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Mathematical model of Coronavirus for an isolated class

Presented by
Nacib Zaineb *Mouici Hanene*

Exammination Committee:

Mr. Salem AbdlMalek	*Univérsité Larbi Tébessa*	*Président*
Mr. Bougherara Lyazid	*Univérsité Larbi Tébessa*	*Examiner*
Mr. BOUAZIZ Khelifa	*Univérsité Larbi Tébessa*	*Supervisor*

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Dedication

We devote this modest work to all, who from near and far have given us their moral

and physical support for the realization of this work.

To our parents for their support during all studies and who never cease to lavish us

with their love.

To every teacher sacrificed for us to reach this level.

To all our brothers and sisters who were exemplary in our lives.

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who are in our hearts, especially our dear parents, for their sacrifices, kindness and neverending support.

ملخص

الهدف من هذا العمل هو دراسة نظام المعادلات التفاضلية العادية من الدرجة الاولى التي تستخدم لتحليل ديناميكيات مرض Covid-19 عبر نموذج رياضي مقترح ،ويتم إجراء تحليل الاستقرار العالمي للنموذج الموسع بواسطة دالة ليابونوف المناسبة، حيث يتم إما بانتشار السكان المعرضون للإصابة او المصابون بالعدوى ويعتمد استقرار المرض على كل من معدل انتقال المرض ومعدل تطور الحالة المعدية الى حالة معزولة او في المستشفى، يمكن ان يلعب الرقم R_0 دورا في تحديد ما اذا كان المرض سينقرض او يستمر ،اذا كان $R_0<1$ فإن التوازن الخالي من الامراض يكون مستقرا بشكل مقارب وغير مستقر عالميا عندما $R_0>1$.

الكلمات المفتاحية :

الاستقرار المحلي ، الاستقرار العالمي ، نقاط التوازن ، دالة ليابونوف

Résumé

Le but de ce travail est d'étudier le système d'équations différentielles ordinaires du premier ordre utilisé pour analyser la dynamique de la maladie COVID-19 via un modèle mathématique proposé. L'analyse de stabilité globale est réalisée pour le modèle étendu par une fonction de Lyapunov appropriée, dans laquelle soit les populations sensibles ou infectieuses sont diffusives. La stabilité de la maladie dépend à la fois du taux de transmission de la maladie et du taux de progression de l'état infectieux vers l'état isolé ou hospitalisé. Le nombre R_0 peut jouer un rôle dans la détermination de l'extinction ou de la persistance de la maladie, si $R_0 < 1$, alors l'équilibre sans maladie est globalement asymptotiquement stable et instable lorsque $R_0 > 1$.

Mots clés : Stabilité locale, Stabilité globale, Points d'équilibre, Fonction de Lyapunov.

Abstract.

The aime of this work is to study system of rst order ordinary differential equations is used to analyse the dynamics of COVID-19 disease via a mathematical model proposed. The global stability analysis is conducted for the extended model by suitable Lyapunov function, in which either susceptible or infective populations are diffusive. The stability of the disease is dependent on both transmission rate of the disease and the progression rate of the infectious state to isolated or hospitalized state. The number R_0 can be played role in determining whether the disease will extinct or persist, if $R_0 < 1$ then the disease-free equilibrium is globally asymptotically stable and unstable when $R_0>1$.

Key words: Stability local, Stability global, Equilibriums points, Lyapunov function

CONTENTS

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Introduction

Mathematical models are useful to understand the behavior of an infection when it enters a community and investigate under which conditions it will be wiped out or continued. Currently, COVID-19 is of great concern to researches, governments, and all people because of the high rate of the infection spread and the significant number of deaths that occurred. In December 2019, coronavirus first reported inWuhan, China, is an infectious disease caused by a newly discovered coronavirus. The virus that causes COVID-19 is mainly transmitted through droplets generated when an infected person coughs, sneezes, or exhales. These droplets are too heavy to hang in the air and quickly fall on floors or surfaces. Coronavirus-confirmed cases reached nearly 174 million in the world, and approximately $3{,}74\times10^6$ people have lost their lives due to this virus [34].

In 2020 The Coronavirus pandemic has spread in Algeria starting from 25 February 2020, when a positive test for Coronavirus disease 2 associated with severe acute respiratory syndrome (SARS-2-CoV) for a sample from an Italian citizen, and then revealed other cases infected with Covid-19, the total of cofirmed cases in Algeria reached 131.283 Including 3.527 deaths, up to 08 june 2021, the Algiers wilaya ranked first with 6506 confirmed cases, followed by the wilaya of Blida with 4453 cases [35].

Some models have made modifications based on the conventional 'SEIR' model [28] and concluded that strictly controlled interventions are critically important to impede COVID-19 outbreak [8] [29]. Several other model instead established a stochastic transition model to evaluate the transmission of COVID-19 and also emphasized the necessity of interventions such as social-distancing, isolation and quarantine [8]. Meanwhile, asymptomatic patients are covert cases which represent a serious threat to public health [32]. A few models have been developed to evaluate the role of coronavirus transmission based on asymptomatic cases [29] [30] [31].

This work is divided into three chapter:

Chapter1: Fundamental concepts and description of COVID-19 model.

In this chapter, we define and introduce the basic functional tools necessary to Reaction-diffusion.

Chapter2: Stability of COVID-19 model.

As we have seen before the Covid-19 model, in this chapter we will study the local and global stability of the equilibriums points of this model in the case of ordinary differential equations.

Chapter3: Reaction-Diffusion system of COVID-19 model.

In this chapter we will study change as Covid-19 spreads to the population, we will examine the local stability and global stability of the system (PDE) of the equilibrium points of the Covid-19 model.

List of tables

List of figures

List of abbreviations and symbols

- The set of the real numbers is denoted by $\mathbb{R}$.
- The set of the real numbers of the n-elements, is denoted by $\mathbb{R}^n$.
- The determinant of real and complex martices is denoted by $\det(A)$.
- The trace of real and complex matrices is denoted by $tr(A)$.
- The invers of real and complex matrices is denoted by A^{-1}.
- The transpose of matrix A is denoted by A^T.
- The diagonal of real and complex matrices is denoted by $diag(A)$.
- The real part of a complex number is denoted by $\mathrm{Re}(A)$.
- The Sobolev spaces, is denoted by $H^1(\Omega)$.
- The spectral radius of A, is denoted by $\rho(A)$.
- R_0 Basic Reproduction Number.
- The space of continuous and derivative functions is denoted by C^1.
- $\dfrac{\partial A_i}{\partial x} = \dfrac{\partial}{\partial x}(A_1, A_2, ...A_3)$ denotes the partial derivative of $(A_1, A_2, ...A_3)$.
- $\Delta A = \sum_{i=1}^{n} \frac{\partial^2 A}{\partial x_i^2}$ denoted the Laplacian operator A,
- $\nabla A = \left(\frac{\partial A}{\partial x_1}, \frac{\partial A}{\partial x_2}, ..., \frac{\partial A}{\partial x_n}\right)$ denoted the gradient of A
- The disease-free equilibrium is denoted by (**DFE)**.
- The ordinary differential equations denoted by (**EDO**).
- The partial differential equations denoted by (**EDP**).

CHAPTER 1

Fundamental concepts and description of COVID-19 model

In this chapter, we define and introduce the basic functional tools necessary to Reaction-diffusion and description of COVID-19 model

Definition 1 *Epidemiology is the subject that studies the patterns of health and illness and associated factors at the population level. The word "epidemiology" is derived from the Greek terms epi, which means "upon," demos, which means " people," and logos, which means "study." This etymology implies that the subject of epidemiology applies only to human populations. The role of father of epidemiology is often assigned to the Greek physician Hippocrates (460–377 B.C.E.), who described the connection between disease and environment. The term "epidemiology" appears to have first been used to describe the study of epidemics in 1802 by the Spanish physician de Villalba in Epidemiologia Espanola. Until the twentieth century, epidemiological studies were mostly concerned with infectious diseases. Nowadays, the leading causes of deaths worldwide are diseases such as stroke and coronary heart disease, positioning diseases that do not transmit from one person to another as a central concern of epidemiology. Among infectious diseases, those that dominate worldwide as a cause of death include lower respiratory infections (such as pneumonia).*

1.1 Reaction-diffusion systems

Diffusion - reaction systems are well established in different life science disciplines .When applied to 'human question' they are used to estimate the demographic processes involved in major human (or animal) dispersal episodes and to estimate the general spread pattern of new ideas or technologies through cultures.This manuscript gives an introduction to diffusion-reaction systems for a non-mathematical audience.We focus on describing dispersal processes and start with modelling and analysing the spread dynamic of a single population under different dispersal and growth hypotheses. Further we focus on the impacts of population interactions on spread behaviour of a particular population.

Definition 2 (**Diffusion**) *:The concept of diffusion originale from physical sciences (Fick's law is regarded as the fundamental principle of diffusion). In its physical sense diffusion is defined as a phenomenon where a certain particle group as a whole spreads according to the irregular motion of each particle. There by the spread is always directed from regions of higher concentration to regions of lower concentration and the time dependence of the distri-bution of the particles in space is given by the so called diffusion equation which is the mathematical formulation of the described spread dynamic. The diffusion theory seeks to explain the spread behaviour of a group of particles (rather than spread behaviour of asingle particle) and consequently the variable of interest is the proportion of the particle group which can be found. In this way phenomena like the diffusion of an ink drop in water or diffusion of heat can be described, of diffusion is applied in biology to describe processes of biodiffusion and to model population dynamics or the spread of infectious diseases among the population, or in a less quantitative way, in social sciences to describe the spread of ideas (diffusion of innovations, lexical diffusion, trans-cultural diffusion).*[33]

Definition 3 (**Reaction**) *:After modelling dispersal phenomena we turn our attention to growth processes. Here we understand growth as the increase or decrease of the variable of interest (e.g. population size number of individuals who use a new technology) due to intrinsic birth-death pro-cesses. Similar to diffusion dynamics, where Fick's law provided the fundamental principle growth behaviour can be characterised by a very small number of basic concepts such as the Malthusian or the logistic growth model. The Malthusian law of growth proposes that the human population of a nation grows exponentially (at least for a while). Contrarily the logistic law postulates that the rate of growth is proportional to both the present population size and the amount of available resources and is therefore bounded if the amount of resources are limited.*[33]

1.2 Interaction between dispersing populations

After considering different approaches for describing reproduction and dispersion, whether for infection, prey or population separately we now study the population dynamic in[19] obtained by combining both mechanisms. We allow the population or infection to grow and to disperse at the same time and are interested in the temporal and spatial behaviour of the population size under different growth models (exponential growth, logistic growth,...). We analyse so called diffusion-reaction systems of the form

$$\frac{\partial A\left(x,t\right)}{\partial t}=D\Delta A\left(x,t\right)+F(A), \tag{1.1}$$

where A is an m-vector the time- and space dependent function A again describes the population size at any location x and time t. The mathematical symbol Δ defines the Laplacian operator, which is the mathematical description of the process of moving the Infection or population from local spatial regions of high density or the most common infection to those of a lower density or the least infection, D =daigonal matrix$(D_1, D_2, ..., D_m)$,the temporal change of the population or infection size at location x is given by the diffusion component $D\Delta A\left(x,t\right)$,and the growth component $F=(F_0, F_1, ..., F_m)$.

So far we focused on describing the dispersal pattern of a single population in an unoccupied habitat. But what happens if the dispersing population comes into contact with other populations? Depending on the kind of interaction one would expect significant changes in the dispersal patterns. [18]Very generally interactions can be divided into three groups:

• Prey-predator interactions:If the growth rate of one population is increased but decreased for the other then we are in a predator-prey situation.

• Competition:If the presence of the population carrying the infection reduces the number of healthy people, then we face competition to spread infection

• Mutualism:If each population's growth rate is enhanced then it's called mutualism.

Example 4 ***(Prey-Predator models).***

When predators are successful at catching prey[18] they will reproduce more reliably and their species will increase in numbers whereas the numbers of their prey will fall. However, the larger predators population will struggle to find enough food to support them and their numbers will fall because of the reduced population of prey species. Eventually the situation will reverse it self as the number of prey increase due to less predation. If the ecosystem is large enough and other factors do not have an excessive effect, this can result in a situation in which populations of predator and prey rise and fall at regular

intervals, with a small time lag between them. This dynamic is described mathematically by the following Lotka-Volterra system

$$\begin{cases} \dfrac{\partial N}{\partial t} = N(a - bP) \\ \\ \dfrac{\partial P}{\partial t} = P(cN - d) \end{cases} \tag{1.2}$$

The time-dependent variables N and$P$ stand for the population sizes of the prey and predator population, respectively, and the terms$\dfrac{\partial N}{\partial t}$ and $\dfrac{\partial P}{\partial t}$ define the temporal change in frequency of both populations, In the absence of any predators (that means $P = 0$) the prey grows exponentially, modelled by the term a$N(a > 0)$. So the coefficient a is the intrinsic growth rate of the prey population if no interactions with the predator population occur. The effect of predation is to reduce the growth aN by the term$-bPN(b > 0)$ which is proportional to both, the prey and the predator population. In the absence of any prey (that means $N = 0$) the predator population is reduced exponentially which is modelled by the term $-dP(d > 0)$. Lastly the predators growth is dependent on the availability of prey and therefore is modelled by the term $cPN(c > 0)$.

1.3 Stability of system Reaction-diffusion

The study of the endemic global stability is not only mathematically important, but also essential in predicting the evolution of the disease in the long run so that prevention and intervention strategies can be effectively designed, and public health administrative efforts can be properly scaled.and public health administrative efforts can be properly scaled. There are some methods, i.e. those based on the monotone dynamical systems(1.1), the geometric approach [22], and Lyapunov functions,[23] to conduct global stability analysis for epidemic models.

Under certain assumptions one might expect that the solution to (1.1) would approach as $t \longrightarrow \infty$ to a solution of the system of steady state equations

$$D\Delta A\,(x,t) + F(A) = 0. \tag{1.3}$$

In D with Neumann boundary data. Solutions of (1.3) with the Neumann boundary condition are called equilibrium solutions, we are interested in constant equilibrium solutions to ((1.3).

Definition 5 ***(equilibrium point)*** *we say that x an equilibrium point of a system*

$$\begin{cases} \frac{d}{dt}x(t) = f(x(t)), \\ x(0) = x_0. \end{cases} \tag{1.4}$$

if x^E verify the equationt

$$f(x^E) = 0. \tag{1.5}$$

for all $t \geq t_0$.

Lemma 6 *[14] Let* $A = \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix}$ *be a 2×2 matrix Then A is Volterra-Lyapunov stable if and only if $a_{11} < 0$, $a_{22} < 0$ and*

$$\det(A) = a_{11}a_{22} - a_{12}a_{21} > 0. \tag{1.6}$$

The characterization of Volterra-lyapunov stable matrices of higher dimensions, however, is much more difficult We need the following definition.

Lemma 7 *[15]If x_o is the disease free equilibrium (DFE) of model*

$$\frac{dx}{dt} = f_i(x) = F_i(x) - V_i(x) \qquad \forall i = \overline{1,n},$$

$F_{\mathbf{i}}(x)$ be the rate of appearance of new infections in compartment i,

$V_i^+(x)$ be the rate of transfer of individuals into compartment i by all other means

$V_i^-(x)$ be the rate of transfer of individuals out of compartment i.

It is assumed that each function is continuously differentiable at least twice in each variable.

and $f_i(x)$ satisfies (A1) through (A5), where

(A1) If $x > 0$, then $F_i, V_i^+, V_i^- \geq 0$ for $i = 1, ..., n$.

(A2) If $x_i = 0$ then $V_i^- = 0$. In particular, if $x \in X_s$ then $V_i^- = 0$ for $i = 1, ..., m$.

(A3) $F_i = 0$ if $i > m$.

(A4) If $x \geq X_s$ then $F_i(x) = 0$ and $V_i^+(x) = 0$ for $i = 1, ..., m$.

(A5) If $F(x)$ is set to zero, then all eigenvalues of $Df(x_{\mathbf{0}})$ have negative real parts.

then the derivatives $DF(x_o)$ and $DV(x_o)$ are partitioned as

$$DF(x_o) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \text{and } DV(x_o) = \begin{bmatrix} V & 0 \\ J_3 & J_4 \end{bmatrix}, \tag{1.7}$$

where F and V are the $m \times m$ matrices defined by

$$F = \left[\frac{\partial F_i}{\partial x}(x_o)\right], \text{and } V = \left[\frac{\partial V_i}{\partial x}(x_o)\right], \text{ with } 1 \leq i, j \leq m. \tag{1.8}$$

Further, F is non-negative, V is a nonsingular M-matrix and all eigenvalues of J_4 have positive real part.

Definition 8 *We denote by $L^2(\Omega)$ the set of integrable square. A functions*

f defined on Ω is called an integrable square if f is measurable.We then define the norme on

$$\| f \|_{L^2(\Omega)} = \left(\int | f |^2\right)^{\frac{1}{2}} = 0 \tag{1.9}$$

Definition 9 *[15]The equilibrium X^E is said to be stable if for everything $\varepsilon > 0$; it exists $\eta > 0$ as for all solution $X(t)$ of (1), we have*

$$\left\|X_0 - X^E\right\| < \eta \quad \Rightarrow \left\|X(t) - X^E\right\| < \varepsilon \tag{1.10}$$

Definition 10 *[15](Locally asymptotically stable) Let $J(X^E) = \frac{\partial f}{\partial X}(X^E)$ the Jacobian matrix of f evaluates at point X^E. Consider the following linear system*

$$\frac{dX}{dt} = AX. \tag{1.11}$$

where $A = J(X^E)$ is say the linearized or the linear approximation of the non-linear system (1.4) in X^E.

The study of the stability of the origin for the linearized allows in certain cases to characterize the stability of the (1.4) . More precisely we have.

• If all the eigenvalues of the matrix A are of strictly negative real part, then the system (1.4) is stable.

• If there is at least one eigenvalue of the matrix A of strictly positive real part then, the system (1.4)is unstable.

Definition 11 (*Globally asymptotically stable*) *The equilibrium point* X^E *is say to be globally asymptotically stable if it is stable, and for any* $X(t)$ *solution for* (1.4), *we have*

$$\lim_{t\to\infty} \left\| X(t) - X^E \right\| = 0 \tag{1.12}$$

Definition 12 *The basic reproduction number* R_0 *is the spectral radius of the next generation matrix, namely* $R_0 = \rho\left(FV^{-1}\right)$. *The following interpretation is given to the matrix* FV^{-1}.

• Let us consider an infected individual introduced into a compartment $FV^{-1}$of a population without disease. The entry $(i;k)$ of the matrix $V^{-1}$is the average time that the individual will spend in compartment i during his life assuming that the infection has been blocked.

• The entry $(j;i)$ of matrix F is the speed at which an infected person in compartment i produces infections in compartment j thus the entry $(j;k)$ of FV^{-1} is the expected number of new infections in compartment j produced by an infected individual originally introduced into compartment k .The spectral radius of the matrix FV^{-1} is the basic reproduction number That is to say $R_0 = \rho\left(FV^{-1}\right)$.

Lemma 13 *For all function* μ *of* $H_1(\Omega)$,*and all function* ν*of* $H_1(\Omega)$,*we have the Green formula*

$$\int_\Omega (\Delta u)\, v = \int_\Omega \frac{\partial u}{\partial \eta} v d\sigma - \int_\Omega \nabla u \nabla v \tag{1.13}$$

Proof 14 *On suppose* $\Delta u = \sum_{i=1}^n \frac{\partial^2 u}{\partial x_i^2}$ *the Laplacian of a distribution* u. *Then, if* u *is a function of* $H_1(\Omega)$

$$-\int_\Omega (\Delta u)\, v = -\sum_{i=1}^n \frac{\partial^2 u}{\partial x_i^2} v dx.$$

$$= \sum_{i=1}^n \left\{ \int_\Omega \frac{\partial u}{\partial x_i}\frac{\partial v}{\partial x_i} - \int_\Omega \frac{\partial u}{\partial x_i} v \eta_i d\sigma \right\},$$

$$= \sum_{i=1}^n \int_\Omega \frac{\partial u}{\partial x_i}\frac{\partial v}{\partial x_i} - \int_\Omega \frac{\partial u}{\partial \eta} v d\sigma,$$

$$\int_\Omega \nabla u \nabla v - \int_{\partial\Omega} \frac{\partial u}{\partial \eta} v d\sigma \tag{1.14}$$

Definition 15 *The spectral radius of a matrix A is defined as the maximum of the absolute values of the eigenvalues of A:*

$$\rho(A) = sup\{|\lambda| : \lambda \in \sigma(A)\},$$

where $\sigma(A)$ denotes the set of eigenvalues of A.

Theorem 16 ***(Routh-Hurwitz)***

Consider the characteristic equation

$$\det(\lambda I_n - A) = \lambda^n + a_1\lambda^{n-1} + a_2\lambda^{n-2} + ... + a_{n-1}\lambda + a_n. \tag{1.15}$$

Determining the n eigenvalues λ of a real $n \times n$ square matrix A,where I is the identity matrix. Then the eigenvalues λ all have negative real parts if

$$\Delta_1 > 0, \Delta_2 > 0, ..., \Delta_{n-1} > 0, \Delta_n > 0, \tag{1.16}$$

where

$$\Delta_1 = a_1, \Delta_2 = \begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix} = a_1a_2 - a_3, \tag{1.17}$$

and

$$\Delta_k = \begin{vmatrix} a_1 & 1 & 0 & 0 & 0 & 0 & \cdots & 0 \\ a_3 & a_2 & a_1 & 1 & 0 & 0 & \cdots & 0 \\ a_5 & a_4 & a_3 & a_2 & a_1 & 1 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ a_{2k-1} & a_{2k-2} & a_{2k-3} & a_{2k-4} & a_{2k-5} & a_{2k-6} & \cdots & a_k \end{vmatrix}. \tag{1.18}$$

Table 1 Routh–Hurwitz criteria

n	Coefficient signs	Additional conditions
2	$a_1 > 0, a_2 > 0$	–
3	$a_1 > 0, a_2 > 0, a_3 > 0$	$a_1a_2 > a_3$
4	$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0$	$a_1a_2a_3 > a_3^2 + a_1^2a_4$
5	$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0,$ $a_5 > 0$	$a_1a_2a_3 > a_3^2 + a_1^2a_4,$ $(a_1a_4 - a_5)(a_1a_2a_3 - a_3^2 - a_1^2a_4) > a_5(a_1a_2 - a_3)^2 + a_1a_5^2$

1.3.1 Stability Based on Lyapunovs Method

The method of Lyapunov functions has been known for many decades The challenge in the application of this method is that there is no systematic way to construct Lyapunov functions (particularly, the determination of the appropriate coefficients is often a matter of luck), so that its success largely depends on trial and error as well as on specific problems.

In this study, we are aiming to establish the global stability of constant steady-state solutions to reaction–diffusion systems with Neumann boundary conditions. The approach here,is to construct Lyapunov function-alsforpartial differential equations(**PDE**) using Lyapunov function als for ordinary differential equations(**ODE**).

Definition 17 ***(function Lyapunov)***

A associated function $V(t)$ or system (1.1) is the Lyapunov if

$$\begin{aligned} &1\text{- } V(t) \geq 0 \text{ and} \\ &2\text{- } \frac{d}{dt}\left[V(t)\right] \leq 0 \quad \forall t \geq 0. \end{aligned} \tag{1.19}$$

Description of the method

Let A be the non-negative vector of concentrations $A_1, ..., A_m$, and let the reaction be governed by the ordinary differential equation[19]

$$\dot{A} = F(A), \quad \text{with } F : \mathbb{R}^m \longrightarrow \mathbb{R}^m \text{ is a } C^1 \text{ function.} \tag{1.20}$$

Let Ω be a bounded domain in $\mathbb{R}^n$ with smooth boundary $\partial\Omega$ and $D = (d_1, ..., d_m)$ with $d_i \geq 0$. Suppose $A^*$is a positive equilibrium of (1.20). Then u

is also a spatially homogeneous steady-state solution to the following reaction–diffusion system with Neumann boundary condition

$$\begin{aligned} &\frac{\partial A}{\partial t} = D\Delta A + F(A) \quad \text{in } \Omega \times (0, +\infty), \\ &\frac{\partial A}{\partial \eta} = 0 \text{ on } \partial\Omega \times (0, +\infty), \\ &A(x, 0) = A_0(x) \quad \text{in } \Omega. \end{aligned} \tag{1.21}$$

where Δ is the Laplacian in the variable $x \in \Omega \subset \mathbb{R}^n$, and$\frac{\partial A}{\partial \eta}$ is the outward normal derivative on $\partial\Omega$. System (1.21) is the kinetic system of (1.20).

Let$V(A)$ be a C^1 function defined on some domain in $\mathbb{R}^m_+$. When $A(t)$ is a solution of (1.20), it is often necessary to compute the time derivative of $V(A(t))$. It holds that

$$\frac{dV(A(t))}{dt} = \nabla V(A) \cdot f(A). \tag{1.22}$$

We assume that the range of $A(t)$ is contained in the domain of $V(A)$. The right hand side is given by the gradient of the function $V(A)$ and the vector field $F(A)$. Thus the right hand side is defined without the fact that $A(t)$ is a solution of (1.20), and it is important for our calculation of Lyapunov functionals.

Let $A(t,x)$ be a solution of (1.21) [19], and we put

$$W = \int_\Omega V(A(t,x))dx. \tag{1.23}$$

Calculating the time derivative of W along the positive solution of model (1.21), we get

$$\begin{array}{l} \frac{dW}{dt} = \int_\Omega \nabla V(A) \cdot (D\Delta A + f(A))\, dx, \\ = \int_\Omega \nabla V(A) f(A) dx + \int_\Omega \nabla V(A) D\Delta A dx. \end{array} \tag{1.24}$$

Hence,

$$\frac{dW}{dt} = \int_\Omega \nabla V(A) f(A) dx + \sum_{i=1}^{m} d_i \int_\Omega \frac{\partial V}{\partial A_i} \Delta A_i dx. \tag{1.25}$$

We assume the integrand of the first term of (1.25) is already calculated as (1.22) for the ordinary differential equation (1.20). The second term is simplified by using Green's formula, and we obtain

$$\int_\Omega \frac{\partial V}{\partial A_i} \Delta A_i = \int_{\partial\Omega} \frac{\partial V}{\partial A_i} \frac{\partial A_i}{\partial \eta} d\eta - \int_\Omega \nabla A_i \nabla \left(\frac{\partial V}{\partial A_i} \right) dx. \tag{1.26}$$

since $\dfrac{\partial A_i}{\partial \eta} = 0$ on $\partial\Omega$, then

$$\int_\Omega \frac{\partial V}{\partial A_i} \Delta A_i dx = - \int_\Omega \int_\Omega \nabla A_i \nabla \left(\frac{\partial V}{\partial A_i} \right) dx. \tag{1.27}$$

Hence

$$\frac{dW}{dt} = \int_\Omega \nabla V(A) \cdot f(A) dx - \sum_{i=1}^{m} d_i \int_\Omega \nabla A_i \nabla (\frac{\partial V}{\partial A_i}) dx. \tag{1.28}$$

So we construct the function V such that

$$d_i \int_\Omega \nabla A_i \nabla(\frac{\partial V}{\partial A_i})dx \geq 0, \quad \text{for all } i = 1...m. \tag{1.29}$$

In literature, many authors constructed explicit Lyapunov functions of the form

$$V = \sum_{i=1}^{m} a_i \left(A_i - A_i^* \ln A_i\right). \tag{1.30}$$

In this case, we have

$$\int_\Omega \nabla A_i \nabla(\frac{\partial V}{\partial A_i})dx = a_i A_i^* \int_\Omega \frac{|\nabla A_i|^2}{A_i^2}dx \geq 0.$$

We summarize the above in the following proposition.

Proposition 18 *[19]1-If the Lyapunov function for the ordinary differential equation (1.20) verifies (1.29), then the function W defined by (1.23) is a Lyapunov functional for the Reaction–diffusion system*

2-If the Lyapunov function for the ordinary differential equation (1.20) is of the form described by (1.30), then W is a Lyapunov functional for the Reaction–diffusion system (1.21).

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

1.4 Model formulation

In the model, total population $N(t)$ is divided into four classes: Susceptible:$S(t)$, Infected $I(t)$, Hospitalized: $H(t)$ and Recovered:$R(t)$ So $N(t) = S(t)+I(t)+H(t)+R(t)$. We have not considered human mobility and it is assumed that the recovery from the disease gives total immunity. Also, for all compartments per day infection rate, recovery time are fixed and natural death rate are considered same for all the compartments. All the hospitalized individuals are isolated and do not come in contact with susceptible individual.

The mathematical model of the transmission of COVID-19 is described as follows:

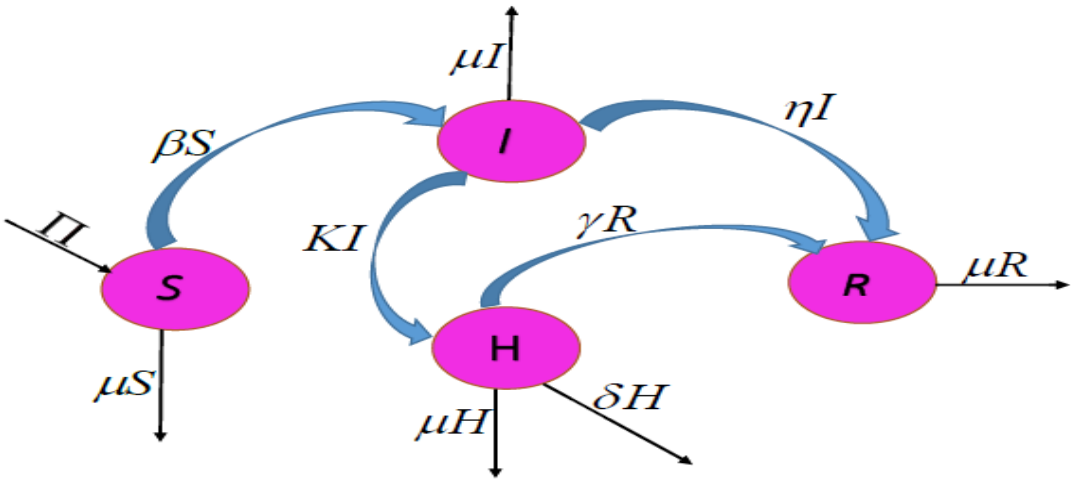


Diagram1-Flow diagram for COVID-19 disease transmission model

$$\begin{cases} \frac{dS(t)}{dt} = \pi - \beta SI - \mu S \\ \frac{dI(t)}{dt} = \beta SI - (k + \eta + \mu) I \\ \frac{dH(t)}{dt} = kI - (\delta + \gamma + \mu) H \\ \frac{dR(t)}{dt} = \eta I + \gamma H - \mu R. \end{cases} . \tag{1.31}$$

The coefficients used into our model are explained in the following :

N : total human population size

S : susceptible population size

I : infected (infectious) population size

H : hospitalized(isolated) population size

R : immunes (recovered) population size

π : recruitment rate

μ : death rate

β: transmission coefficient

η : recovery rate in infected population

γ : recovery rate in isolated population

δ : disease induced death rate

K : progression rate from infected to isolated population

1.4.1 Existence and boundedness

Suppose, $M = \{(S, I, H, R) \in \mathbb{R}^4 : S, I, H, R \geq 0\}$. Then M should be positively invariant. The solution of the system, the state variables S, I, H, R cannot exit M by crossing the boundaries

$$S = 0, I = 0, H = 0, R = 0.$$

From the system (1.31), we have

$$N = S + I + H + R.$$

Thus

$$\begin{gathered} \frac{dN}{dt} = \pi - \mu N - \delta H \leq \pi - \mu N \\ N \leq \frac{\pi}{\mu}\left(1 - \exp\left(-\mu t\right)\right) - N\left(0\right)\exp\left(-\mu t\right). \end{gathered}$$

Hence

$$\limsup N \leq \frac{\pi}{\mu}. \tag{1.32}$$

There for, $S(t), I(t), H(t)$ and $R(t)$ are bounded above by $\frac{\pi}{\mu}$on $[0; d)$ for some $d > 0$. Hence for some $d > 0$ the solution of the system (1.31) are bounded on $[0; d)$.

Assume that, the initial conditions of the system are as follows

$$S(0) > 0, I(0) > 0, H(0) > 0, R(0) > 0. \tag{1.33}$$

Let

$$x(t) = (S(t) + I(t) + H(t) + R(t)) \in \mathbb{R}^4. \tag{1.34}$$

The system (1.31)is written in the form $x = w(x)$ with w_i , $i = 1, 2, 3, 4$ are the components of the vector eld w, which consists of the algebraic polynomials of state variables. Thus wi are continuous autonomous functions on $\mathbb{R}^4$and partial derivatives

$$\frac{\partial w_i}{\partial S}, \frac{\partial w_i}{\partial I}, \frac{\partial w_i}{\partial H}, \frac{\partial w_i}{\partial R} \tag{1.35}$$

exist and are continuous. Hence by Existence and Uniqueness Theorem, for any initial condition $x(0) > 0 \in \mathbb{R}^4$ a unique solution of the system $x = w(x)$ exists

1.4.2 Equilibrium Points and Basic Reroduction Number

Compartmental models are deterministic, that is given the same inputs, they produce the same results every time.They are able to predict the various properties of the spread of the virus can estimate the duration of epidemics and can be used to understand how different situations or interventions can affect the results of the spread. To do this, the R_0 parameter describing the average number of new infections due to a sick individual plays a crucial role. As you can imagine if this number is less than (1.31) then the epidemic will tend to die out. In this case, the disease-free equilibrium (DFE) will be locally asymptotically stable and the disease cannot persist in the population. While it may persist or even spread to the whole population if.This implies that the disease-free equilibrium (DFE) is unstable. Using next generation matrix he baic reproduction of (1.31) is found here. Since the DFE is $E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$ and hence the basic reproduction number can be found using theanalytical approach.

Let

$$\begin{cases} \pi - \beta SI - \mu S = F_1 \\ \beta SI - (k + \eta + \mu) I = F_2 \\ kI - (\delta + \gamma + \mu) I = F_3 \\ \eta I + \gamma H - \mu R = F_4 \end{cases}. \tag{1.36}$$

We extract the Jacobian Matrix where

$$Df = \begin{pmatrix} \frac{dF_1}{dS} & \frac{dF_1}{dI} & \frac{dF_1}{dH} & \frac{dF_1}{dR} \\ \frac{dF_2}{dS} & \frac{dF_2}{dI} & \frac{dF_2}{dH} & \frac{dF_2}{dR} \\ \frac{dF_3}{dS} & \frac{dF_3}{dI} & \frac{dF_3}{dH} & \frac{dF_3}{dH} \\ \frac{dF_4}{dS} & \frac{dF_4}{dI} & \frac{dF_4}{dH} & \frac{dF_4}{dR} \end{pmatrix}, \tag{1.37}$$

by compensation we find

$$Df = \begin{pmatrix} -\beta I - \mu & -\beta S & 0 & 0 \\ -\beta I & \beta SI - (k + \eta + \mu) & 0 & 0 \\ 0 & k & -(\delta + \gamma + \mu) & 0 \\ 0 & \eta & \gamma & -\mu \end{pmatrix}, \tag{1.38}$$

Evaluating the Jacobian matrix (1.38) at E_0 yields

$$Df\left(E_0\right)=\begin{pmatrix} -\mu & -\frac{\beta\pi}{\mu} & 0 & 0 \\ 0 & \frac{\beta\pi}{\mu}-(k+\eta+\mu) & 0 & 0 \\ 0 & k & -(\delta+\gamma+\mu) & 0 \\ 0 & \eta & \gamma & -\mu \end{pmatrix},$$

where

$$W=\begin{pmatrix} \beta S-(k+\eta+\mu) & 0 & 0 \\ k & -(\delta+\gamma+\mu) & 0 \\ \eta & \gamma & -\mu \end{pmatrix}, \tag{1.39}$$

and

$$\begin{aligned} W &= F-V=\begin{pmatrix} \beta S & 0 & 0 \\ k & 0 & 0 \\ \eta & 0 & 0 \end{pmatrix} \\ &-\begin{pmatrix} (k+\eta+\mu) & 0 & 0 \\ 0 & (\delta+\mu+\pi) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}\begin{pmatrix} (k+\eta+\mu) & 0 & 0 \\ 0 & (\delta+\gamma+\mu) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}, \end{aligned} \tag{1.40}$$

where F and V are the 3×3 matrices defined by

$$F\left(E_0\right)=\begin{pmatrix} \frac{\beta\pi}{\mu} & 0 & 0 \\ k & 0 & 0 \\ \eta & 0 & 0 \end{pmatrix},$$

and

$$V\left(E_0\right)=\begin{pmatrix} (k+\eta+\mu) & 0 & 0 \\ 0 & (\delta+\gamma+\mu) & 0 \\ 0 & -\gamma & \mu \end{pmatrix}$$

The conditions listed above allow us to partition the matrix $Df(E_0)$ as shown by the following

$$R=\rho(FV^{-1})=\max(\left|\boldsymbol{\lambda}_1\right|,\left|\boldsymbol{\lambda}_2\right|,\left|\boldsymbol{\lambda}_3\right|) \tag{1.41}$$

$$V^{-1} = \frac{1}{\det(V)} \left(\tilde{V}\right)^t, \tag{1.42}$$

we count $\left(\tilde{V}\right)^t$:

$$\left(\tilde{V}\right)^t = \begin{pmatrix} (\delta+\gamma+\mu)(\mu) & 0 & 0 \\ 0 & (k+\eta+\mu)(\mu) & 0 \\ 0 & (k+\eta+\mu)\,(\boldsymbol{\gamma}) & (k+\eta+\mu)\,(\boldsymbol{\delta}+\boldsymbol{\gamma}+\boldsymbol{\mu}) \end{pmatrix}, \tag{1.43}$$

which has determinant

$$\det(V) = (k+\eta+\mu)\,(\delta+\gamma+\mu)\,(\mu). \tag{1.44}$$

Using (1.43) and (1.44), this can be rewritten as

$$V^{-1} = \frac{1}{(k+\eta+\mu)(\delta+\gamma+\mu)\mu} \begin{pmatrix} (\delta+\gamma+\mu)(\mu) & 0 & 0 \\ 0 & (k+\eta+\mu)\mu & 0 \\ 0 & (k+\eta+\mu)\boldsymbol{\gamma} & (k+\eta+\mu)(\delta+\gamma+\mu) \end{pmatrix}, \tag{1.45}$$

then

$$\begin{aligned} FV^{-1} &= \frac{1}{(k+\eta+\mu)\,(\delta+\gamma+\mu)\,(\mu)} \times \\ &\quad \begin{pmatrix} \frac{\beta\pi}{\mu} & 0 & 0 \\ k & 0 & 0 \\ \eta & 0 & 0 \end{pmatrix} \begin{pmatrix} (\delta+\gamma+\mu)(\mu) & 0 & 0 \\ 0 & (k+\eta+\mu)(\mu) & 0 \\ 0 & (k+\eta+\mu)\,(\gamma) & (k+\eta+\mu)\,(\delta+\gamma+\mu) \end{pmatrix} \\ &= \begin{pmatrix} \frac{\beta\pi(\delta+\gamma+\mu)(\mu)}{\mu(k+\eta+\mu)(\delta+\gamma+\mu)(\mu)} & 0 & 0 \\ \frac{k(\delta+\gamma+\mu)(\mu)}{(k+\eta+\mu)(\delta+\gamma+\mu)(\mu)} & 0 & 0 \\ \frac{\eta(\delta+\gamma+\mu)(\mu)}{(k+\eta+\mu)(\delta+\gamma+\mu)(\mu)} & 0 & 0 \end{pmatrix} \\ &= \begin{pmatrix} \frac{\beta\pi}{\mu(k+\eta+\mu)} & 0 & 0 \\ \frac{k}{(k+\eta+\mu)} & 0 & 0 \\ \frac{\eta}{(k+\eta+\mu)} & 0 & 0 \end{pmatrix} \end{aligned} \tag{1.46}$$

Using (1.46), we obtain

$$det(FV^{-1}-\lambda I_3)=\begin{pmatrix} \frac{\beta\pi}{\mu(k+\eta+\mu)}-\lambda & 0 & 0 \\ \frac{k}{(k+\eta+\mu)} & -\lambda & 0 \\ \frac{\eta}{(k+\eta+\mu)} & 0 & -\lambda \end{pmatrix}$$

$\boldsymbol{\lambda}_{i,i=\overline{1,3}}$ are the eignvalue

$$\begin{cases} \lambda_1=\frac{\beta\pi}{\mu(k+\eta+\mu)} \\ \lambda_2=0 \\ \lambda_3=0 \end{cases} \tag{1.47}$$

$$R_0=\rho(FV^{-1})=\max(|\lambda_1|,|\lambda_2|,|\lambda_3|)=|\lambda_1|$$

$$R_0=\frac{\beta\pi}{\mu(k+\eta+\mu)}.$$

The endemic equilibrium point $E_*=(S^*,I^*,H^*,R^*)$ the system (1.31) as follows:

$$\begin{cases} \pi-\beta SI-\mu S=0 \\ \beta SI-(k+\eta+\mu)I=0 \\ kI_1-(\delta+\gamma+\mu)H=0 \\ \eta I+\gamma H-\mu R=0 \end{cases}, \tag{1.48}$$

we take $\frac{dI^*}{dt}=0$ so $\beta S^*I^*-(k+\eta+\mu)I^*=0$, thus

$$S^*=\frac{(k+\eta+\mu)}{\beta},$$

and $\frac{dS^*}{dt}=0$ so $\pi-\beta S^*I-\mu S^*=0$, we get

$$\pi-\beta\frac{(k+\eta+\mu)}{\beta}I^*-\mu\frac{(k+\eta+\mu)}{\beta}=0.$$

On the other hand

$$I^*=-\frac{\mu}{\beta}+\frac{\pi}{(k+\eta+\mu)},$$

thus

$$I^*=\frac{\mu}{\beta}(R_0-1).$$

However $\frac{dH^*}{dt}=0$ So

$$kI^*-(\delta+\gamma+\mu)C_H^*=0. \tag{1.49}$$

Substitute I^* in (1.49)

$$k\frac{\mu}{\beta}(R_0-1)=(\delta+\gamma+\mu)H^*,$$

thus

$$H^*=k\frac{\mu}{\beta(\delta+\gamma+\mu)}(R_0-1).$$

From the equation $\frac{dS^*}{dt}=0$ So

$$\eta I^*+\gamma H^*-\mu R^*=0. \tag{1.50}$$

Substitute H^* and I^* in (1.50)

$$\eta\frac{\mu}{\beta}(R_0-1)+k\frac{\mu}{\beta(\delta+\gamma+\mu)}(R_0-1)\gamma=\mu R^*.$$

And here is the result

$$R^*=\frac{\eta}{\beta}(R_0-1)+k\frac{\gamma}{\beta(\delta+\gamma+\mu)}(R_0-1),$$

thus

$$R^*=\frac{(R_0-1)}{\beta}\left(\eta+k\frac{\gamma}{(\delta+\gamma+\mu)}\right).$$

So the value of (S^*,I^*,H^*,R^*)

$$\begin{array}{c} S^*=\frac{(k+\eta+\mu)}{\beta} \\ I^*=\frac{\mu}{\beta}(R_0-1) \\ H^*=k\frac{\mu}{\beta(\delta+\gamma+\mu)}(R_0-1) \\ R^*=\frac{(R_0-1)}{\beta}\left(\eta+k\frac{\gamma}{(\delta+\gamma+\mu)}\right) \end{array}. \tag{1.51}$$

CHAPTER 2

Stability of COVID-19 model

As we have seen before the Covid-19 model, in this chapter we will study the local and global stability of the equilibriums points of this model in the case of ordinary differential equations.

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

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

Theorem 19 *Disease free equilibrium point of the system of equations (1.31) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$*

Proof 20 *About E_0, the Jacobian matrix for the system of equations (1.31) is in the block matrix*

$$M = \begin{pmatrix} U_1 & U_2 \\ 0 & F - V \end{pmatrix}. \tag{2.1}$$

If all the eigenvalues of M have negative real parts, then E_0 is asymptotically stable [15]. Since M is upper triangular matrix eigenvalues of M are those of U_1 and $F - V$

$$M = \begin{pmatrix} -\beta I - \mu & -\beta S & 0 & 0 \\ \beta I & \beta S - (k + \eta + \mu) & 0 & 0 \\ 0 & k & -(\delta + \gamma + \mu) & 0 \\ 0 & \eta & \gamma & -\mu \end{pmatrix} \tag{2.2}$$

Using the same method from [1], the stability of E_0 reduces to examining the eigenvalues of the matrices

$$M(E_0) = \begin{pmatrix} -\mu & -\frac{\beta\pi}{\mu} & 0 & 0 \\ 0 & \frac{\beta\pi}{\mu} - (k+\eta+\mu) & 0 & 0 \\ 0 & k & -(\delta+\mu+\pi) & 0 \\ 0 & \eta & \delta & -\mu \end{pmatrix}. \tag{2.3}$$

Two eigenvalues of matrix U_1 are $-\mu, -\mu < 0$. Now, stability of the disease free equilibrium depends on the eigenvalues of $F - V$ where F is non-negative and V is non-singular M-matrix [3]. The eigenvalues of $F-V$ are $-(\delta+\gamma+\mu)$ and $(R_0-1)(k+\eta+\mu)$ Thus the eigenvalues of are $F - V$ negative if $R_0 < 1$ and positive if $R_0 > 1$. It shows that E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

2.2 Local stability of endemic equilibrium

Theorem 21 *The endemic equilibrium point $E_1 = (S^*, I^*, H^*, R^*)$ is stable if $R_0 > 1$.*

Proof 22 *Evaluating the Jacobian matrix (2.2) at $E_1 = (S^*, I^*, H^*, R^*)$ yields*

$$M(E_1) = \begin{pmatrix} \frac{-\beta\mu(R_0-1)}{\beta} - \mu & \frac{-\beta(k+\eta+\mu)}{\beta} & 0 & 0 \\ \mu(R_0-1) & 0 & 0 & 0 \\ 0 & k & -(\delta+\gamma+\mu) & 0 \\ 0 & \eta & \gamma & -\mu \end{pmatrix}, \tag{2.4}$$

which has trace

$$\begin{aligned} tr(M(E_1)) &= -\mu R_0 - (\delta+\gamma+\mu) - \mu) \\ &= -(\mu R_0 + (\delta+\gamma+\mu) + \mu) < 0. \end{aligned} \tag{2.5}$$

The determinant of the Jacobian may be given by

$$\begin{aligned}\det(M(E_1)) = -\mu R_0 &\begin{vmatrix} 0 & 0 & 0 \\ k & -(\delta+\gamma+\mu) & 0 \\ \eta & \gamma & -\mu \end{vmatrix} \\ +(k+\eta+\mu) &\begin{vmatrix} \mu(R_0-1) & 0 & 0 \\ 0 & -(\delta+\gamma+\mu) & 0 \\ 0 & \gamma & -\mu \end{vmatrix},\end{aligned} \tag{2.6}$$

then

$$\begin{aligned}\det(M(E_1)) &= (k+\eta+\mu)\mu(R_0-1)(\delta+\gamma+\mu)\mu \\ &= (k+\eta+\mu)\mu^2(R_0-1)(\delta+\gamma+\mu),\end{aligned} \tag{2.7}$$

we obtain $\det(M(E_1)) > 0$. *Hence, the equilibrium point* E_1 *is locally asymptotically stable.*

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

In this section we will study the global stability of the equilibriums points of the model (1.31).

Theorem 23 (*Global Stability of DFE*) *Assume* $R_0 > 1$. *Then the disease free equilibrium is globally asymptotically stable.*

Proof 24 *We have* $R = N - S - I - H$, *the system* (1.31) *as follows;*

$$\begin{cases} \frac{dS}{dt} = \pi - \beta SI - \mu S \\ \frac{dI}{dt} = \beta SI - pI \\ \frac{dH}{dt} = KI - qH, \end{cases} \tag{2.8}$$

where $P = (k+\eta+\mu)$ *and* $q = (\delta+\gamma+\mu)$.

We know that if disease free equilibrium $E_0 = (\frac{\pi}{\mu}, 0, 0)$ *is globally stable, then* $R(t) \to 0$ *and* E_0 *for the system* (1.31) *is globally stable.*

Consider a Lyapunov function

$$V = \alpha(S - S^* - S^* \ln \frac{S}{S^*}) + \frac{1}{P} I + \frac{1}{K} H,$$

where $\alpha > 0$ *to be determined and ,* $S^* = \frac{\pi}{\mu}$ *at* $E_1, V = 0$.

At first we have to show that $V > 0$ *for all* $(S, I, H) \neq \left(\frac{\pi}{\mu}, 0, 0\right)$ *since* $\frac{1}{p} I > 0$ *and* $\frac{1}{k} H > 0$,*it is sufficient to show that* $\alpha S^* \left(\frac{S}{S^*} - 1 - \ln \frac{S}{S^*}\right) > 0$.

Now differentiating V *with respect to* t, *we get*

$$\begin{aligned}
\frac{dV}{dt} &= \alpha \left[\left(1 - S^* \left(\frac{\frac{1}{S^*}}{\frac{S}{S^*}} \right) \right) \right] \frac{dS}{dt} + \frac{1}{p} \frac{dI}{dt} + \frac{1}{k} \frac{dH}{dt} \\
&= \alpha (1 - \frac{S^*}{S}) \frac{dS}{dt} + \frac{1}{p} \frac{dI}{dt} + \frac{1}{k} \frac{dH}{dt} \\
&= (\alpha - \alpha \frac{S^*}{S})(\pi - \beta SI - \mu S) + \frac{1}{p}(\beta SI - pI) + \frac{1}{k}(kI - qH) \\
&= \alpha\pi - \alpha\beta SI - \alpha\mu S - \alpha\pi \frac{S^*}{S} + \frac{\alpha\beta S^* SI}{S} + \frac{\alpha\mu S^* S}{S} \\
&\quad + \frac{\beta}{p} SI - \frac{p}{p} I + \frac{K}{K} I - \frac{q}{K} H \\
&= \alpha\pi - \alpha\beta SI - \alpha\mu S - \frac{\alpha\pi^2}{\mu C_S} + \frac{\alpha\beta\pi}{\mu} I + \frac{\alpha\mu\pi}{\mu} + \frac{\beta}{p} SI - \frac{q}{K} H \\
&= 2\alpha\pi - \alpha\beta SI - \alpha\mu S - \frac{\alpha\pi^2}{\mu S} + \frac{\alpha\beta\pi}{\mu} I + \frac{\alpha\mu\pi}{\mu} + \frac{\beta}{p} SI - \frac{q}{K} H. \qquad (2.9)
\end{aligned}$$

If we choose $\alpha = \frac{1}{p}$ *then*

$$\frac{dV}{dt} = -\alpha\pi \left[-2 + \frac{\mu}{\pi} + \frac{\pi}{\mu S} \right] + \frac{\alpha\beta\pi}{\mu} I - \frac{q}{K} H. \qquad (2.10)$$

Since from equilibrium equation, we have

$$H = \frac{K}{q} I.$$

Therefore

$$\begin{aligned}
\frac{dV}{dt} &= -\alpha\pi \left[\frac{\mu}{\pi} + \frac{\pi}{\mu S} - 2 \right] + \frac{\alpha\beta\pi}{\mu} I - \frac{q}{K} \frac{K}{q} I \\
&= -\alpha\pi \left[\frac{\mu S}{\pi} + \frac{\pi}{\mu S} - 2 \right] + \left[\frac{\beta\pi}{(k + \eta + \mu)} - 1 \right] I \\
&= -\alpha\pi \left[\frac{\mu S}{\pi} + \frac{\pi}{\mu S} - 2 \right] + [R_0 - 1] I.
\end{aligned}$$

The last term is negative, since $R_0 < 1$.

Let $a = \frac{\mu S}{\pi}$ *then we have the first term is*

$$a + \frac{1}{a} - 2 = \frac{(a-1)^2}{a} > 0. \text{ if } a \neq 1 \tag{2.11}$$

So $a + \frac{1}{a} - 2 > 0$ *for all* $a > 0$ *and* $a \neq 1$. *Hence, we have* $\frac{dV}{dt} < 0$. *Therefore, by Lyapunov's theorem, the disease free equilibrium point is globally asymptotically stable*

2.4 Gobal stability of endemic equilibrium

In this section we will study the global stability of the equilibrium point E_1 of the model (1.31).

Theorem 25 *Assume* $R_0 > 1$ *Then, the endemic equilibrium is globally asymptotically stable.*

Proof 26 *Consider the system of equation (2.8). Consider a Lyapunov function*

$$w = (S - S^* - S^* \ln \frac{S}{S^*}) + (I - I^* - I^* \ln \frac{I}{I^*}) + \frac{k+\eta+\mu}{k}(H - H^* - H^* \ln \frac{H}{H^*}). \tag{2.12}$$

Here,$w = 0$ *when* $(S, I, H) = (S^*, I^*, H^*)$, *otherwise* $w > 0$, w *is also radially unbounded. Now it is remained to show that* $\frac{dw}{dt} < 0$.

Differentiating w *with respect to* t *and using (1.31) we get*

$$\begin{aligned} \frac{dw}{dt} &= \left(1 - \frac{S}{S^*}\right)\frac{dS}{dt} + \left(1 - \frac{I}{I^*}\right)\frac{dI}{dt} + \left(1 - \frac{H}{H^*}\right)\frac{dH}{dt} \\ &= \left(1 - \frac{S}{S^*}\right)(\pi - \beta SI - \mu S) + \left(1 - \frac{I^*}{I}\right)(\beta SI - pI) \\ &\quad + \frac{p}{K}\left(1 - \frac{H}{H^*}\right)(KI - qH) \end{aligned} \tag{2.13}$$

From equilibrium equation $\pi = S^*I^* + \mu S^*$, we get

$$\begin{aligned}
\frac{dw}{dt} &= \beta S^*I^* + \mu S^* - \beta SI - \mu S - \frac{BS^{*^2}I^*}{I} - \frac{\mu I^{*2}}{S} + \beta S^*I \\
&\quad +\mu S + \beta S^*I + \mu S + \beta SI - PI - \beta I^*S + PI^* + PI \\
&\quad -\frac{pq}{k}H - \frac{pH^*I}{H} - \frac{pq}{k}\frac{H^*H}{H} \\
&= -\mu\frac{S^{*2}}{S} + \mu S^* + \mu S + \beta S^*I^* - \frac{\beta S^{*2}I^*}{S} \\
&\quad +\beta S^*I + P^* - \frac{pq}{k}H - \frac{pH^*I}{H} - \frac{pq}{k}H^* - \beta I^*S \\
&= \frac{-\mu\left(S^* - S\right)^2}{S} + \beta S^*I^* - \beta\frac{S^{*2}}{I}I^* + \beta S^*I - \beta I^*S + PI^* \\
&\quad -\frac{1}{K}pqH - pI^*\left(\frac{IH^*}{I^*H}\right) + \frac{1}{K}pqH^*
\end{aligned}$$

Again, we have from equilibrium equation $KI^* = qH^*$ and $\beta S^*I^* = pI^*$

$$\frac{1}{k}pqH^* = pI^*. \tag{2.14}$$

Substituting this into the first equation yields

$$\begin{aligned}
\frac{dw}{dt} &= -\mu\left(S^* - S\right)^2 + \beta S^*I^* - \frac{BS^{*2}I^*}{S} + \beta S^*I - \beta S^*I^* \\
&\quad -\beta S^*I - \beta S^*I^*\left(\frac{IH^*}{I^*H}\right) + \beta S^*I^* \\
&= -\mu\left(S^* - S\right)^2 + \beta S^*I^*\left(3 - \frac{S^*}{S} - \frac{S}{S^*} - \frac{IH^*}{I^*H}\right).
\end{aligned} \tag{2.15}$$

Clearly the first term of right hand side of (2.15) is negative unless $S = S^*$. In the second term we have to show that $\left(3 - \frac{S^*}{S} - \frac{S}{S^*} - \frac{IH^*}{I^*H}\right)$ is non-positive for this suppose

$$a_1 = \frac{S^*}{S}\ ,\ a_2 = \frac{S}{S^*}\ , a_3 = \frac{IH^*}{I^*H}. \tag{2.16}$$

The Geometric Mean of the sequence is

$$\sqrt[3]{a_1a_2a_3} = \sqrt[3]{\frac{IH^*}{I^*H}} > 0.\ \textit{if}\ R_0 > 1$$

The Arithmetic Mean of the sequence is

$$\frac{a_1 + a_2 + a_3}{3} = \frac{\frac{S^*}{S} + \frac{S}{S^*} + \frac{IH^*}{I^*H}}{3}$$

since $AM \geq GM$. It is clear that

$$a_1 + a_2 + a_3 \geq 3\sqrt[3]{\frac{IH^*}{IH}} > 3 \text{ whenever } \sqrt[3]{\frac{IH^*}{IH}} > 1.$$

Thus, the second term is also non-positive, whenever $\sqrt[3]{\frac{IH^*}{IH}} > 1$ *hence,*

$$\frac{dw}{dt} \leq 0. \tag{2.17}$$

Now we apply the Krasovkii-LaSalle Theorem. Consider a set

$$U = \{x \in \mathbb{R}^n / w'(x) = 0\}$$

$\frac{dw}{dt} = 0$ *if and only if* $S = S^*$ *and*

$$\left(\frac{S^*}{S} + \frac{S}{S^*} + \frac{IH^*}{I^*H}\right) = 3, \tag{2.18}$$

since $S = S^*$ then $\frac{dS}{dt} = 0$ from the system (1.31) we get $I = I^*$. Finally, we get from (2.18), $H = H^*$ Therefore the set U consists only one element (S^*, I^*, H^*) Hence the theorem is proved.

CHAPTER 3

Reaction-Diffusion system of the model Covid-19

In the previous chapter, we studied the changes of infection Covid-19 in relation to time, and in this chapter we will study the change in time the place of the spread of Δ infection **Covid-19** spreads to the population, by increasing the mathematical factor in each equation of the system (**1.31**)**.** Where and we will use the same equilibriums point for the system **(EDO)**, we will study the local stability and the global stability of the system **(PDE)** for equilibriums points of the model **Covid-19.**

Let the systeme the reaction-diffusion of the model **Covid-19** is the form:

$$\begin{cases} \frac{dS}{dt} - a\Delta S = \pi - \beta SI - \mu S & \text{in } R_+ \times \Omega \\ \frac{dI}{dt} - b\Delta I = \beta SI - (K + \eta + \mu)I & \text{in } R_+ \times \Omega \\ \frac{dH}{dt} - c\Delta R = KI - (\delta + \gamma + \mu)H & \text{in } R_+ \times \Omega \\ \frac{dR}{dt} - d\Delta R = \eta I + \gamma H - \mu R. & \text{in } R_+ \times \Omega \end{cases} . \tag{3.1}$$

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

Subject to the homogeneous Neumann boundary condition

$$\frac{\partial S}{\partial t} = \frac{\partial I}{\partial t} = \frac{\partial H}{\partial t} = \frac{\partial R}{\partial t} = 0. \tag{3.2}$$

We denote the inde finite sequence of positive eigenvalues for the Laplacian operator Δ over Ω with Neumann boundary conditions by $0 = \lambda_0 \leq \lambda_1 \leq \lambda_2 \leq \lambda_3 \leq ... \rightarrow +\infty$.

Note that the first eigenfunction is a constant, which is why the corresponding eigenvalue is equal to zero. The corresponding sequence of eigenfunctions is denoted by $(\Phi_{ij})_{j=\overline{1,m_i}}$, where $m_i \geq 1$ is the algebraic multiplicity of λ_i. These functions are the solutions of,

$$\begin{cases} -(\Delta\Phi_{ij}) = \lambda_i \Phi_{ij} & \text{in}\Omega \\ \frac{\partial \Phi_{ij}}{\partial \nu} = 0. & \text{on} \ \ \partial\Omega \end{cases} . \tag{3.3}$$

The eigenfunctions are normalized according to,

$$\|\Phi_{ij}\|_2 = \int_\Omega \Phi_{ij}^2(x)dx = 1. \tag{3.4}$$

The set of eigenfunctions$\{\Phi_{ij} : i \geq 0, j = \overline{1, m_i}, \}$forms a complete orthonormal basis in $L_2(\Omega)$. In order to establish the local asymptotic stability of the steady states, we must examine all the eigenvalues of the linearizing operator and if they all have negative real parts, then the solution is locally asymptotically stable.

3.1 Local stability of the equilibruims pionts of the model covid-19

We will study the local stability of the pionts equilibriums of stability of the pionts equilibriums of PDE

$$J = \begin{pmatrix} -\beta I - (\mu + a\lambda_i) & -\beta S & 0 & 0 \\ \beta I & \beta S - (p + b\lambda_i) & 0 & 0 \\ 0 & k & -(q+c\lambda_i) & 0 \\ 0 & \eta & \gamma & -(\mu+d\lambda_i) \end{pmatrix}. \tag{3.5}$$

Let us examine the local stability of the disease-free equilibriumis E_0, Applying the next generation method Now, we compute The basic reproduction number of the model (1.31) , by defintion, we get:

$$R_i = \rho\left(FV_i^{-1}\right), \quad for \ \ i = 0, 1, ..., \tag{3.6}$$

we have

$$J(E_0) = \begin{pmatrix} -(\mu + a\lambda_i) & -\frac{\beta\pi}{\mu} & 0 & 0 \\ 0 & \frac{\beta\pi}{\mu} - (p + b\lambda_i) & 0 & 0 \\ 0 & k & -(q + c\lambda_i) & 0 \\ 0 & \eta & \gamma & -(\mu + d\lambda_i) \end{pmatrix}, \tag{3.7}$$

we have

$$W = \begin{pmatrix} \frac{\beta\pi}{\mu} - (p + b\lambda_i) & 0 & 0 \\ k & -(q + c\lambda_i) & 0 \\ \eta & \gamma & -(\mu + d\lambda_i) \end{pmatrix},$$

and

$$W = F - V = \begin{pmatrix} \frac{\beta\pi}{\mu} & 0 & 0 \\ k & 0 & 0 \\ \eta & \gamma & 0 \end{pmatrix} - \begin{pmatrix} p + b\lambda_i & 0 & 0 \\ 0 & q + c\lambda_i & 0 \\ 0 & 0 & \mu + d\lambda_i \end{pmatrix}.$$

Hence, the stability of E_0 rests on the negativity of the real parts of the eigenvalues of matrices.

The conditions listed above allow us to partition the matrix $J(\boldsymbol{E_0})$ as shown by the following:

$$R_i = \rho(FV^{-1}) = \max\left(|\lambda_1|, |\lambda_2|, |\lambda_3|\right) \tag{3.8}$$

where

$$\begin{aligned} V^{-1} &= \frac{1}{\det(V)} \left(\tilde{V}\right)^t \\ &= \frac{1}{\det(V)} \times \\ &\quad \begin{pmatrix} (p + c\lambda_i)(\mu + d\lambda_i) & 0 & 0 \\ 0 & (q + b\lambda_i)(\mu + d\lambda_i) & 0 \\ 0 & 0 & (q + b\lambda_i)(p + c\lambda_i) \end{pmatrix}, \end{aligned} \tag{3.9}$$

which implies that

$$FV^{-1} = \frac{1}{\det(V)} \begin{pmatrix} \frac{\beta\pi}{\mu} & 0 & 0 \\ k & 0 & 0 \\ \eta & \gamma & 0 \end{pmatrix} \times$$

$$\begin{pmatrix} (p+c\lambda_i)(\mu+d\lambda_i) & 0 & 0 \\ 0 & (p+b\lambda_i)(\mu+d\lambda_i) & 0 \\ 0 & 0 & (q+b\lambda_i)(p+c\lambda_i) \end{pmatrix},$$

$$= \frac{1}{\det(V)} \begin{pmatrix} \frac{\beta\pi(\delta+\gamma+\mu+c\lambda_i)(\mu+d\lambda_i)}{\mu} & 0 & 0 \\ k(p+c\lambda_i)(\mu+d\lambda_i) & 0 & 0 \\ \eta(p+c\lambda_i)(\mu+d\lambda_i) & \gamma(q+b\lambda_i)(\mu+d\lambda_i) & 0 \end{pmatrix}.$$

The determinant of the matrix V may be given by

$$\det(V) = (k+\eta+\mu+b\lambda_i)(\delta+\gamma+\mu+c\lambda_i)(\mu+d\lambda_i), \tag{3.10}$$

it follows that

$$FV^{-1} = \begin{pmatrix} \frac{\beta\pi}{(k+\eta+\mu+b\lambda_i)\mu} & 0 & 0 \\ \frac{k}{k+\eta+\mu+b\lambda_i} & 0 & 0 \\ \frac{\eta}{k+\eta+\mu+b\lambda_i} & \frac{\gamma}{\delta+\gamma+\mu+c\lambda_i} & 0 \end{pmatrix}, \tag{3.11}$$

where

$$\det(FV^{-1}-T_i) = \begin{vmatrix} \frac{\beta\pi}{(k+\eta+\mu+b\lambda_i)\mu} - T_1 & 0 & 0 \\ \frac{k}{k+\eta+\mu+b\lambda_i} & -T_2 & 0 \\ \frac{\eta}{k+\eta+\mu+b\lambda_i} & \frac{\gamma}{\delta+\gamma+\mu+c\lambda_i} & -T_3 \end{vmatrix} \tag{3.12}$$

and

$$\begin{cases} T_1 = \frac{\beta\pi}{(K+\eta+\mu+b\lambda_i)\mu}, \\ T_2 = 0, \\ T_3 = 0. \end{cases} \tag{3.13}$$

This implies that

$$R_i = \frac{\beta\pi}{(K+\eta+\mu+b\lambda_i)\mu}, \tag{3.14}$$

the stability of E_0 reduces to examining the eigenvalues of the matrices

(i) If $R_0 < 1$ and $R_i - 1 < 0$, the disease-free equilibrium $E_0 = (\frac{\pi}{\mu}, 0, 0, 0)$ is locally asymptotically stable.

In the presence of di usion, the equilibrium point $E_0 = (S^0, I^0, H^0, R^0) = (\frac{\pi}{\mu}, 0, 0, 0)$ satisfies

$$F-V=\begin{pmatrix} \frac{\beta\pi}{\mu}-(k+\eta+\mu+b\lambda_i) & 0 & 0 \\ k & -(\delta+\gamma+\mu+c\lambda_i) & 0 \\ \eta & \gamma & -(\mu+d\lambda_i) \end{pmatrix}$$

The eigenvalue of $(F-V)$

$$\det(F-V-T_i)=\begin{vmatrix} \frac{\beta\pi}{\mu}-(p+b\lambda_i)-T_1 & 0 & 0 \\ k & \frac{\beta\pi}{\mu}-(q+b\lambda_i)-T_2 & 0 \\ \eta & \gamma & -(\mu+d\lambda_i)-T_3 \end{vmatrix}, \tag{3.15}$$

which are given for all $i \geq 0$ by

$$\begin{cases} T_1=(R_i-1)(k+\eta+\mu+b\lambda_i) \\ T_2=-(\delta+\gamma+\mu+c\lambda_i) \\ T_3=-(\mu+d\lambda_i). \end{cases} \tag{3.16}$$

The eigenvalues of $F-V$ are $(R_i-1)(k+\eta+\mu+b\lambda_i)$ and $-(\delta+\gamma+\mu+c\lambda_i)$ and $-(\mu+d\lambda_i)$Thus, the eigenvalues of are $F-V$.

Since the Laplacian eigenvalues are positive and in ascending order, both T_1, T_2, T_3 and clearly have negativereal parts for $R_0 \leq 1$ and $R_i < 1$ leading to the local stability of E_0

(ii) If $R_0 > 1$, the positive constant endemic steady equilibrium $E_1 = (S^*, I^*, H^*, R^*)$ is locally asymptotically stable

The second steady state $E_1 = (S^*, I^*, H^*, R^*)$ satisfies

$$J=\begin{pmatrix} -\beta I^*-(\mu+a\lambda_i) & -\beta S^* & 0 & 0 \\ \beta I^* & \beta S^*-(p+b\lambda_i) & 0 & 0 \\ 0 & k & -(q+c\lambda_i) & 0 \\ 0 & \eta & \gamma & -(\mu+d\lambda_i) \end{pmatrix}. \tag{3.17}$$

The corresponding linearization operator is:

$$J(E_1) = \begin{pmatrix} -\beta\frac{\mu}{\beta}(R_0-1) - (\mu + a\lambda_i) & -\beta\frac{(k+\eta+\mu)}{\beta} & 0 & 0 \\ \beta\frac{\mu}{\beta}(R_0-1) & \beta\frac{(k+\eta+\mu)}{\beta} - (p+b\lambda_i) & 0 & 0 \\ .0 & k & -(q+c\lambda_i) & 0 \\ 0 & \eta & \gamma & -(\mu+d\lambda_i) \end{pmatrix}. \tag{3.18}$$

Hence, the stability of E_1 rests on the negativity of the real parts of the eigenvalues of matrix:

$$J(E_1) = \begin{pmatrix} -\mu R_0 + a\lambda_i & -\frac{(k+\eta+\mu)}{\beta} & 0 & 0 \\ \mu(R_0-1) & -b\lambda_i & 0 & 0 \\ .0 & k & -(q+c\lambda_i) & 0 \\ 0 & \eta & \gamma & -(\mu+d\lambda_i) \end{pmatrix},$$

which is guaranteed if the trace and determinant of $J(E_1)$ satis es the conditions $tr(J(E_1)) < 0$ and $det(J(E_1)) > 0$, for all $i > 0$.

Since

$$\begin{aligned} tr(J(E_1)) &= -(\mu R_0 + a\lambda_i) - b\lambda_i - (\delta+\gamma+\mu+c\lambda_i) - (\mu+d\lambda_i) \\ &= -[(2+R_0)\mu + \delta + \gamma + a\lambda_i + b\lambda_i + c\lambda_i + d\lambda_i] < 0. \end{aligned} \tag{3.19}$$

The determinant is given by

$$\begin{aligned} \det(J(E_1)) = &-(\mu R_0 + a\lambda_i) \begin{vmatrix} -b\lambda_i & 0 & 0 \\ k & -(\delta+\gamma+\mu+c\lambda_i) & 0 \\ \eta & \gamma & -(\mu+d\lambda_i) \end{vmatrix} \\ &+(k+\eta+\mu) \begin{vmatrix} \mu(R_0-1) & 0 & 0 \\ 0 & -(\delta+\gamma+\mu+c\lambda_i) & 0 \\ 0 & \gamma & -(\mu+d\lambda_i) \end{vmatrix} \\ &= (\mu R_0 + a\lambda_i) b\lambda_i (\delta+\gamma+\mu+c\lambda_i)(\mu+d\lambda_i) \\ &+(k+\eta+\mu)(\mu(R_0-1))(\delta+\gamma+\mu+c\lambda_i)(\mu+d\lambda_i), \end{aligned} \tag{3.20}$$

which leads to $\det(J(E_1)) > 0$

Hence, E_1 is locally asymptotically stable.

3.2 Global stability of the equilibruims pionts of the model covid-19

We have, the function

$$L(x) = x - 1 - \ln x, \ x > 0 \tag{3.21}$$

Theorem 27 *Assume $R_0 > 1$. E_1 is a globally asymptotically stable endemic steady-state for system (3.1)-(3.2)*

Proof 28 *Consider the system of (3.1). Consider a Lyapunov function*

$$W' = \int_\Omega \left[S^* L\left(\frac{S}{S^*}\right) + I^* L\left(\frac{I}{I^*}\right) + \frac{k+\eta+\mu}{k} H^* L\left(\frac{H}{H^*}\right) \right] dx. \tag{3.22}$$

Differentiating W' with respect to time yields

$$\begin{aligned}
\frac{dW'}{dt} &= \int_\Omega \left[\left(1 - \frac{S^*}{S}\right) \frac{dS}{dt} + \left(1 - \frac{I^*}{I}\right) \frac{dI}{dt} + \frac{k+\eta+\mu}{k} \left(1 - \frac{H^*}{H}\right) \frac{dH}{dt} \right] dx \\
&= \int_\Omega \left(1 - \frac{S^*}{S}\right) (a\Delta S + \pi - \beta SI - \mu S)\, dx \\
&\quad + \int_\Omega \left(1 - \frac{I^*}{I}\right) (b\Delta I + \beta SI - (k+\eta+\mu)\, I)\, dx \\
&\quad + \int_\Omega \frac{k+\eta+\mu}{k} \left(1 - \frac{H^*}{H}\right) (c\Delta H + kI - (\delta+\gamma+\mu)\, H)\, dx. \\
&= M + \acute{M},
\end{aligned}$$

where

$$\begin{aligned}
M &= \int_\Omega \left(1 - \frac{S^*}{S}\right) (a\Delta S)\, dx + \int_\Omega \left(1 - \frac{I^*}{I}\right) (b\Delta I)\, dx \\
&\quad + \int_\Omega \frac{k+\eta+\mu}{k} \left(1 - \frac{H^*}{H}\right) (c\Delta H)\, dx,
\end{aligned} \tag{3.23}$$

and

$$\begin{aligned}
\acute{M} &= \int_\Omega \left(1 - \frac{S^*}{S}\right) (\pi - \beta SI - \mu S) + \left(1 - \frac{I^*}{I}\right) (\beta SI - (k+\eta+\mu)\, I) \\
&\quad + \frac{k+\eta+\mu}{k} \left(1 - \frac{H^*}{H}\right) (kI - (\delta+\gamma+\mu)\, H)\, dx \\
&= \frac{dW}{dt} dx \leq 0.
\end{aligned} \tag{3.24}$$

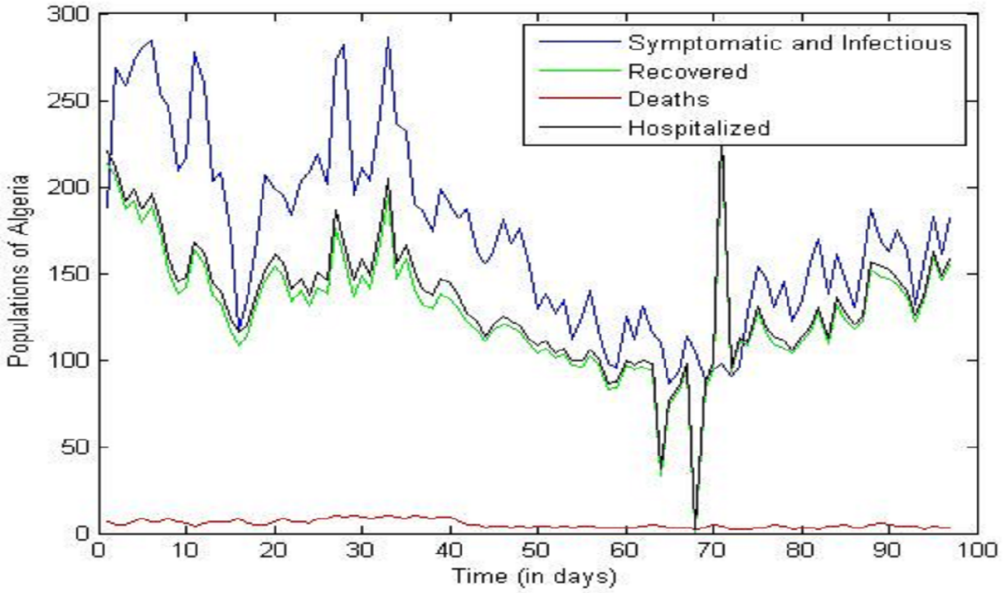
We start by looking at M Using Green's formula and assuming the Neumann boundary conditions, we obtain

$$\begin{aligned} M &= -a\int_\Omega \nabla\left(1-\frac{S^*}{S}\right)\nabla S dx - b\int_\Omega \nabla\left(1-\frac{I^*}{I}\right)\nabla I dx - c\int_\Omega \nabla\left(1-\frac{H^*}{H}\right)\nabla H dx \\ &= -a\int_\Omega \frac{S^*}{S^2}\left|\nabla S\right|^2 dx - b\int_\Omega \frac{I^*}{I^2}\left|\nabla I\right|^2 dx - a\int_\Omega \frac{H^*}{H^2}\left|\nabla H\right|^2 dx \leq 0. \end{aligned} \tag{3.25}$$

Hence $\frac{dW'}{dt} < 0$, and, consequently, W' is non increasing in time with $W'(t) = 0$ only at the steady state E_1. The global asymptotic stability of E_1 follows from Lyapunov's direct method's.

3.3 Model Validation of Algeria

the results are shown in Figure 1 illustrates the daily reported data of COVID-19 cases of Algeria [35]. It contains total confirmed cases, recovered cases and death cases due to COVID-19 infection from February 24 to May 31, 2021.



Reported cumulated data of COVID-19 in Algeria from February 24 to May 22, 2021

3.4 Conclusion

Based on the transmission dynamics of COVID-19 in Algeria, we constructed a time dependent simple SIHR mathematical model. We computed the disease free equilibrium point, endemic equilibrium point and basic reproduction number of the model. Also, we discussed about the local and global stability of the disease at the equilibrium points. The disease free equilibrium point is locally and globally stable when $R_0 < 1$ and unstable when $R_0 > 1$. Further, the endemic equilibrium point is locally and globally stable when $R_0 > 1$. Thus, for the stability of disease COVID-19, we need to get $R_0 < 1$ using diffierent types of control measures such as social distancing, self-isolation, testing facilities, face mask wearing etc...

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