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Theme

Mathematical Modeling for Exploring the Influence of Infectious Diseases

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بِسْمِ اللَّهِ الرَحمَنِ الرَحِيمْ

"وَقُلِ اعْمَلُواْ فَسَيَرَى اللّهُ عَمَلَكُمْ وَرَسُولُهُ
وَالْمُؤْمِنُونَ وَسَتُرَدُّونَ إِلَى عَالِمِ الْغَيْبِ وَالشَّهَادَةِ
فَيُنَبِّئُكُم بِمَا كُنتُمْ تَعْمَلُونَ" [التوبة: 105]

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Notation

$\mathbb{N}_0$	Natural numbers including zero $\{0, 1, 2, 3, \ldots\}$.
$\mathbb{N}$	Natural numbers excluding zero $\{1, 2, 3, \ldots\}$.
$\mathbb{R}$	Real numbers $(-\infty, \infty)$.
$\mathbb{R}_+$	Positive real numbers $(0, \infty)$.
$\mathbb{R}^*$	Nonzero real numbers $(-\infty, 0) \cup (0, \infty)$.
$C(J, \mathbb{C})$	The BANACH space of all continuous functions φ on $J \subset \mathbb{R}$, for which $\|\varphi\|_\infty = \sup_{\eta \in J} \|\varphi(\eta)\|$.
ODE	Ordinary differential equations.
$\mathfrak{Rb}$	Basic reproduction number.
$\mathfrak{Dfe}$	Disease-free equilibruim point.
$\mathfrak{Eqp}$	Endemic equilibrium point.

Contents

Introduction

Mathematical modeling has become a powerful and effective tool for studying health phenomena in a structured and quantitative manner. It allows scientists to translate biological and behavioral processes into equations that can be analyzed and used to make accurate predictions about the development of epidemics. In this context, mathematical modeling forms a foundation for better understanding how diseases spread and interact with various health factors [11].

Chronic diseases are among the main health challenges faced by healthcare systems around the world. These diseases have long-term effects on people's lives, the quality of healthcare, and both social and economic stability.

Common chronic diseases include diabetes, high blood pressure, heart and vascular diseases, and chronic respiratory illnesses. These conditions usually require lifelong treatment and regular medical follow-up. Their impact is not only limited to direct health issues, but they also increase vulnerability in society. People with chronic diseases are more likely to get infections and suffer complications, as was clearly seen during the COVID-19 pandemic, where those most affected by the virus were already living with chronic conditions.

Recent global health crises, especially the COVID-19 pandemic [20], have shown how fragile health systems can be and how hard it is to predict how complex diseases behave, especially when they overlap with chronic illnesses. Because of this, there is an urgent need for accurate scientific tools to understand how different health factors interact, how diseases spread, and how to design effective interventions. In this context, mathematical modelling has become a powerful and useful tool to study health phenomena in a structured and quantitative way. It allows scientists to translate biological and behavioural processes into equations that can be analysed and used to make accurate predictions about the development of epidemics.

This is why the first chapter provides a complete theoretical introduction to mathematical modelling, explaining what it is, the different types of models, and how they are used

in applied sciences. The focus is especially on their use in the health field, which benefits greatly from this approach. The chapter also covers how modelling has evolved over time and gives examples of different types of models, such as linear and nonlinear models, and deterministic and stochastic ones. This helps build a strong background for readers before going into the details of the specific model used in the study.

As the science of mathematical epidemiology has progressed, there has been a need to create more complex and accurate models that better reflect social and population realities. This is especially important as chronic and infectious diseases are now more likely to appear together. For this reason, the second chapter gives a detailed explanation of different mathematical models used in epidemiology, such as SI, SIS, SIR, SIRS, SEIR, and others. These models are classified based on their features and applications.

In the third chapter, the focus shifts to the SECIR model, a deterministic model based on differential equations. This model is unique because it includes a category for people with chronic diseases, which adds a new layer of understanding of how chronic conditions affect the spread of infections. The SECIR model includes five main categories: S (Susceptible), E (Exposed), C (Chronic), I (Infected), R (Recovered).

This chapter presents a mathematical model explaining how a disease spreads among different social groups, considering immune and health factors. It explains the model's assumptions and the biological meaning of the mathematical relationships used. The chapter also includes a detailed mathematical analysis to ensure the solutions exist and are valid, using mathematical theorems, and identifies biologically feasible values. The basic reproduction number $\mathfrak{R}\mathfrak{b}$ is calculated to assess the disease's ability to spread. Additionally, the stability of the disease-free and endemic states is analyzed. Finally, the chapter offers practical recommendations to support prevention strategies, such as enhancing vaccination campaigns and improving medical follow-up.

CHAPTER 1

APPLICATIONS OF MATHEMATICAL MODELLING IN BIOLOGY

1.1 Introduction to Mathematical Modelling

Mathematical modelling is a powerful tool that employs mathematical structures and techniques to represent real-world systems. In biology, it provides insights into complex processes, predicts outcomes, and informs decision-making [13]. Dynamical systems, a key framework in mathematical modelling, describe the evolution of a system over time based on fixed rules, often expressed through differential equations [9, 10, 11, 12].

Types of Models

Mathematical models can be broadly categorized into two types:

1. **Deterministic Models**

   (a) **Continuous:** Described using differential equations.

   (b) **Discrete:** Described using difference equations.

2. **Stochastic Models**
   These incorporate randomness and are used when systems involve probabilistic outcomes.

Both deterministic and stochastic models fall under the umbrella of dynamical systems, which describe how systems evolve over time.

Deterministic vs. Non-Deterministic Systems

- **Deterministic Systems:** The future state is uniquely determined by the current state and fixed rules.

- **Non-Deterministic Systems:** The evolution involves randomness or multiple possible outcomes.

Diffusion Processes in Biological Systems

Definition 1.1 (Diffussion)**.** *Diffusion is the movement of particles from an area of high concentration to an area of low concentration. This process is fundamental in biological systems and can be modeled mathematically.*

Biological Examples of Diffusion

1. Oxygen and carbon dioxide exhange in the lungs.
2. Nutrient transport across cell membranes.
3. Spread of infectious diseases within a population.

Applications of Mathematical Modelling in Biology

- **Epidemiology:** Modelling the spread of infectious diseases (e.g., COVID-19, Malaria).
- **Ecology:** Studying population dynamics and predator-prey interactions.
- **Genetics:** Analyzing gene regulatory networks and genetic drift.
- **Physiology:** Understanding heart rhythms and neural activity.

Steps in Building Mathematical Models

1. **Identify the Problem:** Define the biological system or process to be modeled.
2. **Formulate the Model:** Choose appropriate mathematical structures (e.g., differential equations).
3. **Analyse the Model:** Solve equations, determine feasible regions, and investigate properties like existence, uniqueness, and stability.
4. **Obtain Necessary Tools:** Use metrics like the basic reproduction number ($\mathfrak{Rb}$) and equilibrium points.
5. **Validate the Model:** Compare predictions with real-world data.
6. **Refine the Model:** Adjust parameters or assumptions based on validation results.

1.2 Compartmental Models in Epidemiology

Compartmental models divide populations into compartments representing different health states (e.g., Susceptible, Infected, Recovered). These models are widely used to study disease dynamics.

Assumptions in Compartmental Models

1. **Closed Population:** No immigration or emigration.
2. **Constant Population Size:** The population remains fixed during the study period.
3. **Homogeneous Mixing:** Individuals interact uniformly, with a constant contact rate.
4. **No Social Structure:** Factors like age, gender, or social groups do not influence transmission rates.
5. **Fixed Infectious Period:** Individuals remain infectious for the same average duration.
6. **Permanent Immunity:** Recovered individuals cannot be reinfected.
7. **No Latency Period:** Individuals become infectious immediately upon exposure.
8. **No Mutations:** The disease does not evolve or change during the study period.

These assumptions can be modified to reflect more complex dynamics, such as reinfection, latency periods, or population heterogeneity.

Advantages and Limitations of Compartmental Models

Advantages

Discribe how these models are straight for word constract and interpret, especially in understanding general trends.

Limitations

Point out that compartmental models often oversimplify reality, ignoring factors like spatial structure, age distribution, and individual heterogeneity, which may effect accuracy.

Single Population Dynamics

Let $N(t)$ represent the number of individuals in a population at time t, with N_0 being the initial population size.

Key Processes

- **Birth Rate** (b): Increase in population due to reproduction.
- **Death Rate** (d): Decrease in population due to mortality.
- **Immigration** $(i(t))$: External individuals entering the population.
- **Emigration** $(e(t))$: Individuals leaving the population.

Exponential Growth Model

The population grows or declines exponentially under the constant birth or death rate. For a closed population $(i = e = 0)$

$$\begin{aligned} \frac{dN}{dt} &= bN(t) - dN(t) + i(t) - e(t) \\ &= (b-d)N(t) = rN(t), \end{aligned}$$

where $r = b - d$ is the net growth rate. The solution is

$$N(t) = N_0 \exp^{rt}.$$

- If $r > 0$, the population grows exponentially.
- If $r < 0$, the population declines.
- If $r = 0$, the population remains constant.

Logistic Growth Model

Exponential growth is unrealistic over long periods due to resource limitations. The logistic model incorporates a carrying capacity K, representing the maximum sustainable population size:

$$\frac{dN}{dt} = rN(t)\left(1 - \frac{N(t)}{K}\right).$$

Therefore

$$\frac{K}{N\left(K-N\right)}dN = rdt. \tag{1.1}$$

As

$$\frac{K}{N\left(K-N\right)} = \frac{1}{N} + \frac{1}{K-N},$$

then (1.1) becomes

$$\frac{dN}{N} + \frac{dN}{K-N} = rdt.$$

If we integrate it, we get

$$\ln|N| - \ln|K-N| = rt + c_1,$$

then

$$\frac{N}{K-N} = c_0 \exp\left(rt\right).$$

Thus

$$N\left(t\right) = \frac{K}{1 + \left(\frac{K-N_0}{N_0}\right)\exp\left(-rt\right)}.$$

- If $t = 0 \Rightarrow N\left(t\right) = N_0$.
- If $t \to \infty \Rightarrow N\left(t\right) \to K$.

Basic Multi-Population Dynamics

SI Model

SI is a simple mathematical model used in epidemiology to describe the spread of infectious diseases in a population N divided into two compartments:

- Susceptible (S): individuals who can contract the disease.
- Infectious (I): individuals who have the disease and can transmit it to susceptible individuals.

The dynamics are described by:

$$\begin{cases} \frac{dS}{dt} = -\frac{\beta I}{N}S, \\ \frac{dI}{dt} = \frac{\beta I}{N}S, \end{cases}$$

where β is the transmission rate.

SIS Model

SIS model [21] is an extension of the SI model used in epidemiology to describe the spread of infectious diseases where individuals can recover from the infection but do not develop lasting immunity. After recovery, individuals return to the susceptible state, making it possible for them to be infected again.

The model of SIS is often described by the following differentail equations.

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

where γ is the recovery rate.

SIR Model

SIR model is a fundamental mathematical model used in epidemiology to describe how infectious diseases spread through a population over time. Unlike the SI and SIS models, the SIR model includes recovery with immunity, meaning that once individuals recover from the disease, they are no longer susceptible to reinfection.

The model of SIR is governed by a system of differentail equations:

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

Where $S(t)$, $I(t)$, and $R(t)$ represent susceptible, infectious, and recovered population at time t. Additionally, β is the transmission rate and γ is the recovery rate.

SEIR Model

SEIR model is an extension of the SIR model used in epidemiology to describe the spread of infectious diseases that have an incubation period-a delay between when they become infectious, this delay is represented by the exposed (E) compartment.

This model of SEIR is described by the following system of differential equations:

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

Where δ is the incubation rate (rate at which exposed individuals become infectious). Here, $\frac{1}{\delta}$ is the average incubation period.

1.3 Basic Reproduction Number and Equilibrium Points

Basic Reproduction Number

The basic reproduction number [10] is a key epidemiological metric used to describe the contagiousnness or transmissibility of an infectious disease. It represents the average number of secondary infections produced by a single infected individual in a completely susceptible population (i.e., no one is immune, and there are no interventions like vaccines or public health measures in place).

We give $\mathfrak{Rb}$ by the following relationship

$$\mathfrak{Rb} = \frac{\text{the infection transmission rate}}{\text{the recovery rate}} = \frac{\beta}{\gamma}$$

in case where the models are simple such as (SI, SIR, ...) and without less of immunity.

- $\mathfrak{Rb} > 1$: the infection will likely spread in the population, potentially leading to an outbreak or epidemic.
- $\mathfrak{Rb} = 1$: the disease will remain stable in the population, without causing large outbreaks.
- $\mathfrak{Rb} < 1$: the infection will likely die out over time, as each infected person transmits the disease to less than one other person on average.

Equilibrium Points

Equilibrium points are states where the system does not change over time. For a system of differential equations:

$$\frac{dx}{dt} = f(x),$$

an equilibrium point x^* satisfies $f(x^*) = 0$.

Stability is determined by analyzing the eigenvalues of the Jacobian matrix:

- **Stable:** where the eigenvalues of the Jacobian matrix are negative.
- **Unstable:** where the eigenvalues of the Jacobian matrix are non-negative.

CHAPTER 2

MATHEMATICAL EPIDEMIOLOGICAL MODELS

Epidemiology is the study of the distribution and determinants of chronic and infectious disease prevalence in humans. In 1760, Daniel Bernoulli[1] used mathematics to describe the spread of smallpox in a variolated population. Since then epidemiological modelling has suffered dips in popularity and reprisals in interest. Today, the most interesting problems in disease tracking, control, and prevention are modeled mathematically using epidemiological processes. More recently, researchers are turning to computer simulations to verify analytic results. When analytic methods become too arduous or even impossible, these simulations can provide insight into complicated situations.

2.1 SI Model

We note that $N(t)$ is the number of individuals in a population at time t, and N_0 the number of individuals in the beginning.

In 1906 appeared the first dynamic model of Hammer. We assume that the population divided into two compartments S and I, then

$$N = S(t) + I(t),$$

The chart of SI is presented as follows:

[1]Daniel Bernoulli is a Swiss doctor, physicist and mathematician, born in Groningen on February 8, 1700, died in Basel on March 17, 1782.

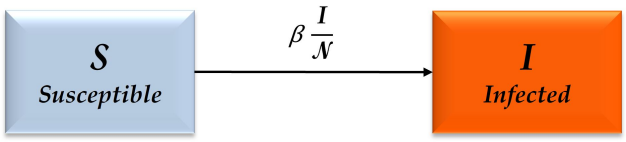

Figure 2.1: SI Model

where

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

The total population is constant

$$\begin{aligned} N &= S + I \Rightarrow \frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} = 0. \\ &\Rightarrow N = \text{constant.} \end{aligned}$$

Equilibruim points

We calculate

$$\frac{dS}{dt} = \frac{dI}{dt} = 0.$$

We have

$$\frac{dI}{dt} = \frac{\beta I}{N} S = \frac{\beta}{N} I \left(N - I\right),$$

then

$$\frac{dI}{dt} = 0 \Rightarrow \begin{cases} I = 0, \\ I = N. \end{cases}$$

The disease-free equilibruim point is existed when $I = 0$. Consequently,

$$\mathfrak{Dfe} = (S_0, I_0) = (N, 0).$$

The endemic equilibruim point if

$$\mathfrak{Eqp} = (S^*, I^*) = (0, N).$$

Stability of Equilibruim Point

If we put

$$g\left(S, I\right) = \frac{\beta}{N} I \left(N - I\right),$$

then

$$\frac{\partial g}{\partial S} = 0, \qquad \text{and} \qquad \frac{\partial g}{\partial I} = \beta - \frac{2\beta I}{N}.$$

If we replace the value of $\mathfrak{Dfe}$ we get

$$\frac{\partial g}{\partial I}(\mathfrak{Dfe}) = \beta > 0,$$

then $\mathfrak{Dfe}$ is unstable.

When we replace $\mathfrak{Eqp}$ we obtain

$$\frac{\partial g}{\partial I}(\mathfrak{Eqp}) = -\beta < 0,$$

then $\mathfrak{Eqp}$ is stable.

2.2 SIS Model

SIS model was proposed by Kermack[1] and Mckendrick[2] in 1932.The chart is outlined below

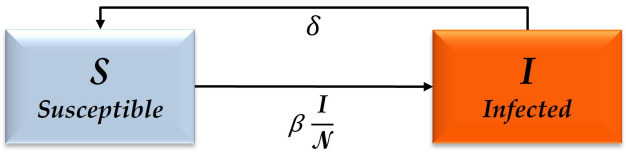

Figure 2.2: SIS Model

The ODE for the SIS model can be analytically solved to understand the disease dynamics as follows:

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

with γ is the removal rate of infection.

The total population is constant

$$\begin{aligned} N &= S + I \Rightarrow \frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} = 0. \\ &\Rightarrow N = \text{constant}. \end{aligned}$$

[1]William Kermack (1898-1970) he was a Scottish biochemist, and epidemiologist,best known for developing the Kermack Mckendrick model with Anderson Mckendrick.

[2]Anderson Gray Mckendrick (1876-1943) was aScottish physician, epidemiologist, and mathematician known for his contribution to mathematical epidemiology.

Basic Reproduction Number $(\mathfrak{Rb})$

As the SIS model [16] is composed of one infection component I, we obtain

$$\mathfrak{Rb} = \frac{\beta}{\gamma}.$$

Equilibruim points

At equilibrium, we solve

$$\frac{dS}{dt} = \frac{dI}{dt} = 0.$$

We have

$$\begin{aligned} \frac{dI}{dt} &= \frac{\beta I}{N}(N-I) - \gamma I \\ &= \left[\frac{\beta}{N}(N-I) - \gamma\right] I. \end{aligned}$$

Therefore,

$$\frac{dI}{dt} = 0 \Rightarrow \begin{cases} I = 0, \\ \frac{\beta}{N}(N-I) - \gamma = 0. \end{cases}$$

Then the disease-free equilibruim point is existed when $I = 0$. Consequently,

$$\mathfrak{Dfe} = (S_0, I_0) = (N, 0).$$

The endemic equilibrium point is exists when $\mathfrak{Rb} > 1$. Consequently,

$$\mathfrak{Eqp} = (S^*, I^*) = N\left(\frac{1}{\mathfrak{Rb}}, \frac{\mathfrak{Rb}-1}{\mathfrak{Rb}}\right).$$

Stability of equilibrium point

If we put

$$g(S, I) = \frac{\beta}{N} I(N-I) - \gamma I,$$

then

$$\frac{\partial g}{\partial S} = 0, \qquad \text{and} \qquad \frac{\partial g}{\partial I} = \beta - \gamma - \frac{2\beta I}{N}.$$

If we replace the value of $\mathfrak{Dfe}$ we get

$$\frac{\partial g}{\partial I}(\mathfrak{Dfe}) = \beta - \gamma = \gamma(\mathfrak{Rb} - 1).$$

Therefore, $\mathfrak{Dfe}$ is stable if and only if $\mathfrak{Rb} < 1$.

When we replace $\mathfrak{Eqp}$ we obtain

$$\frac{\partial g}{\partial I}\left(\mathfrak{Eqp}\right) = -\gamma\left(\mathfrak{Rb} - 1\right).$$

Then, $\mathfrak{Eqp}$ is stable when $\mathfrak{Rb} > 1$.

2.3 SIR Model

The chart is listed below

Figure 2.3: SIR Model

The system of ODEs describing the dynamics of the SIR model is expressed as follows:

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

Summing the three equations

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt}. \\ &= -\beta\frac{I}{N}S + \beta\frac{I}{N}S - \gamma I + \gamma I = 0, \end{aligned}$$

thus,

$$\frac{dN}{dt} = 0.$$

Hence, N is constant.

Equilibrium Points and Basic Reproduction Number

Equilibrium points

Equilibrium points are found by setting

$$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0.$$

The disease-free equilibrium point is existed when $I = 0$. Consequently,

$$\mathfrak{Dfe} = (S_0, I_0, R_0) = (N, 0, 0)\,.$$

The endemic equilibruim point is existed if $I > 0$, then $\gamma = 0$. That leads us to the SI model which we studied before in detail.

Basic Reproduction Number

Basic reproduction number $\mathfrak{Rb}$ is the number of new infections generated by one infected individual in a completely susceptible population. Since the SIR model includes an infection component I, we obtain,

$$\begin{aligned} \frac{dI}{dt} &= f(t) - v(t)\,. \\ &= \beta\frac{I(t)}{N}S(t) - \gamma I(t)\,, \end{aligned}$$

where f is the rate of new infection and v is the rate of other transitions;

$$f(t) = \beta\frac{I(t)}{N}S(t)\,, \qquad v(t) = \gamma I(t)\,.$$

The new infection matrix $\mathcal{F}$ and the transition matrix $\mathcal{V}$ are assessed at the disease-free equilibrium point as follows

$$\mathcal{F} = \left.\frac{df}{dI}\right|_{\mathfrak{Dfe}} = \frac{\beta S_0}{N} = \beta, \qquad \text{and} \qquad \mathcal{V} = \left.\frac{dv}{dI}\right|_{\mathfrak{Dfe}} = \gamma.$$

From this, we get

$$\mathcal{V}^{-1} = \frac{1}{\gamma}.$$

Consequently,

$$\mathfrak{Rb} = \rho\left(\mathcal{F}\mathcal{V}^{-1}\right) = \frac{\beta}{\gamma}.$$

Stability of the Disease-Free Equilibrium Point

To determine the stability of the disease-free equilibrium $\mathfrak{Dfe}$, we compute the Jacobian matrix at this point:

$$J = \begin{pmatrix} \frac{\partial\left(-\beta\frac{I}{N}S\right)}{\partial S} & \frac{\partial\left(-\beta\frac{I}{N}S\right)}{\partial I} & \frac{\partial\left(-\beta\frac{I}{N}S\right)}{\partial R} \\ \frac{\partial\left(\beta\frac{I}{N}S-\gamma I\right)}{\partial S} & \frac{\partial\left(\beta\frac{I}{N}S-\gamma I\right)}{\partial I} & \frac{\partial\left(\beta\frac{I}{N}S-\gamma I\right)}{\partial R} \\ \frac{\partial(\gamma I)}{\partial S} & \frac{\partial(\gamma I)}{\partial I} & \frac{\partial(\gamma I)}{\partial R} \end{pmatrix},$$

then

$$J = \begin{pmatrix} -\beta\frac{I}{N} & -\beta\frac{S}{N} & 0 \\ \beta\frac{I}{N} & \beta\frac{S}{N} - \gamma & 0 \\ 0 & \gamma & 0 \end{pmatrix}.$$

Consequently,

$$J_{\mathfrak{Dfe}} = \begin{pmatrix} 0 & -\beta & 0 \\ 0 & \beta - \gamma & 0 \\ 0 & \gamma & 0 \end{pmatrix}.$$

The stability of $\mathfrak{Dfe}$ depends on the eigenvalues of the Jacobian matrix. Solving the characteristic equation:

$$\det(J_{\mathfrak{Dfe}} - \lambda I_{3\times 3}) = 0.$$

Or

$$\begin{vmatrix} -\lambda & -\beta & 0 \\ 0 & \beta - \gamma - \lambda & 0 \\ 0 & \gamma & -\lambda \end{vmatrix} = 0.$$

The characteristic equation is given by,

$$(-\lambda)(\beta - \gamma - \lambda)(-\lambda) = 0.$$

Thus,

$$\lambda^2 [\gamma(\mathfrak{Rb} - 1) - \lambda] = 0.$$

$$\lambda_1 = 0, \qquad \lambda_2 = -\beta + \gamma, \qquad \lambda_3 = -\gamma.$$

Then $\lambda_2 = \gamma(\mathfrak{Rb} - 1)$ will be negative if $\mathfrak{Rb} = \frac{\beta}{\gamma} < 1$ (i.e. $\beta < \gamma$), and thus the disease-free equilibrium point is stable when $\mathfrak{Rb} < 1$.

2.4 SIRS Model

SIRS model accounts for loss of immunity after recovery, meaning individuals in the R compartment can return to the S compartment at a rate δ. The chart is shown below

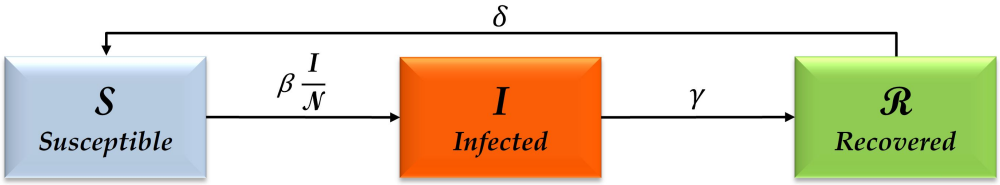

Figure 2.4: SIRS Model

The system of equations for the model is given below:

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

Summing the three equations:

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \\ &= -\beta \frac{I}{N} S + \delta R + \beta \frac{I}{N} S - \gamma I + \gamma I - \delta R \\ &= 0. \end{aligned}$$

Hance, N is constant.

As N is constant, we can normalize the model SIRS, we put

$$\mathcal{S}(t) = \frac{S(t)}{N}, \qquad \mathcal{I}(t) = \frac{I(t)}{N}, \qquad \mathcal{R}(t) = \frac{R(t)}{N},$$

Then we get

$$\begin{cases} \frac{d\mathcal{S}}{dt} = -\beta \mathcal{S}\mathcal{I} + \sigma \mathcal{R}, \\ \frac{d\mathcal{I}}{dt} = \beta \mathcal{S}\mathcal{I} - \gamma \mathcal{I}, \\ \frac{d\mathcal{R}}{dt} = \gamma \mathcal{I} - \delta \mathcal{R}. \end{cases}$$

Equilibrium Points and Basic Reproduction Number

To find the equilibrium points $\left(\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0\right)$.

If $I = 0$ (Disease-free Equilibrium), then $\mathfrak{Dfe} = (S_0, I_0, R_0) = (1, 0, 0)$.

If $I > 0$ (Endemic Equilibrium)

From $\frac{dI}{dt} = \frac{dR}{dt} = 0$, we find

$$S = \frac{\gamma}{\beta}, \qquad R = \frac{\gamma}{\delta} I.$$

Then

$$\begin{aligned} I &= 1 - S - R \\ &= 1 - \frac{\gamma}{\beta} - \frac{\gamma}{\delta} I. \\ \Rightarrow \quad I^* &= \frac{1 - \frac{\gamma}{\beta}}{1 + \frac{\gamma}{\delta}}. \end{aligned}$$

Thus

$$\mathfrak{Eqp} = (S^*, I^*, R^*) = \left(\frac{\gamma}{\beta}, I^*, \frac{\gamma}{\delta} I^* \right).$$

Basic Reproduction Number ($\mathfrak{Rb}$)

Because the SIRS model is composed for one infection component I, we obtain:

$$f - v = \beta I(t) S(t) - \gamma I(t).$$

Accordingly,

$$f = \beta I(t) S(t), \qquad v = \gamma I(t).$$

Here, f is the rate of appearance of new infections, and v is the rate of other transitions.

The new infection matrix $\mathcal{F}$ and transition matrix $\mathcal{V}$ are assessed at the disease-free equilibrium point, as follows:

$$\mathcal{F} = \left. \frac{df}{dI} \right|_{\mathfrak{Dfe}} = \frac{\beta S_0}{N} = \beta, \qquad \text{and} \qquad \mathcal{V} = \left. \frac{dv}{dI} \right|_{\mathfrak{Dfe}} = \gamma.$$

It follows that

$$\mathcal{V}^{-1} = \frac{1}{\gamma},$$

Then

$$\mathcal{F}\mathcal{V}^{-1} = \frac{\beta S_0}{\gamma}.$$

Thus

$$\mathfrak{Rb} = \frac{\beta}{\gamma}.$$

We can rewrite $I^*$of the endimic equilibrium point as follows

$$\begin{aligned} I^* &= \frac{1 - \frac{\gamma}{\beta}}{1 + \frac{\gamma}{\delta}} = \frac{\frac{\gamma}{\beta}\left(\frac{\beta}{\gamma} - 1\right)}{\frac{\gamma}{\delta}\left(\frac{\delta}{\gamma} + 1\right)} \\ &= \frac{\delta(\mathfrak{Rb} - 1)}{\delta \mathfrak{Rb} + \beta}, \end{aligned}$$

and

$$\mathfrak{Eqp} = (S^*, I^*, R^*) = \left(\frac{\gamma}{\beta}, \frac{\delta\left(\mathfrak{Rb} - 1\right)}{\delta\mathfrak{Rb} + \beta}, \frac{\gamma\left(\mathfrak{Rb} - 1\right)}{\delta\mathfrak{Rb} + \beta} \right).$$

The endemics points exists when $\mathfrak{Rb} > 1$.

Stability Analysis of the Equilibrium Points

After computing the Jacobian matrix, we get

$$J = \begin{pmatrix} -\beta I & -\beta S & \delta \\ \beta I & \beta S - \gamma & 0 \\ 0 & \gamma & -\delta \end{pmatrix}.$$

At the disease-free equilibrium point $\mathfrak{Dfe} = (S_0 = 1,\ I_0 = 0,\ R_0 = 0)$

$$J_{\mathfrak{Dfe}} - \lambda I_{3\times 3} = \begin{pmatrix} -\lambda & -\beta & \delta \\ 0 & \beta - \gamma - \lambda & 0 \\ 0 & \gamma & -\delta - \lambda \end{pmatrix}.$$

Thus

$$\lambda\left(\delta + \lambda\right)\left(\gamma\left(\mathfrak{Rb} - 1\right) - \lambda\right) = 0.$$

If $\mathfrak{Rb} = \frac{\beta}{\gamma} < 1$, (i.e., $\beta < \gamma$) , then all the eigenvalues are negative, therefore the disease-free equilibrium is stable when $\mathfrak{Rb} < 1$.

$$J_{\mathfrak{Dfe}*} = \begin{pmatrix} -\frac{\delta\gamma(\mathfrak{Rb}-1)}{\delta+\gamma} & -\gamma & \delta \\ \frac{\delta\gamma(\mathfrak{Rb}-1)}{\delta+\gamma} & 0 & 0 \\ 0 & \gamma & -\delta \end{pmatrix}.$$

Let $\psi = \frac{\delta\gamma(\mathfrak{Rb}-1)}{\delta+\gamma}$, then

$$J_{\mathfrak{Eqp}} - \lambda I_{3\times 3} = \begin{pmatrix} -\psi - \lambda & -\gamma & \delta \\ \psi & -\lambda & 0 \\ 0 & \gamma & -\delta - \lambda \end{pmatrix}.$$

The characteristic polynomial is given by

$$\begin{aligned} P\left(\lambda\right) &= \left(-\psi - \lambda\right)\left(-\lambda\right)\left(-\delta - \lambda\right) - \psi\left(-\psi - \lambda\right)\left(-\delta - \lambda\right) \\ &= -\left(\psi + \lambda\right)^2\left(\delta + \lambda\right). \end{aligned}$$

Then $\lambda = -\delta$ or $\lambda = -\psi = -\frac{\delta\gamma(\mathfrak{Rb}-1)}{\delta+\gamma}$ will be negative if $\mathfrak{Rb} = \frac{\beta}{\gamma} > 1$, (i.e., $\beta > \gamma$) , and thus the endemic equilibrium point $\mathfrak{Eqp}$ is stable when $\mathfrak{Rb} > 1$.

2.5 SEIRS Model

The chart of SEIRS is depicted below

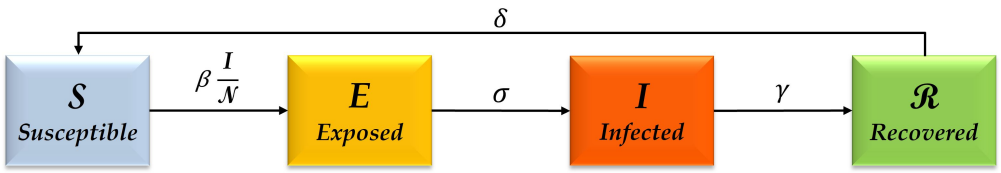

Figure 2.5: SEIRS Model

The dynamics of the SEIRS model are as follows :

$$\begin{cases} \frac{dS}{dt} = \Lambda N + \delta R - \left(\beta \frac{I}{N} + \upsilon + \mu\right) S, \\ \frac{dE}{dt} = \beta \frac{I}{N} S - (\sigma + \mu) E, \\ \frac{dI}{dt} = \sigma E - (\gamma + \mu) I, \\ \frac{dR}{dt} = \upsilon S + \gamma I - (\delta - \mu) R. \end{cases}$$

Here

- S is the susceptible individuals.
- E is the exposed individuals.
- I expresses the infected individuals.
- R presents the recovered individuals.
- Λ is the birth rate.
- β the infection transmission rate.
- υ represents the vaccination rate.
- σ shows the transition rate from E to I.
- γ presents the recovery rate.
- δ is the immunity loss rate.
- μ expresses the death rate.

Remark 2.1. *If we set $\delta = 0$, SEIRS becomes SEIR, which represents the same model with no loss of immunity and the calculations remain the same in both models.*

The Boundedness of the Total Population

Let

$$N(t) = S(t) + E(t) + I(t) + R(t),$$

This implies that

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}.$$

Now, adding all the equations of the system, we obtain:

$$\begin{aligned} \frac{dN}{dt} &= \Lambda N - \mu N \\ &= (\Lambda - \mu) N. \end{aligned}$$

Separation of variables

$$\begin{aligned} \ln N - \ln N_0 &= (\Lambda - \mu) t \\ N &= N_0 \exp((\Lambda - \mu) t). \end{aligned}$$

So, we get

$$N(t) \leq N_0 \exp(\Lambda T).$$

Where T is the maximum time in our study to the model and N_0 is the initial total population at $t = 0$. Then, N is bounded.We assume the existence of a positive constant

$$\mathcal{N} \leq N_0 \exp(\Lambda T).$$

To normalize the SEIRS model, we put:

$$\mathcal{S}(t) = \frac{S(t)}{\mathcal{N}}, \quad \mathcal{E}(t) = \frac{E(t)}{\mathcal{N}}, \quad \mathcal{I}(t) = \frac{I(t)}{\mathcal{N}}, \quad \mathcal{R}(t) = \frac{R(t)}{\mathcal{N}},$$

then, we obtain

$$\begin{cases} \frac{d\mathcal{S}}{dt} = \Lambda - (\beta\mathcal{I} + \upsilon + \mu)\mathcal{S} + \delta\mathcal{R}, \\ \frac{d\mathcal{E}}{dt} = \beta\mathcal{I}\mathcal{S} - (\sigma + \mu)\mathcal{E}, \\ \frac{d\mathcal{I}}{dt} = \sigma\mathcal{E} - (\gamma + \mu)\mathcal{I}, \\ \frac{d\mathcal{R}}{dt} = \upsilon\mathcal{S} + \gamma\mathcal{I} - (\delta + \mu)\mathcal{R}. \end{cases}$$

Disease-free eqiulibruim ($\mathfrak{D}\mathfrak{f}\mathfrak{p}$)

When $\mathcal{I} = 0$ and $\mathcal{E} = 0$. The equations simplify to:

$$\begin{cases} \frac{d\mathcal{S}}{dt} = \Lambda + \delta\mathcal{R} - (\upsilon + \mu)\mathcal{S} = 0, \\ \frac{d\mathcal{R}}{dt} = \upsilon\mathcal{S} - (\delta + \mu)\mathcal{R} = 0. \end{cases}$$

From these equations, we deduse:

$$\mathcal{S} = \frac{\Lambda(\delta+\mu)}{(\upsilon+\mu)(\delta+\mu)-\delta\upsilon}, \qquad \mathcal{R} = \frac{\upsilon\mathcal{S}^*}{\delta+\mu}.$$

Thus, the disease-free equilibrium is :

$$\mathfrak{Dfp} = (\mathcal{S}_0, \mathcal{E}_0, \mathcal{I}_0, \mathcal{R}_0) = \left(\frac{\Lambda(\delta+\mu)}{(\upsilon+\mu)(\delta+\mu)-\delta\upsilon}, 0, 0, \frac{\upsilon\mathcal{S}^*}{\delta+\mu}\right).$$

Endemic Equilibrium Points

$$\begin{cases} 0 = \Lambda + \delta\mathcal{R}(t) - (\beta\mathcal{I}(t) + \upsilon + \mu)\mathcal{S}(t), & (eq1) \\ 0 = \beta\mathcal{I}(t)\mathcal{S}(t) - (\sigma+\mu)\mathcal{E}(t), & (eq2) \\ 0 = \sigma\mathcal{E}(t) - (\gamma+\mu)\mathcal{I}(t), & (eq3) \\ 0 = \upsilon\mathcal{S}(t) + \gamma\mathcal{I}(t) - (\delta+\mu)\mathcal{R}(t). & (eq4) \end{cases}$$

From equations (eq3), and (eq4), we have

$$\mathcal{I}(t) = \frac{\sigma}{\gamma+\mu}\mathcal{E}(t), \qquad \mathcal{R}(t) = \frac{\upsilon}{\delta+\mu}\mathcal{S}(t) + \frac{\gamma\sigma}{(\delta+\mu)(\gamma+\mu)}\mathcal{E}(t).$$

If we add (eq1) to (eq2) we obtain

$$\mathcal{S}^* = \frac{\Lambda(\delta+\mu)}{\mu(\gamma+\mu+\delta)} - \frac{\delta(\gamma+\delta+\mu)+(\gamma+\mu)(\sigma+\mu)}{(\gamma+\mu)(\upsilon+\mu+\delta)}\mathcal{E}^*.$$

Equation (eq2) gives us

$$\mathcal{E}^* = \frac{(\gamma+\mu)(\sigma+\mu)(\delta+\upsilon+\mu)}{\beta[\delta(\gamma+\delta+\mu)+(\gamma+\mu)(\delta+\mu)]}\left(\frac{\beta\Lambda(\delta+\mu)}{\mu(\gamma+\mu)(\sigma+\mu)(\delta+\upsilon+\mu)} - 1\right).$$

Then

$$\mathfrak{Eqp} = (\mathcal{S}^*, \mathcal{E}^*, \mathcal{I}^*, \mathcal{R}^*) = \left(\frac{(\sigma+\mu)(\gamma+\mu)}{\beta\sigma}, \frac{(\gamma+\mu)}{\sigma}\mathcal{I}^*, \mathcal{I}^*, \frac{\upsilon\mathcal{S}^*+\gamma\mathcal{I}^*}{\delta+\mu}\right).$$

Consequently, we obtain the required endemic equilibrium point $\mathfrak{Eqp}$, which exists for

$$\frac{\beta\Lambda(\delta+\mu)}{\mu(\gamma+\mu)(\sigma+\mu)(\delta+\upsilon+\mu)} > 1.$$

Basic reproduction number ($\mathfrak{Rb}$)

Because the SEIRS model is composed of two infection components $\mathcal{E}$ and $\mathcal{I}$, we obtain:

$$f_i - v_i = \begin{pmatrix} \beta\mathcal{I}\mathcal{S} - (\sigma+\mu)\mathcal{E} \\ \sigma\mathcal{E} - (\gamma+\mu)\mathcal{I} \end{pmatrix}.$$

Accordingly,

$$f_i = \begin{pmatrix} \beta \mathcal{I} \mathcal{S} \\ 0 \end{pmatrix}, \qquad v_i = \begin{pmatrix} (\sigma + \mu)\, \mathcal{E} \\ -\sigma \mathcal{E} + (\gamma + \mu)\, \mathcal{I} \end{pmatrix}$$

The new infection matrix $\mathcal{F}$ and transition matrix $\mathcal{V}$ are assessed at the disease-free equilibrium point, as follows:

$$\mathcal{F} = \left. \frac{df_i}{dI} \right|_{\mathfrak{Dfe}}, \qquad \text{and} \qquad \mathcal{V} = \left. \frac{dv_i}{dI} \right|_{\mathfrak{Dfe}}$$

Then

$$\mathcal{F} = \begin{pmatrix} 0 & \beta \mathcal{S}_0 \\ 0 & 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} \sigma + \mu & 0 \\ -\sigma & \gamma + \mu \end{pmatrix}.$$

It follows that

$$\mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{\sigma+\mu} & 0 \\ \frac{\sigma}{(\gamma+\mu)(\sigma+\mu)} & \frac{1}{\gamma+\mu} \end{pmatrix}.$$

Then

$$\mathfrak{Rb} = \frac{\beta \sigma}{(\sigma + \mu)\,(\gamma + \mu)}.$$

Thus, the endemic equilibrium point $\mathfrak{Eqp}$ exists when $\mathfrak{Rb} > 1$.

Stability Analysis Of The Equilibrium Points Using The Jacobian Matrix

We define

$$J = \begin{pmatrix} -(\beta \mathcal{I} + \upsilon + \mu) & 0 & -\beta \mathcal{S} & \delta \\ \beta \mathcal{I} & -(\sigma + \mu) & \beta \mathcal{S} & 0 \\ 0 & \sigma & -(\gamma + \mu) & 0 \\ \upsilon & 0 & \gamma & -(\delta + \mu) \end{pmatrix}.$$

The system has two equilibrium points, we analyse the eigenvalues of the Jacobian matrix at these points.

Stabilility of the Disease-Free equilibrium

Substituting

$$S = \frac{\Lambda\,(\delta + \mu)}{(\upsilon + \mu)\,(\delta + \mu) - \delta \upsilon}, \qquad E = 0, \qquad I = 0, \qquad R = \frac{\Lambda \upsilon}{\mu\,(\delta + \upsilon + \mu)}.$$

To determine stability, we find its the eigenvalues by solving

$$\det(J_{\mathfrak{Dfe}} - \lambda I_{4\times 4}) = 0.$$

Then

$$\begin{vmatrix} -(\upsilon+\mu)-\lambda & 0 & -\frac{\beta\Lambda(\delta+\mu)}{(\upsilon+\mu)(\delta+\mu)-\delta\upsilon} & \delta \\ 0 & -(\sigma+\mu)-\lambda & \frac{\beta\Lambda(\delta+\mu)}{(\upsilon+\mu)(\delta+\mu)-\delta\upsilon} & 0 \\ 0 & \sigma & -(\gamma+\mu)-\lambda & 0 \\ \upsilon & 0 & \gamma & -(\delta+\mu)-\lambda \end{vmatrix} = 0.$$

Then

$$\mathfrak{Rb} = \frac{\beta\sigma}{(\sigma+\mu)(\gamma+\mu)}.$$

$$\omega = \frac{\beta\Lambda(\delta+\mu)}{(\upsilon+\mu)(\delta+\mu)-\delta\upsilon}$$

then

$$\mathfrak{Rb} = \frac{\omega}{(\gamma+\mu)(\sigma+\mu)}.$$

Let

$$\det(J_{\mathfrak{Dfe}} - \lambda I_{4\times 4}) = \lambda^2 + (\gamma+\mu+\sigma+\mu)\lambda + (\sigma+\mu)(\gamma+\mu) - \sigma\omega = 0.$$

Thus

$$\lambda^2 + a\lambda + b = 0,$$

where

$$\begin{aligned} a &= \gamma+\mu+\sigma+\mu = \gamma+\sigma+2\mu, \\ b &= (\sigma+\mu)(\gamma+\mu) - \sigma\omega. \end{aligned}$$

The general solution is

$$\lambda = \frac{-a \pm \sqrt{a^2-4b}}{2}.$$

We have the following eigenvalues

$$\lambda_1 = -\mu, \qquad \lambda_2 = -(\delta+\upsilon+\mu),$$

and λ_i are negative for each $i \in \{3,4\}$, if and only if $1-\sigma\mathfrak{Rb} > 0$, which means

$$\mathfrak{Rb} < \frac{1}{\sigma}.$$

- If $\mathfrak{Rb} < 1$, Then, making all eigenvalues negative. Thus $\mathfrak{Dfp}$ is stable.
- If $\mathfrak{Rb} > 1$, Then $\lambda_2 > 0$, meaning $\mathfrak{Dfp}$ is unstable.

I'll compute the eigenvaluse at the endemic equilibrium using

$$S^* = \frac{(\sigma+\mu)(\gamma+\mu)}{\beta\sigma}, \;\; I^* > 0.$$

The Jacobian Matrix are:

$$J_{\mathfrak{Eqp}} = \begin{pmatrix} -(\beta\mathcal{I}^* + \upsilon + \mu) & 0 & -\beta\mathcal{S}^* & \delta \\ \beta\mathcal{I}^* & -(\sigma+\mu) & \beta\mathcal{S}^* & 0 \\ 0 & \sigma & -(\gamma+\mu) & 0 \\ \upsilon & 0 & \gamma & -(\delta+\mu) \end{pmatrix}.$$

To determine stability, we find its the eigenvalues by solving

$$\det(J_{\mathfrak{Eqp}} - \lambda I_{4\times 4}) = 0.$$

Then

$$\begin{vmatrix} -(\beta\mathcal{I}^* + \upsilon + \mu) - \lambda & 0 & -\beta\mathcal{S}^* & \delta \\ \beta\mathcal{I}^* & -(\sigma+\mu) - \lambda & \beta\mathcal{S}^* & 0 \\ 0 & \sigma & -(\gamma+\mu) - \lambda & 0 \\ \upsilon & 0 & \gamma & -(\delta+\mu) - \lambda \end{vmatrix} = 0.$$

For $\mathfrak{Rb} > 1$, we get:

$$\lambda_1 = -\mu, \qquad \lambda_2 = \mu(\mathfrak{Rb} - 1).$$

- If $\mathfrak{Rb} > 1,$ the endemic equilibrium exists and is stable, meaning the disease can persists in the population.
- If $\mathfrak{Rb} < 1,$ the endemic equilibrium does not exist, meaning the disease disappears.

CHAPTER 3

INFLUENCE OF INFECTIOUS DISEASES ON POPULATION WITH CHRONIC CONDITIONS

Chronic diseases are typically characterized as conditions that persist for one year or more, require ongoing medical attention, and often limit daily activities. Individuals suffering from chronic conditions, including heart disease, diabetes, cancer, obstructive pulmonary disease, chronic kidney disease, and obesity, exhibit an increased susceptibility to respiratory distress induced by infectious diseases. This susceptibility underscores the intricate interplay between chronic and infectious diseases and the compounded health challenges faced by these individuals [3].

Mathematical modelling is a valuable tool for understanding and controlling infectious diseases [11]. However, traditional integer-order derivative models are commonly used to capture dynamic interactions between distinct points in real-world scenarios. To enhance the realism and applicability of such models, we introduce ordinary differential equations, which play an important role across diverse fields such as optimization, artificial intelligence, and medical diagnostics.

In our study, we employ the SECIR model, dividing the total population (denoted as N) into five distinct classes: Susceptible S, Exposed E, Chronic diseases C, Infected I, and Recovered R (see [3]). The parameters of the SECIR model are defined as follows:

◦ Λ is the influx rate that expresses the arrival of people (birth rate and visitors from other societies).

◦ We denote by $\mu \leq \Lambda$ the rate of natural death population.

◦ In addition, σ signifies the death rate of infected individuals with chronic conditions, excluding infectious diseases. The inclusion of σ accounts for the higher significance of the death rate among individuals with chronic diseases compared with other compartments.

◦ β_1 and β_2 are the contact rates between the susceptible S and infectious populations C

and I respectively.

◦ θ and δ describe the rates of transfer of exposed E to infectious populations C and I respectively.

◦ p and q represent the recovery rates from infectious populations C and I respectively.

◦ κ and γ are the death rates of the populations C and I due to the infectious diseases respectively.

◦ υ is the vaccine rate of the suspected population, and τ is the rate of transfer of recovered population R even those who were vaccinated to the susceptible population S, (taking into account some infectious diseases have various versions, and the vaccine might not always be fully effective due to mutations).

In this study, we focus on using ordinary derivatives to construct and analyse our mathematical model. This approach allows for the effective description of disease dynamics without resorting to fractional-order techniques, while still providing reliable insights into the structure and behaviour of the system under study (see [2, 5, 9, 17, 20, 1]).

Motivated by the above-mentioned work, for $0 \leq t \leq T < \infty$, we have:

$$\begin{cases} \frac{dS(t)}{dt} &= \Lambda N\left(t\right) - \left(\frac{\beta_1 C(t)+\beta_2 I(t)}{N(t)} + \upsilon + \mu\right) S\left(t\right) + \tau R\left(t\right), \\ \frac{dE(t)}{dt} &= \frac{\beta_1 C(t)+\beta_2 I(t)}{N(t)} S\left(t\right) - \left(\theta + \delta + \mu\right) E\left(t\right), \\ \frac{dC(t)}{dt} &= \theta E\left(t\right) - \left(p + \kappa + \sigma + \mu\right) C\left(t\right), \\ \frac{dI(t)}{dt} &= \delta E\left(t\right) - \left(q + \gamma + \mu\right) I\left(t\right), \\ \frac{dR(t)}{dt} &= \upsilon S\left(t\right) + pC\left(t\right) + qI\left(t\right) - \left(\tau + \mu\right) R\left(t\right). \end{cases} \tag{3.1}$$

The alterations in infectious disease transmission within the SECIR model (3.1) can be interpreted through reference to the following chart:

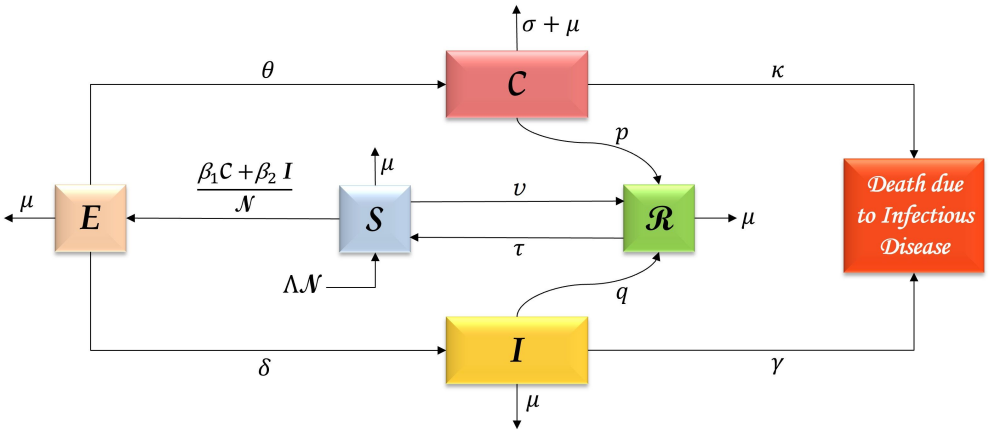

Figure 3.1: Transmission scheme for the SECIR model of an infectious disease.

Our primary objective is to efficiently study the characteristics, behaviour, and dynamics of the system of nonlinear ordinary differential equations (3.1). We focus on determining the main properties of the solution in a biologically feasible region, including stability, existence, boundedness, and computation of equilibrium points. Using fixed-point theory, we establish the conditions for obtaining a unique solution.

All the following results are presented in detail in [3].

3.1 Dynamic Analysis of the Feasible Region

In this section, we elucidate the essential definitions derived from the mathematical modelling. The space under consideration is the Banach space of continuous functions $C\left([0,1],\mathbb{R}\right)$, characterized by the norm

$$\|u\|_{\infty}=\sup_{t\in[0,T]}|u\left(t\right)|.$$

3.1.1 Positivity and Boundedness of the Model

The SECIR model (3.1) is investigated within a biologically feasible region in $\mathbb{R}_{+}^{5}$, as defined in the subsequent lemma.

Lemma 3.1. *Let N_0 be the initial total population at $t=0$, where $0\leq t\leq T<\infty$, then the solution of the considered population dynamic model is restricted to the feasible region given by*

$$(S,E,C,I,R)\in\mathbb{R}_{+}^{5},\ S\left(t\right)+E\left(t\right)+C\left(t\right)+I\left(t\right)+R\left(t\right)=N\left(t\right)\leq N_{0}\exp\left(\left(\Lambda-\mu\right)T\right).$$

This shows the boundedness of the population dynamic model under consideration.

Proof. Let

$$N\left(t\right)=S\left(t\right)+E\left(t\right)+C\left(t\right)+I\left(t\right)+R\left(t\right),$$

this implies that

$$\frac{dN\left(t\right)}{dt}=\frac{dS\left(t\right)}{dt}+\frac{dE\left(t\right)}{dt}+\frac{dC\left(t\right)}{dt}+\frac{dI\left(t\right)}{dt}+\frac{dR\left(t\right)}{dt}.$$

Now, adding all the equations of (3.1), we get

$$\begin{aligned}\frac{dN\left(t\right)}{dt} &= \left(\Lambda-\mu\right)N\left(t\right)-\left(\kappa+\sigma\right)C\left(t\right)-\gamma I\left(t\right)\\ &\leq \left(\Lambda-\mu\right)N\left(t\right).\end{aligned}$$

Thus,

$$N(t) \leq N_0 + (\Lambda - \mu) \int_0^t N(\tau)\, d\tau.$$

After using Gronwall lemma, we obtain

$$N(t) \leq N_0 \exp\left((\Lambda - \mu) T\right),$$

where N_0 is the initial total population at $t = 0$. □

In the subsequent sections of this thesis, we assume the existence of a positive constant

$$\mathcal{N} \leq N_0 \exp\left((\Lambda - \mu) T\right),$$

for which the total population N remains fixed throughout our study, which can be expressed as $N(t) = \mathcal{N}$, for any $t \in [0, T]$. This assumption is made to normalize the SECIR model (3.1). Therefore, we put:

$$\mathcal{S}(t) = \tfrac{S(t)}{\mathcal{N}}, \quad \mathcal{E}(t) = \tfrac{E(t)}{\mathcal{N}}, \quad \mathcal{C}(t) = \tfrac{C(t)}{\mathcal{N}}, \quad \mathcal{I}(t) = \tfrac{I(t)}{\mathcal{N}}, \quad \mathcal{R}(t) = \tfrac{R(t)}{\mathcal{N}}, \tag{3.2}$$

then we obtain

$$\begin{cases} \frac{d\mathcal{S}(t)}{dt} &= \Lambda - (\beta_1 \mathcal{C}(t) + \beta_2 \mathcal{I}(t) + \upsilon + \mu)\, \mathcal{S}(t) + \tau \mathcal{R}(t), \\ \frac{d\mathcal{E}(t)}{dt} &= (\beta_1 \mathcal{C}(t) + \beta_2 \mathcal{I}(t))\, \mathcal{S}(t) - (\theta + \delta + \mu)\, \mathcal{E}(t), \\ \frac{d\mathcal{C}(t)}{dt} &= \theta \mathcal{E}(t) - (p + \kappa + \sigma + \mu)\, \mathcal{C}(t), \\ \frac{d\mathcal{I}(t)}{dt} &= \delta \mathcal{E}(t) - (q + \gamma + \mu)\, \mathcal{I}(t), \\ \frac{d\mathcal{R}(t)}{dt} &= \upsilon \mathcal{S}(t) + p\mathcal{C}(t) + q\mathcal{I}(t) - (\tau + \mu)\, \mathcal{R}(t), \end{cases} \tag{3.3}$$

along with the positive initial conditions

$$\mathcal{S}(0) = u_1, \quad \mathcal{E}(0) = u_2, \quad \mathcal{C}(0) = u_3, \quad \mathcal{I}(0) = u_4, \quad \mathcal{R}(0) = u_5. \tag{3.4}$$

3.1.2 Existence Results of Solutions for the Normalized Model

In this section, we explore the existence and uniqueness of solutions to problem (3.6)–(3.7) through the field of fixed-point theory. Our investigation employs Banach's theorems, as outlined in [6, 7, 8].

Let $u = (\mathcal{S}, \mathcal{E}, \mathcal{C}, \mathcal{I}, \mathcal{R}) \in \Omega$, where $\Omega = [C([0, T], [0, 1])]^5$ is a Banach space equipped with the norm

$$\|u\|_\Omega = \max\left\{\|\mathcal{S}\|_\infty, \|\mathcal{E}\|_\infty, \|\mathcal{C}\|_\infty, \|\mathcal{I}\|_\infty, \|\mathcal{R}\|_\infty\right\},$$

and let $f = (f_1, f_2, f_3, f_4, f_5)$, be such that

$$\begin{cases} f_1(t, u(t)) &= \Lambda - (\beta_1 \mathcal{C}(t) + \beta_2 \mathcal{I}(t) + \upsilon + \mu)\mathcal{S}(t) + \tau \mathcal{R}(t), \\ f_2(t, u(t)) &= (\beta_1 \mathcal{C}(t) + \beta_2 \mathcal{I}(t))\mathcal{S}(t) - (\delta + \theta + \mu)\mathcal{E}(t), \\ f_3(t, u(t)) &= \theta \mathcal{E}(t) - (p + \kappa + \mu + \sigma)\mathcal{C}(t), \\ f_4(t, u(t)) &= \delta \mathcal{E}(t) - (q + \gamma + \mu)\mathcal{I}(t), \\ f_5(t, u(t)) &= \upsilon \mathcal{S}(t) + p\mathcal{C}(t) + q\mathcal{I}(t) - (\tau + \mu)\mathcal{R}(t), \end{cases} \tag{3.5}$$

it is clear that f is a continuous function.

By applying integral to both sides of the system

$$\frac{du(t)}{dt} = f(t, u(t)), \tag{3.6}$$

taking into account the conditions

$$u(0) = u_0 = (u_1, u_2, u_3, u_4, u_5), \tag{3.7}$$

and we obtain the following system of ordinary integral equations

$$u(t) = u_0 + \int_0^t f(\tau, u(\tau))\, d\tau,$$

which is equivalent to the original problem (3.6)–(3.7).

Theorem 3.1. *Let $\beta_1, \beta_2, p, q, \theta, \delta, \kappa, \gamma, \tau, \upsilon, \sigma, \mu, T \in \mathbb{R}_+$, be such that*

$$\ell = \max\{\beta_1 + \beta_2 + \upsilon, \delta + \theta, p + \kappa + \sigma, q + \gamma, \tau\}.$$

We assume

$$T(\ell + \mu) < 1. \tag{3.8}$$

Hence, there exists a unique solution to problem (3.6)–(3.7) on $[0, T]$.

Proof. Our proof begins with a transformation of problem (3.6)–(3.7) into the fixed-point problem $\mathcal{A}u(t) = u(t)$, with

$$\mathcal{A}u(t) = (\mathcal{A}_1 u(t), \mathcal{A}_2 u(t), \mathcal{A}_3 u(t), \mathcal{A}_4 u(t), \mathcal{A}_5 u(t))$$

and

$$\mathcal{A}u(t) = u_0 + \int_0^t f(\tau, u(\tau))\, d\tau. \tag{3.9}$$

We observe that when $u \in \Omega$, then $(\mathcal{A}_i u)_{1 \le i \le 5}$ are indeed continuous. Consequently, $\mathcal{A}u$ is an element of Ω and is equipped with the norm

$$\|\mathcal{A}u\|_\Omega = \max_{1 \le i \le 5} \|\mathcal{A}_i u\|_\infty .$$

Problem (3.6)–(3.7) is equivalent to (3.9), which means that $\mathcal{A}$ includes fixed points that solve the aforementioned problem. Let $u, v \in \Omega$, then

$$|\mathcal{A}_i u(t) - \mathcal{A}_i v(t)| \leq \int_0^t |f_i(\tau, u(\tau)) - f_i(\tau, v(\tau))| \, d\tau, \quad \forall i = \overline{1,5}. \tag{3.10}$$

For all $t \in [0, T]$, we have:

$$\begin{aligned}
|f_1(t, u(t)) - f_1(t, v(t))| &\leq \max\{\tau, \beta_1 + \beta_2 + \upsilon + \mu\} \|u - v\|_\Omega, \\
|f_2(t, u(t)) - f_2(t, v(t))| &\leq \max\{\beta_1 + \beta_2, \delta + \theta + \mu\} \|u - v\|_\Omega, \\
|f_3(t, u(t)) - f_3(t, v(t))| &\leq \max\{\theta, p + \kappa + \mu + \sigma\} \|u - v\|_\Omega, \\
|f_4(t, u(t)) - f_4(t, v(t))| &\leq \max\{\delta, q + \gamma + \mu\} \|u - v\|_\Omega, \\
|f_5(t, u(t)) - f_5(t, v(t))| &\leq \max\{q, p, \upsilon, \tau + \mu\} \|u - v\|_\Omega.
\end{aligned}$$

Then

$$|f_i(t, u(t)) - f_i(t, v(t))| \leq (\ell + \mu) \|u - v\|_\Omega, \quad \forall i = \overline{1,5}. \tag{3.11}$$

From (3.10) we find

$$\|\mathcal{A}_i u - \mathcal{A}_i v\|_\infty \leq T(\ell + \mu) \|u - v\|_\Omega, \quad \forall i = \overline{1,5},$$

and

$$\|\mathcal{A}u - \mathcal{A}v\|_\Omega \leq T(\ell + \mu) \|u - v\|_\Omega.$$

Thus, according to (3.8), $\mathcal{A}$ is considered a contraction operator.

Banach's contraction principle helps us infer that $\mathcal{A}$ has only one fixed point, which is the unique solution to problem (3.6)–(3.7) on $[0, T]$. □

3.2 Analysis for the SECIR Model

For a future analysis, we assume that the vaccine rate υ of the suspected population satisfies the following inequality:

$$\max\{p, q\} \leq \upsilon \leq \mu + \min\{\delta + \theta, p + \kappa + \sigma, q + \gamma\}. \tag{3.12}$$

Basic Reproduction Number and Equilibrium Points

In this section, we determine the basic reproduction number of system (3.3) using the next-generation matrix method. The basic reproduction number, denoted as $(\mathfrak{Rp})$, represents the average number of secondary infections generated when a single infection is introduced into the susceptible population. It can be computed as the spectral radius of matrix $\mathcal{FV}^{-1}$.

Theorem 3.2. *The basic reproduction number of system (3.3) is expressed as*

$$\mathfrak{Rp} = \frac{\Lambda\left(\tau+\mu\right)}{\mu\left(\theta+\delta+\mu\right)\left(\tau+\upsilon+\mu\right)}\left(\frac{\theta\beta_1}{p+\kappa+\sigma+\mu}+\frac{\delta\beta_2}{q+\gamma+\mu}\right). \tag{3.13}$$

Proof. In the SECIR model, consisting of infection components $\mathcal{E}$, $\mathcal{C}$, and $\mathcal{I}$, we obtain

$$\bar{f}_i - \bar{v}_i = \begin{pmatrix} \left(\beta_1\mathcal{C}+\beta_2\mathcal{I}\right)\mathcal{S} - \left(\theta+\delta+\mu\right)\mathcal{E} \\ \theta\mathcal{E} - \left(p+\kappa+\sigma+\mu\right)\mathcal{C} \\ \delta\mathcal{E} - \left(q+\gamma+\mu\right)\mathcal{I} \end{pmatrix}.$$

Accordingly,

$$\bar{f}_i = \begin{pmatrix} \left(\beta_1\mathcal{C}+\beta_2\mathcal{I}\right)\mathcal{S} \\ 0 \\ 0 \end{pmatrix}, \quad \bar{v}_i = \begin{pmatrix} \left(\theta+\delta+\mu\right)\mathcal{E} \\ \left(p+\kappa+\sigma+\mu\right)\mathcal{C} - \theta\mathcal{E} \\ \left(q+\gamma+\mu\right)\mathcal{I} - \delta\mathcal{E} \end{pmatrix}.$$

Here, $\bar{f}_i$ represents the rate of new infections appearing in compartment i, and $\bar{v}_i$ denotes the rate of transitions between compartment i and other infected compartments for each $i \in \{1,2,3\}$.

The new infection matrix $\mathcal{F}$ and transition matrix $\mathcal{V}$ are evaluated at the disease-free equilibrium point $(\mathfrak{D}\mathfrak{f}\mathfrak{p})$ according to 3.3 as follows:

$$\mathcal{F} = \begin{pmatrix} 0 & \beta_1\mathcal{S}_0 & \beta_2\mathcal{S}_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \theta+\mu+\delta & 0 & 0 \\ -\theta & p+\kappa+\sigma+\mu & 0 \\ -\delta & 0 & q+\gamma+\mu \end{pmatrix}.$$

According to the applied method principle, the basic reproduction number is defined as the spectral radius of the matrix product $\mathcal{F}\mathcal{V}^{-1}$ and is expressed by (3.13). □

The initial phase of comprehending a differential equation involves, as customary, identifying its equilibrium points. In epidemiology, we focus on two distinct categories of equilibrium points:

- Disease-free equilibrium is defined as the point at which no disease (or death from disease) is present in the population, which is represented in the model as $\mathcal{E} = \mathcal{C} = \mathcal{I} = 0$.
- The other equilibrium points, where $\mathcal{C} \neq 0, \mathcal{I} \neq 0$, are denoted as endemic equilibrium points (or outbreak equilibrium points).

Based on (3.12), we define the positive real values

$$\begin{aligned}
&\lambda_1 = \delta + \theta + \mu, && \lambda_2 = p + \kappa + \sigma + \mu,\\
&\lambda_3 = q + \gamma + \mu, && \eta_1 = \upsilon\left(\kappa + \sigma\right) + \mu\left(\upsilon - p\right),\\
&\eta_2 = \upsilon\gamma\delta + \mu\delta\left(\upsilon - q\right), && \eta_3 = \lambda_1\left(\lambda_3 - \upsilon\right) + \lambda_2\left(\lambda_1 - \upsilon\right) + \lambda_3\left(\lambda_2 - \upsilon\right),\\
&\eta_4 = \frac{\eta_1\theta}{\lambda_2} + \frac{\eta_2\delta}{\lambda_3}, && \eta_5 = \mu + \frac{\theta\left(\kappa + \sigma + \mu\right)}{\lambda_2} + \frac{\delta\left(\gamma + \mu\right)}{\lambda_3},
\end{aligned}$$

to simplify the calculation for getting the following theorem.

Theorem 3.3. *The system (3.3) has two types of equilibrium points*

1. *Disease-free equilibrium*

$$\mathfrak{Dfp} = \left(\mathcal{S}_0, \mathcal{E}_0, \mathcal{C}_0, \mathcal{I}_0, \mathcal{R}_0\right) = \left(\frac{\Lambda\left(\tau + \mu\right)}{\mu\lambda_4}, 0, 0, 0, \frac{\Lambda\upsilon}{\mu\lambda_4}\right).$$

2. *Endemic equilibrium point* $\mathfrak{Eqp} = \left(\mathcal{S}^*, \mathcal{E}^*, \mathcal{C}^*, \mathcal{I}^*, \mathcal{R}^*\right)$ *which is*

$$\mathfrak{Eqp} = \left(\frac{\lambda_1}{\lambda_6}, \frac{\lambda_1\left(\mathfrak{Rp} - 1\right)}{\lambda_5\lambda_6}, \frac{\theta\lambda_1\left(\mathfrak{Rp} - 1\right)}{\lambda_2\lambda_5\lambda_6}, \frac{\delta\lambda_1\left(\mathfrak{Rp} - 1\right)}{\lambda_3\lambda_5\lambda_6}, \mathcal{R}^*\right),$$

where

$$\mathcal{R}^* = \mathcal{R}_0\left(1 - \frac{\lambda_0\lambda_1}{\lambda_5\lambda_6}\left(\mathfrak{Rp} - 1\right)\right),$$

with

$$\begin{aligned}
&\lambda_0 = \frac{\eta_4 + \upsilon\mu}{\Lambda\upsilon}, && \lambda_4 = \tau + \upsilon + \mu,\\
&\lambda_5 = \frac{\tau\eta_5 + \mu\lambda_1}{\mu\lambda_4}, && \lambda_6 = \frac{\beta_1\theta}{\lambda_2} + \frac{\beta_2\delta}{\lambda_3}.
\end{aligned}$$

The existence of the endemic equilibrium point is contingent upon $\mathfrak{Rp} > 1$.

Proof. To determine the equilibrium point of model (3.3), we set $\frac{du(t)}{dt} = \vec{0}$, with $u = \left(\mathcal{S}, \mathcal{E}, \mathcal{C}, \mathcal{I}, \mathcal{R}\right)$. Therefore

$$\begin{cases}
0 &= \Lambda - \left(\beta_1\mathcal{C}\left(t\right) + \beta_2\mathcal{I}\left(t\right) + \upsilon + \mu\right)\mathcal{S}\left(t\right) + \tau\mathcal{R}\left(t\right) & \left(eq1\right)\\
0 &= \left(\beta_1\mathcal{C}\left(t\right) + \beta_2\mathcal{I}\left(t\right)\right)\mathcal{S}\left(t\right) - \lambda_1\mathcal{E}\left(t\right) & \left(eq2\right)\\
0 &= \theta\mathcal{E}\left(t\right) - \lambda_2\mathcal{C}\left(t\right) & \left(eq3\right)\\
0 &= \delta\mathcal{E}\left(t\right) - \lambda_3\mathcal{I}\left(t\right) & \left(eq4\right)\\
0 &= \upsilon\mathcal{S}\left(t\right) + p\mathcal{C}\left(t\right) + q\mathcal{I}\left(t\right) - \left(\tau + \mu\right)\mathcal{R}\left(t\right) & \left(eq5\right)
\end{cases}$$

From equations $\left(eq3\right)$ and $\left(eq4\right)$ we find

$$\mathcal{C}\left(t\right) = \frac{\theta}{\lambda_2}\mathcal{E}\left(t\right) \quad \text{and} \quad \mathcal{I}\left(t\right) = \frac{\delta}{\lambda_3}\mathcal{E}\left(t\right).$$

If we add $(eq1)$ to $(eq2)$ we obtain

$$\mathcal{R}(t) = \frac{1}{\tau}\left[(\upsilon + \mu)\,\mathcal{S}(t) + \lambda_1 \mathcal{E}(t) - \Lambda\right],$$

then we replace it in $(eq5)$ to get

$$\mathcal{S}(t) = \mathcal{S}_0 - \lambda_5 \mathcal{E}(t),$$

then

$$\mathcal{R}(t) = \mathcal{R}_0\left[1 - \lambda_0 \mathcal{E}(t)\right].$$

1. If $\mathcal{E} = 0$, we can easily obtain the disease-free equilibrium point $\mathfrak{Dfp}$.

2. In the case $\mathcal{E} \neq 0$, $(eq2)$ gives us

$$\mathcal{E}^* = \frac{\lambda_1}{\lambda_5 \lambda_6}\left(\mathfrak{Rp} - 1\right).$$

Consequently, we find the required endemic equilibrium point $\mathfrak{Eqp}$ exists for $\mathfrak{Rp} > 1$.

Hence, the theorem is proved. □

Local Asymptotic Stability

Let φ_1, φ_2 and φ_3 be such that

$$\begin{aligned}
\varphi_1 &= \lambda_8 + \frac{\lambda_1\lambda_2\lambda_3\left(\lambda_4 + \mu\right)\left(1 - \mathfrak{Rp}\right)}{\mu\lambda_4}, \\
\varphi_2 &= \lambda_8 + \frac{\mu\lambda_4\lambda_7 + \lambda_1\lambda_2\lambda_3\left(1 - \mathfrak{Rp}\right)}{\lambda_4 + \mu}, \\
\varphi_3 &= \lambda_8 + \mu\lambda_7 + \lambda_4\left(\lambda_7 + \mu\right),
\end{aligned}$$

where

$$\lambda_7 = \lambda_1 + \lambda_2 + \lambda_3, \qquad \lambda_8 = \lambda_1\lambda_2 + \lambda_1\lambda_3 + \lambda_2\lambda_3.$$

Theorem 3.4. *Suppose that*

$$\mathcal{S}_0\left(\beta_1\theta + \beta_2\delta\right) < \min\left\{\varphi_1, \varphi_2, \varphi_3\right\}, \tag{3.14}$$

then, $\mathfrak{Dfp}$ *of system (3.3) achieves local asymptotic stability when* $\mathfrak{Rp} < 1$.

Proof. The Jacobian matrix for system (3.3) can be determined as follows

$$J=\begin{pmatrix} \frac{\partial f_1}{\partial \mathcal{S}} & \frac{\partial f_1}{\partial \mathcal{E}} & \frac{\partial f_1}{\partial \mathcal{C}} & \frac{\partial f_1}{\partial \mathcal{I}} & \frac{\partial f_1}{\partial \mathcal{R}} \\ \frac{\partial f_2}{\partial \mathcal{S}} & \frac{\partial f_2}{\partial \mathcal{E}} & \frac{\partial f_2}{\partial \mathcal{C}} & \frac{\partial f_2}{\partial \mathcal{I}} & \frac{\partial f_2}{\partial \mathcal{R}} \\ \frac{\partial f_3}{\partial \mathcal{S}} & \frac{\partial f_3}{\partial \mathcal{E}} & \frac{\partial f_3}{\partial \mathcal{C}} & \frac{\partial f_3}{\partial \mathcal{I}} & \frac{\partial f_3}{\partial \mathcal{R}} \\ \frac{\partial f_4}{\partial \mathcal{S}} & \frac{\partial f_4}{\partial \mathcal{E}} & \frac{\partial f_4}{\partial \mathcal{C}} & \frac{\partial f_4}{\partial \mathcal{I}} & \frac{\partial f_4}{\partial \mathcal{R}} \\ \frac{\partial f_5}{\partial \mathcal{S}} & \frac{\partial f_5}{\partial \mathcal{E}} & \frac{\partial f_5}{\partial \mathcal{C}} & \frac{\partial f_5}{\partial \mathcal{I}} & \frac{\partial f_5}{\partial \mathcal{R}} \end{pmatrix}$$

where $f_{1\leq i\leq 5}\left(t,u\left(t\right)\right)$ represents the right hand-side of (3.3). Then

$$J=\begin{pmatrix} -\left(\beta_1\mathcal{C}+\beta_2\mathcal{I}+\upsilon+\mu\right) & 0 & -\beta_1\mathcal{S} & -\beta_2\mathcal{S} & \tau \\ \beta_1\mathcal{C}+\beta_2\mathcal{I} & -\lambda_1 & \beta_1\mathcal{S} & \beta_2\mathcal{S} & 0 \\ 0 & \theta & -\lambda_2 & 0 & 0 \\ 0 & \delta & 0 & -\lambda_3 & 0 \\ \upsilon & 0 & p & q & -\left(\tau+\mu\right) \end{pmatrix},$$

The eigenvalues of $J\left(\mathfrak{D}\mathfrak{f}\mathfrak{p}\right)$ are given as the roots of the following characteristic polynomial

$$P\left(x\right)=-\left(x^5+c_4x^4+c_3x^3+c_2x^2+xc_1+c_0\right),$$

where

$$\begin{aligned} c_0 &= \mu\lambda_1\lambda_2\lambda_3\lambda_4\left(1-\mathfrak{R}\mathfrak{p}\right), \\ c_1 &= \mu\lambda_4\left(\varphi_1-\mathcal{S}_0\left(\beta_1\theta+\beta_2\delta\right)\right), \\ c_2 &= \left(\lambda_4+\mu\right)\left(\varphi_2-\mathcal{S}_0\left(\beta_1\theta+\beta_2\delta\right)\right), \\ c_3 &= \varphi_3-\mathcal{S}_0\left(\beta_1\theta+\beta_2\delta\right), \\ c_4 &= \lambda_4+\lambda_7+\mu. \end{aligned}$$

As $\mathcal{S}_0\left(\beta_1\theta+\beta_2\delta\right)<\min\left\{\varphi_1,\varphi_2,\varphi_3\right\}$, we find c_0,c_1,c_2,c_3, and c_4 are nonnegative coefficients when $\mathfrak{R}\mathfrak{p}<1$. According to Descartes' rule, the roots of $P\left(x\right)$ are negative reals or complexes of negative real parts, which makes $\mathfrak{D}\mathfrak{f}\mathfrak{p}$ locally asymptotically stable. □

Now, let ψ_1, ψ_2 and ψ_3 be such that

$$\begin{aligned} \psi_1 &= \frac{\lambda_9\left(\lambda_1\lambda_2\lambda_3+\tau\lambda_{10}\right)+\mu\lambda_8\left(\lambda_4+\lambda_9\right)}{\mu\lambda_4}, \\ \psi_2 &= \frac{\tau\eta_3+\mu\lambda_8+\left(\lambda_9+\upsilon+\mu\right)\left(\lambda_8+\lambda_7\left(\tau+\mu\right)\right)}{\lambda_4+\mu}, \\ \psi_3 &= \lambda_4\left(\lambda_7+\mu\right)+\mu\lambda_7+\lambda_8+\lambda_9\left(\lambda_7+\tau+\mu\right), \end{aligned}$$

where

$$\begin{aligned} \lambda_9 &= \beta_1\mathcal{C}^*+\beta_2\mathcal{I}^*=\tfrac{\lambda_1}{\lambda_5}\left(\mathfrak{R}\mathfrak{p}-1\right), \\ \lambda_{10} &= \theta\left(\kappa+\sigma+\mu\right)+\delta\left(\gamma+\mu\right)+\left(\delta+\mu\right)\lambda_2+\left(\theta+\mu\right)\lambda_3+\lambda_2\lambda_3. \end{aligned}$$

Theorem 3.5. *If we put*

$$\mathcal{S}^* \left(\beta_1 \theta + \beta_2 \delta\right) < \min\left\{\psi_1, \psi_2, \psi_3\right\},$$

then, $\mathfrak{Eqp}$ *of system (3.3) achieves local asymptotic stability when* $\mathfrak{Rp} > 1$.

Proof. As we have seen in the previous theorem, the Jacobian matrix $J\left(\mathfrak{Eqp}\right)$ for (3.3) is given by

$$J_{\mathfrak{Eqp}} = \begin{pmatrix} -\left(\beta_1 \mathcal{C}^* + \beta_2 \mathcal{I}^* + \upsilon + \mu\right) & 0 & -\beta_1 \mathcal{S}^* & -\beta_2 \mathcal{S}^* & \tau \\ \beta_1 \mathcal{C}^* + \beta_2 \mathcal{I}^* & -\lambda_1 & \beta_1 \mathcal{S}^* & \beta_2 \mathcal{S}^* & 0 \\ 0 & \theta & -\lambda_2 & 0 & 0 \\ 0 & \delta & 0 & -\lambda_3 & 0 \\ \upsilon & 0 & p & q & -\left(\tau + \mu\right) \end{pmatrix}.$$

The characteristic polynomial is given by

$$P\left(x\right) = -\left(x^5 + c_4 x^4 + c_3 x^3 + c_2 x^2 + x c_1 + c_0\right),$$

where

$$\begin{aligned} c_0 &= \lambda_9 \left(\mu \lambda_1 \lambda_2 \lambda_3 + \tau \lambda_{11}\right), \\ c_1 &= \mu \lambda_4 \left(\psi_1 - \mathcal{S}^* \left(\beta_1 \theta + \beta_2 \delta\right)\right), \\ c_2 &= \left(\lambda_4 + \mu\right) \left(\psi_2 - \mathcal{S}^* \left(\beta_1 \theta + \beta_2 \delta\right)\right), \\ c_3 &= \psi_3 - \mathcal{S}^* \left(\beta_1 \theta + \beta_2 \delta\right), \\ c_4 &= \lambda_4 + \lambda_7 + \lambda_9 + \mu, \end{aligned}$$

with

$$\lambda_{11} = \mu \lambda_2 \lambda_3 + \delta \lambda_2 \left(\gamma + \mu\right) + \theta \left(\kappa + \mu + \sigma\right) \lambda_3.$$

According to Descartes' rule, if c_0, c_1, c_2, c_3, and c_4 are positive coefficients, then the roots of $P\left(x\right)$ are negative reals or complexes of negative real parts. Therefore, the required results were obtained. □

Conclusion

This research represented an important step toward enhancing scientific understanding of how chronic diseases influenced the spread of infectious diseases and offered practical solutions based on precise mathematical analysis. Through mathematical modeling using classical models such as SI, SIS, SIR, SEIR, and more advanced models like SECIR, the study focused on the impact of infectious diseases such as COVID-19 on individuals with chronic conditions like diabetes and heart disease.

The findings obtained through the SECIR model went beyond the theoretical understanding of disease dynamics; they provided a strong scientific foundation for designing more effective health policies. Decision-makers in the health sector used these mathematical tools to develop prevention and treatment strategies that strengthened the preparedness of health systems for future epidemics.

The model's results showed clear insights into disease transmission dynamics, highlighting critical factors affecting vulnerable groups. Additionally, analyzing the stability of equilibrium points allowed accurate predictions of whether the disease would die out or persist, which was crucial for planning interventions. This approach confirmed the model's importance as a predictive tool for public health decision-making.

In the future, we hope to have the opportunity to study other diseases and epidemics, such as tuberculosis and malaria, and their impact on public health using mathematical modelling.

Bibliography

[1] S. Annas, M. I. Pratama, M. Rifandi, W. Sanusi, and S. Side, *Stability analysis and numerical simulation of SEIR model for pandemic COVID-19 spread in Indonesia*, Chaos, Solit. Fract., **139** (2020), 110072.

[2] B. Basti and N. Benhamidouche, *Existence results of self-similar solutions to the Caputo-type's space-fractional heat equation*, Surveys in Math. and its Appls., **15** (2020), 153–168.

[3] B. Basti, B. Chennaf, M. A. Boubekeur, and S. Bounouiga, *Fractional Mathematical Model for Exploring the Inflence of Infectious Diseases on Population with Chronic Conditions*, Advanced Theory and Simulations, **7**(8) (2024), 2301285.

[4] B. Basti and N. Benhamidouche, *Global existence and blow-up of generalized self-similar solutions to nonlinear degenerate diffusion equation not in divergence form*, Appl. Math. E-Notes, **20** (2020), 367–387.

[5] B. Basti, R. Djemiat, and N. Benhamidouche, *Theoretical studies on the existence and uniqueness of solutions for a multidimensional nonlinear time and space-fractional reaction-diffusion/wave equation*, Mem. Differ. Equ. Math. Phys., **89** (2023), 1–16.

[6] B. Basti, Y. Arioua, and N. Benhamidouche, *Existence and uniqueness of solutions for nonlinear Katugampola fractional differential equations*, J. Math. Appl., **42** (2019), 35–61.

[7] B. Basti, Y. Arioua, and N. Benhamidouche, *Existence results for nonlinear Katugampola fractional differential equations with an integral condition*, Acta Math. Univ. Comenian., **89**(2) (2020), 243–260.

[8] B. Basti and Y. Arioua, *Existence study of solutions for a system of n-nonlinear FDEs with integral conditions*, J. Math. Phys. Anal. Geom., **18**(3) (2022), 350–367.

[9] B. Basti, N. Hammami, I. Berrabah, F. Nouioua, R. Djemiat, and N. Benhamidouche, *Stability analysis and existence of solutions for a modified SIRD model of COVID-19 with fractional derivatives*, Symmetry, **13**(8) (2021), 1431.

[10] B. Basti, *Nonlinear diffusion problems, (Course of Master two PDEs and application)*, Med Boudiaf university of M'sila , (2024/2025).

[11] F. Brauer, C. Castillo-Chavez, and Z. Feng, *Mathematical models in epidemiology*, New York, Springer-Verlag, 2019.

[12] S. Bounouiga, B. Basti and N. Benhamidouche, *Mathematical Exploration of Malaria Transmission Dynamics: Insights from Fractional Models and Numerical Simulation*, Advanced Theory and Simulations, **8**(2) (2025), 2400630.

[13] E. A. Bender, *An Introduction to Mathematical Modeling*, Courier Corporation, 2000.

[14] Y. Chen, F. Liu, Q. Yu, and T. Li, *Review of fractional epidemic models*, Appl. Math. Model., **97** (2021), 281–307.

[15] R. Cui, K. Y. Lam and Y. Lou, *Dynamics and asymptotic profiles of steady states of an epidemic model in advective environments*, J. Differential Equations, **263** (2017), 2343–2373.

[16] R. Cui, Y. Lou, *A spatial SIS model in advective heterogeneous environments*, J. Differential Equations, **261** (2016), 3305–3343.

[17] R. Djemiat, B. Basti, and N. Benhamidouche, *Analytical studies on the global existence and blow-up of solutions for a free boundary problem of two-dimensional diffusion equations of moving fractional order*, Adv. Theory Nonlinear Anal. Appl. **6**(8) (2022), 287–299.

[18] L. Edelstein-Keshet, *Mathematical models in biology*, Society for Industrial and Applied Mathematics, 2005.

[19] K. S. Nisar, M. Farman, M. Abdel-Aty, and J. Cao, *A review on epidemic models in sight of fractional calculus*, Alex. Eng. J., **75** (2023), 81–113.

[20] K. S. Nisar, S. Ahmad, A. Ullah, K. Shah, H. Alrabaiah, and M. Arfan, *Mathematical analysis of SIRD model of COVID-19 with Caputo's fractional derivative based on real data*, Results in Physics **21** (2021), 103772.

[21] R. Peng, X. Zhao, *A reaction-diffusion SIS epidemic model in a time-periodic environment*, Nonlinearity, **25** (2012), 1451–1471.

تَـــمَ بِحَمْدِ اللهِ

"الْحَمْدُ لِلَّهِ الَّذِي لَهُ مَا فِي السَّمَاوَاتِ وَمَا فِي الْأَرْضِ وَلَهُ الْحَمْدُ فِي الْآخِرَةِ وَهُوَ الْحَكِيمُ الْخَبِيرُ" [سبأ: 01]

الملخص:

تستعرض هذه المذكرة دور النمذجة الرياضية في فهم انتشار الأمراض المعدية، كما تعتمد في دراسة النماذج الكلاسيكية SI ، SIS، SIR، SIRS نظريا وتطبيقيا وتطوّر نموذجًا موسّعًا SECIR يأخذ في الاعتبار الحالة الصحية المزمنة. تُظهر النتائج أن الأمراض المزمنة تزيد من خطر العدوى وتُسرّع انتشار الأوبئة بين الفئات الهشّة. كما يتم تحليل مؤشرات أساسية مثل معدل التكاثر الأساسي Rp في ضوء العوامل الاجتماعية والبيئية والصحية. وتُبرز الدراسة أهمية توظيف النماذج الرياضية لتوجيه السياسات الصحية، خصوصًا في تخطيط حملات التلقيح وتحديد أولويات المتابعة. وتؤكد المذكرة على ضرورة تعزيز التعاون بين علماء الرياضيات وخبراء الأوبئة للاستعداد الفعّال للأوبئة المستقبلية.

الكلمات المفتاحية: النمذجة الرياضية، الأمراض المعدية، الأمراض المزمنة، النماذج الوبائية، نقاط التوازن، استقرار نقاط التوازن، عدد التكاثر الأساسي، نظرية النقطة الثابتة.

Abstract:

This thesis explores the role of mathematical modeling in understanding the spread of infectious diseases, with a focus on individuals with chronic conditions. It builds on classical models SI, SIS, SIR, SIRS and develops an extended SECIR model that accounts for chronic health status. The study shows how chronic diseases can increase infection risk and accelerate epidemic spread among vulnerable populations. It analyzes key indicators like the basic reproduction number Rp, considering social, environmental, and health factors. The findings highlight the importance of using mathematical models to guide public health policies, especially in vaccination planning and prioritizing care for high-risk groups. The thesis underscores the value of collaboration between mathematicians and epidemiologists for effective epidemic preparedness.

Key words: Mathematical modeling, Infectious diseases, Chronic diseases, Epidemiological models, Equilibrium Points, Stability of Equilibrium Points, Basic Reproduction Number, Fixed Point Theorem.