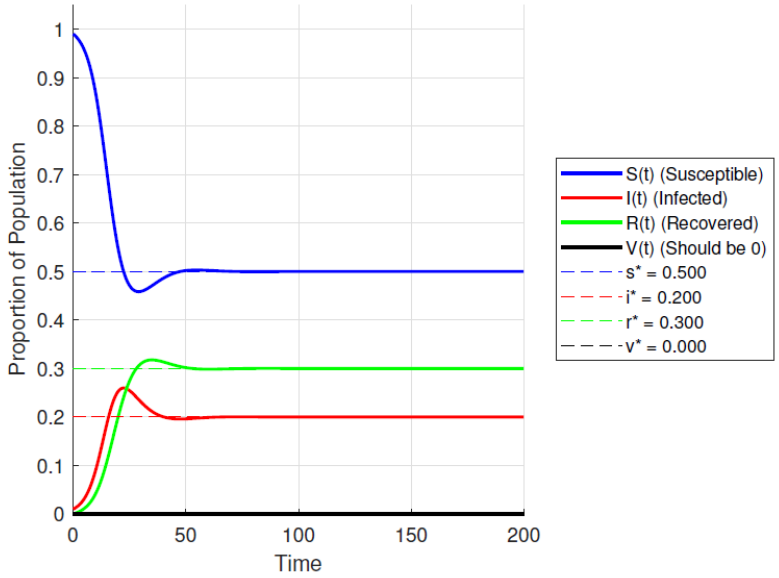

Figure 3.3: SIRV model simulation without Vaccination at endemic equilibrium point with (p = 0, a = 0.5, b = 0.1, c = 0.15, Δt = 0.1).

CHAPTER 4

DISCRETIZATION BY STANDARD FINITE DIFFERENCE TECHNIQUES

4.1 Discrete Malaria model (vector-type propagation)

We consider the system of Malaria Model[8] :

$$\frac{dS_h(t)}{dt} = aN - \frac{cS_hI_m}{N} + gR_h - aS_h, \tag{4.1}$$

$$\frac{dI_h(t)}{dt} = \frac{cS_hI_m}{N} + hR_h - (f+a)I_h, \tag{4.2}$$

$$\frac{dR_h(t)}{dt} = fI_h - (g+h+a)\,R_h, \tag{4.3}$$

$$\frac{dS_m(t)}{dt} = bM - \frac{eS_mI_h}{N} - \frac{dS_mR_h}{N} - bS_m, \tag{4.4}$$

$$\frac{dI_m(t)}{dt} = \frac{eS_mI_h}{N} + \frac{dS_mR_h}{N} - bI_m. \tag{4.5}$$

To discretize the given system of ordinary differential equations (ODEs) using the forward Euler method with a uniform grid, we will replace the continuous derivatives with their discrete approximations $\frac{dX}{dt} \simeq \frac{X_{n+1}-X_n}{\Delta t}$, where Δt is the uniform time step and X_n represents

the value of variable X at time $t_n = n\Delta t$.

$$\frac{S_h^{n+1} - S_h^n}{\Delta t} = aN - \frac{cS_h^n I_m^n}{N} + gR_h^n - aS_h^n, \tag{4.6}$$

$$\frac{I_h^{n+1} - I_h^n}{\Delta t} = \frac{cS_h^n I_m^n}{N} + hR_h^n - (f+a)I_h^n, \tag{4.7}$$

$$\frac{R_h^{n+1} - R_h^n}{\Delta t} = fI_h^n - (g+h+a)\,R_h^n, \tag{4.8}$$

$$\frac{S_m^{n+1} - S_m^n}{\Delta t} = bM - \frac{eS_m^n I_h^n}{N} - \frac{dS_m^n R_h^n}{N} - bS_m^n, \tag{4.9}$$

$$\frac{I_m^{n+1} - I_m^n}{\Delta t} = \frac{eS_m^n I_h^n}{N} + \frac{dS_m^n R_h^n}{N} - bI_m^n. \tag{4.10}$$

So

$$S_h^{n+1} = S_h^n + \Delta t\left(aN - \frac{cS_h^n I_m^n}{N} + gR_h^n - aS_h^n\right), \tag{4.11}$$

$$I_h^{n+1} = I_h^n + \Delta t\left(\frac{cS_h^n I_m^n}{N} + hR_h^n - (f+a)I_h^n\right), \tag{4.12}$$

$$R_h^{n+1} = R_h^n + \Delta t\left(fI_h^n - (g+h+a)\,R_h^n\right), \tag{4.13}$$

$$S_m^{n+1} = S_m^n + \Delta t\left(bM - \frac{eS_m^n I_h^n}{N} - \frac{dS_m^n R_h^n}{N} - bS_m^n\right), \tag{4.14}$$

$$I_m^{n+1} = I_m^n + \Delta t\left(\frac{eS_m^n I_h^n}{N} + \frac{dS_m^n R_h^n}{N} - bI_m^n\right). \tag{4.15}$$

4.2 Stability analysis of the discrete equilibrium point for the Ross-Macdonald model

4.2.1 Linearize Around the Equilibrium

We compute the Jacobian matrix of the discretized system at the equilibrium point. The Jacobian matrix is derived by finding the partial derivatives of each discretized equation with respect to:

$$J = \left.\frac{\partial F}{\partial X}\right|_{X=X^*}. \tag{4.16}$$

4.2.1.1 Eigenvalue analysis

The characteristic equation is given by:

$$\det(J - \lambda I) = 0. \tag{4.17}$$

The solutions to this equation are the eigenvalues, which determine the stability of the discrete system:

If $|\lambda_i| < 1$, for all eigenvalues, the system is stable.

If $|\lambda_i| \geqslant 1$, for any eigenvalue, the system is unstable.

4.2.1.2 Jacobian matrix of the discrete system

The Jacobian matrix is:

$$J = I + \Delta t \cdot \begin{pmatrix} -\frac{cI_m}{N} - a & 0 & g & 0 & -\frac{cS_h}{N} \\ \frac{cI_m}{N} & -(f+a) & h & 0 & \frac{cS_h}{N} \\ 0 & f & -(g+h+a) & 0 & 0 \\ 0 & -\frac{eS_m}{N} & -\frac{dS_m}{N} & -\frac{eI_h}{N} - b - \frac{dR_h}{N} & 0 \\ 0 & \frac{eS_m}{N} & \frac{dS_m}{N} & \frac{eI_h}{N} + \frac{dR_h}{N} & -b \end{pmatrix}, \tag{4.18}$$

where I is the 5×5 identity matrix.

The jacobian matrix at the Disease-Free Equilibrium point $(N, 0, 0, M, 0)$:

$$J = I + \Delta t \cdot \begin{pmatrix} -a & 0 & g & 0 & -c \\ 0 & -(f+a) & h & 0 & c \\ 0 & f & -(g+h+a) & 0 & 0 \\ 0 & 0 & 0 & -b & 0 \\ 0 & 0 & 0 & 0 & -b \end{pmatrix}. \tag{4.19}$$

The matrix J is given by $J = I + \Delta t \cdot A$, where I is the identity matrix and A is the matrix:

$$A = \begin{pmatrix} -a & 0 & g & 0 & -c \\ 0 & -(f+a) & h & 0 & c \\ 0 & f & -(g+h+a) & 0 & 0 \\ 0 & 0 & 0 & -b & 0 \\ 0 & 0 & 0 & 0 & -b \end{pmatrix}.$$

The eigenvalues of J are $1 + \Delta t \cdot \lambda_A$.

First, we find the eigenvalues of A by solving the characteristic equation $\det(A - \lambda I) = 0$:

$$\det(A-\lambda I) = \begin{vmatrix} -a-\lambda & 0 & g & 0 & -c \\ 0 & -(f+a)-\lambda & h & 0 & c \\ 0 & f & -(g+h+a)-\lambda & 0 & 0 \\ 0 & 0 & 0 & -b-\lambda & 0 \\ 0 & 0 & 0 & 0 & -b-\lambda \end{vmatrix} = 0.$$

We can expand the determinant. Due to the zeros in the last two rows, this simplifies significantly.

Expanding along the last row:

$$(-b-\lambda)\cdot\det\begin{vmatrix} -a-\lambda & 0 & g & 0 \\ 0 & -(f+a)-\lambda & h & 0 \\ 0 & f & -(g+h+a)-\lambda & 0 \\ 0 & 0 & 0 & -b-\lambda \end{vmatrix} = 0.$$

Expanding the 4×4 determinant along its last row:

$$(-b-\lambda)(-b-\lambda)\cdot\det\begin{vmatrix} -a-\lambda & 0 & g \\ 0 & -(f+a)-\lambda & h \\ 0 & f & -(g+h+a)-\lambda \end{vmatrix} = 0.$$

For that

$$(-b-\lambda)^2(-a-\lambda)\left[(-(f+a)-\lambda)(-(g+h+a)-\lambda)-(h)(f)\right]-0+g(0-0)=0.$$

So the characteristic equation for A is:

$$(-b-\lambda)^2(-a-\lambda)\left[((f+a)+\lambda)((g+h+a)+\lambda)-hf\right]=0.$$

From this equation, we find the eigenvalues of A:

- From $(-b-\lambda)^2 = 0$, we get $\lambda_1 = -b$ (this eigenvalue has a multiplicity of 2).
- From $(-a-\lambda) = 0$, we get $\lambda_2 = -a$.

4.2 Stability analysis of the discrete equilibrium point for the Ross-Macdonald model

- From $((f+a)+\lambda)((g+h+a)+\lambda)-hf=0$:

  Let $X=\lambda+a$. The equation becomes $(f+X)(g+h+X)-hf=0$.

  $X^2+(g+h)X+fX+f(g+h)-hf=0$.

  $X^2+(f+g+h)X+fg+fh-hf=0$.

  $X^2+(f+g+h)X+fg=0$.

  Using the quadratic formula to solve for X:

  Suppose $(f+g+h)^2-4fg>0$.

  $X=\frac{-(f+g+h)\pm\sqrt{(f+g+h)^2-4fg}}{2}$.

  Since $X=\lambda+a,$ *we have* $\lambda=X-a$.

  So the other two eigenvalues of A are: $\lambda_{3,4}=\frac{-(f+g+h)\pm\sqrt{(f+g+h)^2-4fg}}{2}-a$.

Then the eigenvalues of A are:

- $\lambda_{A1}=-b$ (this eigenvalue has a multiplicity of 2).
- $\lambda_{A2}=-a$.
- $\lambda_{A3}=\frac{-(f+g+h)+\sqrt{(f+g+h)^2-4fg}}{2}-a$.
- $\lambda_{A4}=\frac{-(f+g+h)-\sqrt{(f+g+h)^2-4fg}}{2}-a$.

Now, the eigenvalues of $J=I+\Delta t\cdot A$, which are $\Lambda=1+\Delta t\cdot\lambda_A$.

- $\Lambda_1=1+\Delta t(-b)=1-b\Delta t$ (this eigenvalue has a multiplicity of 2).
- $\Lambda_2=1+\Delta t(-a)=1-a\Delta t$.
- $\Lambda_3=1+\Delta t\left(\frac{-(f+g+h)+\sqrt{(f+g+h)^2-4fg}}{2}-a\right)$,
  $\Lambda_3=1-a\Delta t+\frac{\Delta t}{2}\left(-(f+g+h)+\sqrt{(f+g+h)^2-4fg}\right)$.
- $\Lambda_4=1+\Delta t\left(\frac{-(f+g+h)-\sqrt{(f+g+h)^2-4fg}}{2}-a\right)$,
  $\Lambda_4=1-a\Delta t+\frac{\Delta t}{2}\left(-(f+g+h)-\sqrt{(f+g+h)^2-4fg}\right)$.

These are the four eigenvalues of the matrix J.

For stability, each eigenvalue must be less than 1.

The conditions are as follows:

- For $\Lambda_1 = 1 - b\Delta t$.
  should be $|b\Delta t - 1| < 1 \Rightarrow\ 0 < \Delta t < \dfrac{2}{b}$.

- For $\Lambda_2 = 1 - a\Delta t$
  should be $|a\Delta t - 1| < 1 \Rightarrow\ 0 < \Delta t < \dfrac{2}{a}$.

- For
  $\Lambda_3 = 1 - a\Delta t + \frac{\Delta t}{2}\left(-(f+g+h) + \sqrt{(f+g+h)^2 - 4fg}\right)$.
  should be
  $\left|-1 + \frac{\Delta t}{2}\left(2a(f+g+h) - \sqrt{(f+g+h)^2 - 4fg}\right)\right| < 1,$
  $0 < \Delta t < \dfrac{4}{2a + (f+g+h) - \sqrt{(f+g+h)^2 - 4fg}},$
  $0 < \Delta t < \beta_1$, where $\beta_1 = \dfrac{4}{2a + (f+g+h) - \sqrt{(f+g+h)^2 - 4fg}}$.

- For $\Lambda_4 = 1 - a\Delta t + \frac{\Delta t}{2}\left(-(f+g+h) - \sqrt{(f+g+h)^2 - 4fg}\right)$.
  should be
  $\left|-1 + \frac{\Delta t}{2}\left(2a + (f+g+h) + \sqrt{(f+g+h)^2 - 4fg}\right)\right| < 1,$
  $0 < \Delta t < \dfrac{4}{2a + (f+g+h) + \sqrt{(f+g+h)^2 - 4fg}}.$
  $0 < \Delta t < \beta_2$, where $\beta_2 = \dfrac{4}{2a + (f+g+h) + \sqrt{(f+g+h)^2 - 4fg}}$.

Theorem 4.2.1. *To achieve stability of the discretized model at the disease equilibrium point, the time step Δt must satisfy:*

$$\Delta t \leqslant \min\left\{\frac{2}{b},\ \frac{2}{a},\ \beta_1,\ \beta_2\right\}.$$

where $\beta_1 = \dfrac{4}{2a + (f+g+h) - \sqrt{(f+g+h)^2 - 4fg}}$ *and*
$\beta_2 = \dfrac{4}{2a + (f+g+h) + \sqrt{(f+g+h)^2 - 4fg}}$.

4.3 Numerical simulations

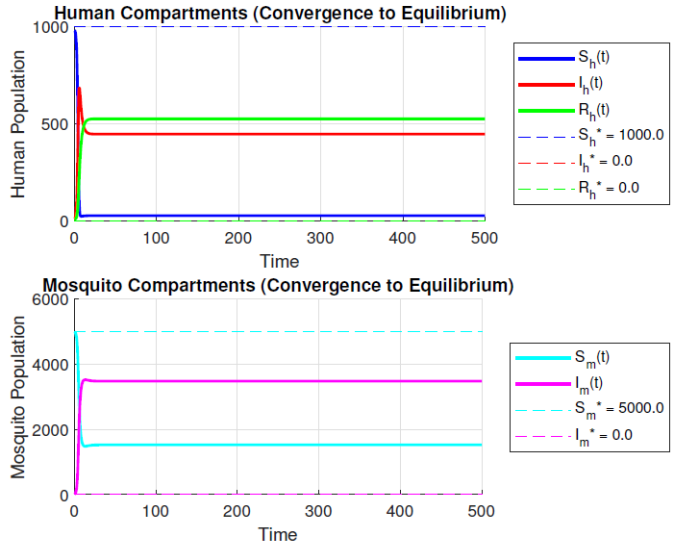

Figure 4.1: Human and Mosquito compartments model at endemic equilibrium with (N = 1000, a = 0.02, c = 0.75, g = 0.1, h = 0.05, f = 0.2, μ = 0.02, M = 5000, b = 0.1, e = 0.5, d = 0.01).

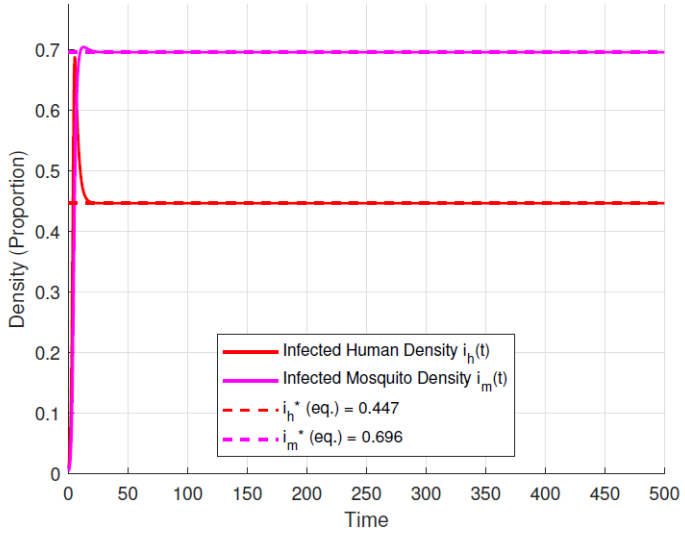

Figure 4.2: Infected Human and Mosquito Densities.

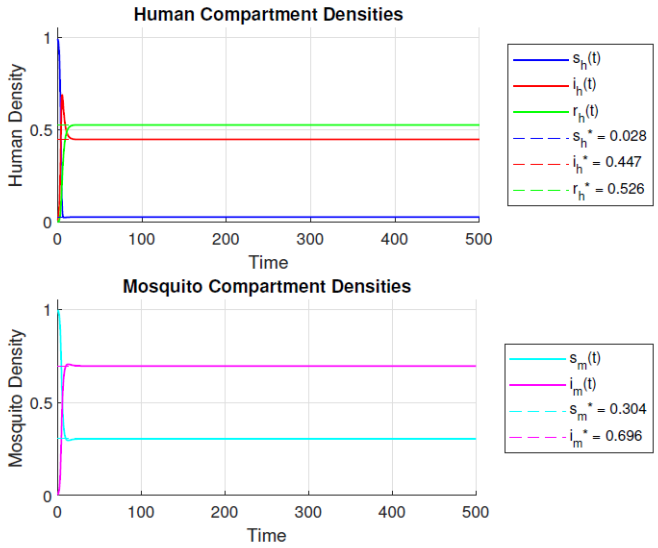

Figure 4.3: Density based Human-Mosquito model.

CONCLUSION

This thesis successfully demonstrates the application of mathematical modeling to analyze and understand the propagation of epidemics through various theoretical frameworks. The research systematically investigates SIR, SIRV, and vector-borne disease models, consistently determining the equilibrium points and rigorously analyzing their local and global stability. Key findings revolve around the conditions, often dictated by the basic reproduction number and model-specific parameters like vaccination rates, that determine whether a disease will die out or persist endemically within a population.

Furthermore, the work extends these continuous models to their discrete counterparts using finite difference methods, analyzing their qualitative properties and the stability of their numerical solutions. This dual approach, combining continuous theoretical analysis with discrete numerical considerations, underscores the thesis's contribution to a comprehensive understanding of epidemic dynamics.

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