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## FACULTY OF MECHANICAL ENGINEERING
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## INSTITUTE OF MATHEMATICS
ÚSTAV MATEMATIKY


# DELAY DIFFERENTIAL EQUATIONS IN DYNAMIC SYSTEMS
DIFERENCIÁLNÍ ROVNICE SE ZPOŽDĚNÍM V DYNAMICKÝCH SYSTÉMECH

**MASTER'S THESIS**
DIPLOMOVÁ PRÁCE


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FACULTY OF MECHANICAL ENGINEERING

# Assignment Master's Thesis

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| Institut: | Institute of Mathematics |
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| Degree programm: | Applied Sciences in Engineering |
| Branch: | Mathematical Engineering |
| Supervisor: | **doc. Mgr. Zdeněk Opluštil, Ph.D.** |
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As provided for by the Act No. 111/98 Coll. on higher education institutions and the BUT Study and Examination Regulations, the director of the Institute hereby assigns the following topic of Master's Thesis:

## Delay Differential Equations in Dynamic Systems

**Brief Description:**

Delay differential equations are important in many areas of science. For example, they are used in biology, medicine, physics, and engineering applications. A time delay arises because a finite time is required to respond to a process change in some mathematical models. It turns out that in some models, modified dynamical systems with delayed differential equations better describe real situations. As in the ordinary case, the solution of equations with delay can be found rarely. So, it is very important to study their qualitative properties, particularly the stability of solutions.

**Master's Thesis goals:**

Studying the basic properties of differential equations with delay, especially in view of the stability of their solution. Application of acquired knowledge to specific dynamic systems describing real models. Numerical simulation of studied models in suitable mathematical software.

**Recommended bibliography:**

KOLMANOVSKII, V., MYSHKIS, A. Introduction to the Theory and Applications of Functional Differential Equations, Kluwer Academic Press Publishers, Dodrecht, 1992.

SMITH, H. An introduction to delay differential equations with applications to the life sciences, Springer New York, 2011.

STROGATZ, S. H. Nonlinear Dynamics and Chaos, 2nd ed. Westview Press, 2015.

Deadline for submission Master's Thesis is given by the Schedule of the Academic year 2020/21

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**Abstract**

This thesis is a review of Delay Differential Equations in Dynamical systems.Starting with a general overview of Delay Differential Equations, we present the concept on Delay Differentials and the application of its models,ranging from biology and population dynamics to physics and engineering.We will also give an overview on Dynamical systems and delay differential equations in the dynamic systems .An area for modelling with delay differentials equations is Epidemiology.Emphasis is given to the development of the Susceptible-Infected-Removed(SIR) epidemiological model without and with time delay.We the analyse our two models under equilibra and local stability using assumed data of COVID -19 .Results would be compared between the model without delays and model with delays.

**keywords**

Delay Differential equation, Dynamical systems,stability,Epidemic models

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I declare that I have worked on this thesis independently under a supervision of doc. Mgr. Zdeněk Opluštil, Ph.D. and using the sources listed in the bibliography.

Martha Dokyi

First, I would like to thank my supervisor , doc. Mgr. Zdeněk Opluštil, Ph.D. for his guidance,patience and time throughout this whole masters project in Brno University of Technology.
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May the good Lord,bless you all abundantly above,all that you wish and ask for.Amen.
Martha Dokyi

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# 1 INTRODUCTION

Delay Differential equations also called time delays has attracted much attention over 200 years to the field of nonlinear dynamics. It is used for analysis and prediction in various fields of biology,physics and engineering.

Time delays has its solutions depending on the past history of the system to predict the present .It is meaningless for a system not to have a history. Since most dynamic systems are governed by the principle of causality(the future state of the system is independent of the past but depends solely on the present to predict the future), it is important to include the history of the system if we want a realistic model of the system.
If the model does not depend on history then it generally consists of ordinary differential equations or partial differential equations.
Other model incorporating history generally include Retarded Functional differential equations(RFDEs) and Neutral Functional Differential equations(NFDEs).
Examples of differential equations with past dependence are Delayed Logistic Equation with a Discrete Delay, Delayed Logistics Equation with Distributed delay,Delayed Lotka-Volterra Predator-Prey Systems,Neutral Delay Logistic equation,Delay models in Physiology,Delayed Epidemic models amongst others. see [40]

Time delays can make unstable a stable equilibrium and can cause populations to fluctuate and this makes it more complicated than the ordinary differential equation.
In this thesis, we review a Delay Differential Equation model arising from analysing an epidemiological system.
Epidemiology aims at investigating diseases and state of health of a specific population in order to find solutions to health related problems of that population.
Time delay mathematical models are used to model the complex dynamics of the diseases in the population.

## 1.1 BACKGROUND

The study into delay differential equations has been ongoing for at least two centuries. This claim has been referenced to E. Schmitt (1911).
Some of the early work, like most fields of mathematics, started from geometry and number theory. The importance of considering hereditary effects in modelling physical systems was emphasized at the International Conference of Mathematicians by Picard (1908). Volterra (1931) in his book explained and outlined the role of hereditary effects on models for the interaction of species.

In the Soviet Union of the 1940s, more than in other parts of the world, this subject gained pace due to the consideration of meaningful models in engineering systems and control. At the time it was evident, especially to engineers, that physical systems had the occurrence of hereditary effects but there was little theory to explain their observations in these models.
In the last half century, there has been tremendous development in the theory of differential equations to the extent that it is now imbued in the vocabulary of researchers across multiple disciplines.

Dynamical systems theory also known as Chaos Theory comprises of methods for analysing differential equations and iterated mapping. A given deterministic dynamical system can be proven to have stable or unstable solutions but this does not necessarily mean that the phenomenon it describes behaves likewise; that is dependent on the mathematical model's quality.

More than half the research work on delay differential equations dealt primarily with linear differential equations and the preservation of stability of equilibra under small non-linear perturbations when the linearization was stable (or unstable) see [1].
Laplace transforms are naturally used for linear equations with constant coefficients. This resulted in the expansions of solutions in terms of eigenfunctions and convergence properties of these expansions.

Understanding the extent to which one could apply Lyapunov's second method (1891) was important to study the stability of equilibra. It could be said that the birth of the modern theory came forth from Lyapunov's second method see[1].
Poincaré's work on celestial mechanics(Poincaré, 1899) introduced Qualitative Theory of Dynamical systems.The methods he developed prepared the foundation for the local and global analysis of non-linear differential equations and many other concepts and theories.

With the periodically-disturbed pendulum, Poincare showed that mechanical systems with two or more degrees of freedom might have homoclinic orbits and hence might not be integrable.

G.D.Birkhoff (1927) showed that near any homoclinic point of a two-dimensional map,there is an infinite sequence of periodic orbits whose periods approach infinity. He also showed (Birkhoff 1932) annulus maps having orbits with different periods can possess complicated limit sets separating their domains of attraction.

The theory of dynamical systems does not address specific phenomena nor does it propose certain models of reality. Instead,it provides a non strict set of methods for analysing ODEs and iterated mappings. Its canonical problem have the form:

$$\dot{x}_j = f_j\left(x_1, x_2, \ldots, x_n; \mu_1, \ldots, \mu_k\right), \text{ or } x_j(l+1) = F_j\left(x_1(T), \ldots, x_n(T); \mu_1, \ldots, \mu_k\right) \quad (1.1)$$

where $x_1, \ldots, x_n$ are state variables and $\mu_1, \ldots, \mu_k$ are external control parameters, usually regarded as fixed for the purpose of solving (1.1) to obtain orbits $\mathbf{x}(t) = (x_1(t), x_2(t), \ldots, x_n(t))$ or $\{\mathbf{x}(l)\}_{l=0}^{\infty}$. see [14]

The solutions curves of the above equation (all orbits) are studied alongside the dependence of the set of solutions (i.e.phase portrait) on the parameters and the description of qualitative properties. These study are emphasized by this non-strict set of methods.

The qualitative theory of dynamical systems is a mathematical theory founded on analysis, geometry and topology.

Currently its reach has gone beyond the field of mathematical sciences. It is providing a unifying structure that classifies dynamical systems across a wide range of applications.

Delay differential equations share the quite many similarities with the general ODEs: existence, uniqueness, continuity of solutions and dependence of parameters. DDEs only go a step further to add some technicalities due to the infinite dimensional character of the problem.

## 1.2 SOME MODELS INCORPORATING TIME DELAYS

The following information was taken from see([40]).

### 1.2.1 Delayed Logistic Equation with a Discrete Delay

Generally, the simplest type of dependence of the past is that which only its state variable depends on the past, but the derivative of the state variable does not. This is the known retarded functional differential equation (RFDE) or the retarded difference differential equation (RDDE)

$$\dot{x}(t) = F(t, x(t), x(t-\tau)), \quad \dot{x} = \frac{dx}{dt} \tag{1.2}$$

The Wright's equation is a well-known special case of 1.2.

$$\dot{x}(t) = \gamma x(t)[1 - x(t-\tau)/K] \tag{1.3}$$

Equation 1.3 is frequently referred to as the delayed logistic equation with a discrete delay. It has known applications in probability methods in distribution of prime numbers see([40]).

For the study of population dynamics, only non-negative solutions of 1.3 are considered.

### 1.2.2 Delayed Logistic Equation with a Distributed Delay

In a population, the immunological resistance of its members to a parasite that lives its complete life cycle in a host, without causing harm to the host, is dependent on the exposure of the host to the population of the parasite. "Characteristically, the increase is exponential during early stages of infection when the host offers an ideal environment. Subsequently, when the host becomes resistant and represents a less suitable environment, the rate of increase declines to zero and the population then rapidly decreases." Michel (1969)

The following integro-differential equations is considered an appropriate model for the parasite population growth:

$$\frac{dN}{dt} = rN\left[1 - \frac{N}{K} - \int_0^t N(s)G(t-s)ds\right]. \tag{1.4}$$

Instantenous self-crowding term is followed by a pollution term. This is most suitable when the integral is taken from t = 0, the time the a host ingests or comes in contact with

the the parasites. The simplest memory function can then be easily adopted, $G(t) = k$, where k is a constant.

### 1.2.3 Delayed Epidemic Model

"Mathematical biologist A.J Lotka investigated, in a series of papers from 1912 on, a differential equation model of malarial epidemics due to Ross(1911). In particular, he examined the effects of incubation delays. " The equations as given by Lotka for human population:

$$\dot{h}(t) = bgm(t)(p - h(t))/p - Mh(t) - rh(t) \tag{1.5}$$

for the mosquito population, we have:

$$\dot{m}(t) = bfh(t)(q - m(t))/p - Nm(t) - sm(t) \tag{1.6}$$

the total number of humans is given by p, the total population of the mosquitoes is given by q. These two parameters are treated as constants. The function h(t) stands for human population carrying the malaria organism. m(t) stands for mosquito population carrying the malaria organism. p - h(t) and q - h(t) stand for the healthy population.

A fixed proportion of each of these populations is assumed to be infective, with the infective populations being $fh$ and $gm$, respectively. The quantities $M$ and $N$ are death rates, while $r$ and $s$ are recovery rates. It is assumed that each mosquito bites $b$ people in unit time, and that each person receives $a$ bites in unit time.

For our present purposes what is of most interest is the modification to include incubation delays, quoted from Ross (1911) to be $u = 0.5$ month in human and $v = 0.6$ month in mosquito. We thus have

$$\dot{h}(t) = bgm(t-u)[p - h(t-u)]/p - (M+r)h(t)$$
$$\dot{m}(t) = bfh(t-v)[q - m(t-v)]/p - (N+S)m(t)$$

The delay is from the time of a bite to the time at which the human or mosquito is infective.

# 2 DYNAMICAL SYSTEMS

Dynamical systems generally,is a rule which describes the evolution of a state of a mathematical model over time and it is given by systems of a differential equations.
Mathematical models based on real situations are non-linear which makes it quite difficult to solve. The systems has its current state depending fully on previous state.

The aim of dynamical systems is to understand the behavior of the chosen system,given rules for which the system evolves.
These rules help define the changes in the system,given the states of the physical problem.

The states are variables simply called the ***state variables*** and it is anything that can be represented with a number.
Examples of these state variables are;population of a colony, the amount of money in an account, temperature,density of a chemical in a solution, the position of a particle and so on.

Over the years Dynamical systems has been involved in the study of various mathematical models which is used in various fields like; physics,biology,economics,chemistry and so on.Example is, in the analysis of environmental problems, we have physical models used a quantitative tools which constitute dynamical systems.

A system measured in integer time values is called a Discrete Dynamical system (the state of the system evolves in discrete time steps) while a system with continuous measuring of time is referred to as Continuous Dynamical system whose system of evolution occurs smoothly over time.

In this thesis we focus on a continuous dynamical system, which measures time continuously and the system given by ordinary differential equations.
We restrict ourselves to autonomous systems of ordinary differential equations of the system in $\mathbb{R}^n$, that is for a system given as;

$$\mathbf{x}' = f(\mathbf{x})$$

where function $f : \mathbb{R}^n \to \mathbb{R}^n$ is a $C^1$ function and does not depend on the variable t.By $\mathrm{x}'$ it means the time derivative of $\mathrm{x}(\mathrm{x} = \mathrm{x}(t))$. The non autonomous systems are not considered here, as any non autonomous system $\mathbf{x}^{'} = f(\mathbf{x}, t)$ with $\mathbf{x} \in \mathbb{R}^n$ can be rewritten as autonomous with $\mathrm{x} \in \mathbb{R}^{n+1}$, by letting $x_{n+1} = t$.

## 2.1 DEFINITION OF DYNAMICAL SYSTEM

The following theory can be found in [16],[24],[35].

**Definition 2.1.** A smooth continuous dynamical system denotes a pair $\{\Omega, \phi\}$, where $\Omega$ is a state space and $\phi : \mathbb{R} \times \Omega \to \Omega$ is a continuously differentiable function ($\phi \in C^1(\Omega)$) satisfying

(i) $\phi_0(\mathbf{x}) = \mathbf{x}, \forall \mathbf{x} \in \Omega_1$
(ii) $\phi_{t+s}(\mathbf{x}) = \phi_{\mathrm{t}}\left(\phi_s(\mathbf{x})\right), \forall \mathbf{x} \in \Omega$ and $t, s \in \mathbb{R}$

The function $\phi$ is often called an evolution operator, where $\phi_t(\mathbf{x}) = \phi(t, \mathbf{x})$.

**Definition 2.2.** Let $x_0 \in \Omega$ be an initial state of a system. For a fixed time $t \in \mathbb{R}$ the evolution operator $\phi$ transforms $\mathbf{x}_0$ into some state $\mathbf{x}(t)$ at time t, i.e.

$$\mathbf{x}(t) = \phi_t(\mathbf{x}_0)$$

*Remark* 1. $\Omega$, the state space usually refers to $\mathbb{R}^n$, as that is what would be considered in this thesis.

**Definition 2.3.** Suppose an initial value problem of an autonomous system of ODEs

$$\mathbf{x}' = f(\mathbf{x})$$
$$\mathbf{x}(t_0) = \mathbf{x}_0$$

where $f : E \to \mathbb{R}^n, E$ is an open subset of $\mathbb{R}^n, f \in C^1(E)$ and $x_1 \in E$ is the initial value.
Then $\mathbf{x}(t)$ is a solution of the initial value problem $(2.1) - (2.2)$ on an interval I if $t_0 \in I$, $\mathrm{x}(t_0) = \mathrm{x}_0$ and $\mathrm{x}(t)$ is a solution of the system of ODEs (1.1) on the interval $I$.

*Remark* 2. We assume $f$ to be defined for all $\mathrm{x} \in \mathbb{R}^n$, i.e. $f : \mathbb{R}^n \to \mathbb{R}^n$.

**Theorem 2.1.** *(The Existence and Uniqueness Theorem): Consider the initial value problem* $(2.1) - (2.2)$*, where* $f : E \to \mathbb{R}^n, f \in C^1(E)$*. Then there exists an* $a > 0$ *such that the initial value problem has a unique solution* $\mathbf{x}(t)$ *on the interval* $[-a, a]$.

**Theorem 2.2.** *Theorem 2: Consider the initial value problem* $(2.1) - (2.2)$*, then for each* $\mathbf{x}_0 \in E$ *there is a maximal interval* $J = (\alpha, \beta)$ *on which the initial value problem has a unique solution* $\mathbf{x}(t)$

**Definition 2.4.** Let $E \subseteq \mathbb{R}^n$ and $f \in C^1(E)$. Let $\phi(t, x_0)$ be the solution of $(2.1) - (2.2)$ defined on its maximal interval $J(\mathrm{x}_0), \mathrm{x}_0 \in E$.
Then for $t \in J(\mathrm{x}_0)$, the family of evolution operators $\phi_t$ defined by

$$\phi_t(\mathbf{x}_0) = \phi(t, \mathbf{x}_0)$$

is called the flow of the system (2.1). $\phi_t$ is referred to as the flow of the vector field $f$.

**Definition 2.5.** Suppose the initial value $\mathrm{x}_0$ is fixed and $J = J(\mathrm{x}_0)$. Then the mapping $\phi(\cdot, \mathbf{x}_0) : J \to E$ defines a solution curve or a trajectory of the system (2.1) through the point $\mathrm{x}_0 \in E$.
The trajectory is visualized as a motion along a curve $\Gamma$ through the point $\mathrm{x}_0$. The arrow then indicates the orientation of the curve as time increases.

The phase portrait of the system (2.1) refers to the set of all solution curves of (2.1) for different initial points satisfying the initial value problem $(2.1) - (2.2)$ in the phase space. The solution curves in the phase space never intersect each other.

**Definition 2.6.** A point $\mathbf{x}^* \in \mathbf{E}$ is called equilibrium point (fixed point, critical point) of the system (2.1) if $f(\mathbf{x}^*) = 0$($\mathbf{0}$ means the zero vector ). Moreover, for any trajectory starting in $\mathbf{x}^*$, i.e. $\mathbf{x}(0) = \mathbf{x}^*$, is $\mathbf{x}(t) = \phi_t(\mathbf{x}^*) \equiv \mathbf{x}^*$ for any $t \in \mathbb{R}$

In general, trajectories of the solution $\mathbf{x}(t)$ can be divided into 3 main categories:

(i) Fixed point - the solution $\mathbf{x}(\mathbf{t})$ is constant, i.e. trajectory stays in the fixed point for all time.

(ii) Cycle, periodic orbit - the solution $\mathbf{x}(\mathbf{t})$ is periodic, i.e. the trajectory forms a closed curve and stays on this curve for all time.

(iii) Open curve - the trajectory is an injective map never intersecting itself.

## 2.2 Linear system

Suppose the system given in (2.1) is linear, i.e. function $f$ consists of linear terms only, $f : \mathbb{R}^n \to \mathbb{R}^n$. Then the system can be rewritten as

$$\mathbf{x}' = A\mathbf{x}$$

where $\mathbf{x} \in \mathbb{R}^n$, $A$ is an $n \times n$ matrix and the following theorem holds.

**Theorem 2.3.** *(The Fundamental Theorem for Linear Systems): Let $A$ be an $n \times n$ matrix. Then for a given $\mathrm{x}_0 \in \mathbb{R}^n$, the initial value problem $\mathbf{x}' = \mathbf{A}\mathrm{x}, \mathrm{x}(\mathbf{0}) = \mathrm{x}_\mathbf{0}$ has a unique solution for all $t \in \mathbb{R}$ given by*

$$\mathbf{x}(t) = e^{At}\mathbf{x}_0$$

## 2.3 Nonlinear system

On an interval $I$, a unique solution of an initial valued problem of a non-linear dynamical system exists according to Theorem 3 and in very few cases can these non-linear cases be solved analytically unlike in the linear cases.

Non-linear systems usually are made up of topological,geometrical and analytical techniques in the investigation of their behaviours. As part of non-linear system's analysis,numerical methods plays an important role.

**Linearization of nonlinear dynamical systems**

Nonlinear dynamical systems are investigated in the neighborhood of its equilibrium points.The local behaviour of the nonlinear system $\mathbf{x}' = \mathbf{f}(\mathbf{x})$ near a hyperbolic equilibrium point $\mathbf{x}^*$ is qualitatively determined by the behaviour of the linear system $\mathbf{x}' = \mathbf{A}(\mathrm{x})$, where $A$ is the Jacobian matrix evaluated at point $\mathbf{x}^*$.

*Remark* 3. The Jacobian matrix $J$ evaluated at a fixed point $\mathbf{x}^* \in \mathrm{R}^{\mathbf{n}}$ is given by $n \times n$ matrix

$$J = Df\left(\mathbf{x}^*\right) = \begin{pmatrix} \frac{f_i(\mathbf{x}^*)}{\partial x_1} & \cdots & \frac{L_1(x^*)}{\partial x_*} \\ \vdots & \ddots & \vdots \\ \frac{\ln(\mathbf{x}^*)}{\partial x_1} & \cdots & \frac{f_n(x^*)}{\partial x_x} \end{pmatrix}$$

The eigenvalues $\lambda$ of the Jacobian matrix can be computed as the roots of characteristic polynomial

$$P(\lambda) = \det(J - \lambda I)$$

where I represents the identity matrix.

**Definition 2.7.** An equilibrium point $\mathbf{x}^*$ of the system (1.1) is called hyperbolic if none of the eigenvalues of the Jacobian matrix $J = Df(\mathbf{x}^*)$ has zero real part. Otherwise, the equilibrium point is called non-hyperbolic.

If the fixed point $\mathbf{x}^*$ is hyperbolic, then according to Hartman-Grobman Theorem [1] there exists a neighborhood of this point, in which the nonlinear system $\mathrm{x}' = \mathbf{f}(\mathrm{x})$ is topologically conjugate to the system $\mathbf{x}' = \mathbf{A}(\mathrm{x})$, where $A$ is the linearization matrix, i.e. $A = Df\left(\mathbf{x}^*\right)$.

## 2.4 **Stability** :

Stability of s system is when a small perturbation of initial data yields a small change in the solution. Deviation of the solution caused by a perturbation of initial data which disappears as $t \to \infty$ makes the solution attractive or asymptotically stable.
In dynamical systems, it is more common to refer to a stability of equilibrium points of the given system.

**Definition 2.8.** Let $\phi_t$ denotes the flow of the system (2.1) defined for all $t \in \mathbb{R}$. An equilibrium point $\mathrm{x}^*$ is (locally) stable if for all $\varepsilon > 0$ there exists a $\delta > 0$ such that for all $\mathbf{x} \in \mathbf{N}_\delta\left(\mathrm{x}^*\right)$ and $t \geq 0$ then

$$\phi_t(\mathbf{x}) \in N_\delta\left(\mathbf{x}^*\right)$$

Furthermore, $\mathbf{x}^*$ is (locally) asymptotically stable if it is stable and if there exists a $\delta > 0$ such that for all $\mathbf{x} \in N_\delta(\mathbf{x}^*)$

$$\lim_{t\to\infty} \phi_t(\mathbf{x}) = \mathbf{x}^*$$

The equilibrium point is said to be unstable if it is not stable.

*Remark* 4. The equilibrium point's stability is determined by the sign of real parts of the eigenvalues $\lambda$ of the Jacobian matrix.

**The following theorem holds** :

**Theorem 2.4.** *Let $J = Df(\mathbf{x}^*)$ be the Jacobian matrix for the system (1.1) evaluated at a fixed point* $\mathrm{x}^*$ *and let $\lambda_i$ be its eigenvalues.*

*(i) If $\Re(\lambda_i) < 0$ for all $\lambda_i$, then the fixed point $\mathbf{x}^*$ is asymptotically stable.*

*(ii) If $\Re(\lambda_j) > 0$ for at least one $\lambda_i$, then the fixed point $\mathbf{x}^*$ is unstable.*

*(iii) If $R(\lambda_i) = 0$ for at least one $\lambda_i$, then the fired point $\mathbf{x}^*$ is non-hyperbolic and its stability cannot be determined by the linearization method.*

Classification of basic fixed points can be found in the literature. For further investigation of non-hyperbolic points, it is possible to use other methods which can help to determine their stability. The stability according to Lyapunov is defined as follows:

**Theorem 2.5.** *(Lyapunov Function): Suppose the nonlinear system (2.1) with an equilibrium point $\mathbf{x}^*, \mathbf{x}^* \in \mathbf{E}$, where $E$ is an open subset in* $\mathrm{R}^n$. *Now, suppose that there exists a function $V : E \to \mathbb{R}^n$ satisfying*

*(i) $V(\mathbf{x}^*) = 0$*

*(ii) $V(\mathbf{x}) > 0$ if $\mathbf{x} \neq \mathbf{x}^*$.*

*Then*

*(i) if $\dot{V}(\mathbf{x}) \leq \mathbf{0}$ for $\forall \mathrm{x} \in E, \mathbf{x}^*$ is stable.*

*(ii) if $\dot{V}(\mathbf{x}) < \mathbf{0}$ for $\forall \mathbf{x}^* \in \mathbf{E} \backslash \{\mathbf{x}^*\}, \mathbf{x}^*$ is asymptotically stable.*

*(iii) if $\bar{V}(\mathbf{x}) > \mathbf{0}$ for $\forall \mathrm{x} \in E \backslash \{\mathbf{x}^*\}, \mathbf{x}^*$ is unstable.*

*The function $V$ is called the Lyapunov function. The term $\dot{V}(\mathbf{x}) = \mathbf{DV}(\mathbf{x})\mathbf{f}(\mathbf{x})$, where $DV = \left(\frac{\partial V}{\partial x_1}, \ldots, \frac{\partial V}{\partial x_n}\right)$ (see [16][24][35]).*

# 3 DELAY DIFFERENTIAL EQUATIONS

## 3.1 INTRODUCTION

Delay differential equations(time delays) or simply, the system of differential equations with time lags are equations whose solutions depends on the history of a system.
The solution depends on time,$t = t_0$. Time delays can be grouped under Neutral or Retarded and Continuous or Discrete.
Delay differential equations over many years have been used in many fields in applied mathematics for example study of epidemics,automation,predator system analysis, other areas in engineering and biology.

## 3.2 DELAY DIFFERENTIAL EQUATION (DDE)

Let us consider the non-autonomous system of delay differential equations

$$\mathbf{x}'(t) = f(t, \mathbf{x}_t),\ t \geq t_0, \tag{3.1}$$

where $\mathbf{x} \in \mathbb{R}^n$, $t_0$ is the initial time, $f$ is a continuous function $f\colon \mathbb{R} \times C \to \mathbb{R}^n$, $C = C([-r, 0], \mathbb{R}^n)$ is usually called the state of the dynamical system at time $t$ and $\mathbf{x}_t \in C$ is defined by

$$\mathbf{x}_t(\theta) := \mathbf{x}(t + \theta),\ -r \leq \theta \leq 0.$$

A *solution* of the system (3.1) on the interval $[t_0, t_1)$ is a continuous function $\mathbf{x}\colon [t_0 - r, t_1) \to \mathbb{R}^n$ which satisfies (3.1) on $[t_0, t_1)$ for some $t_1 > t_0$.

Initial condition for the system (3.1) is given by

$$\mathbf{x}_{t_0} = \phi \tag{3.2}$$

where $\phi \in C([-r, 0], \mathbb{R}^n)$ is the state of the system at time $t_0$, i.e.

$$\mathbf{x}(t_0 + \theta) = \phi(\theta),\ -r \leq \theta \leq 0$$

Given $t_0$ and $\phi \in C([-r, 0], \mathbb{R}^n)$, we say $\mathbf{x}(t)$ is *solution* of the initial value problem (3.1)– (3.3) if it is a solution of the system (3.1) on $[t_0, t_1)$ and satisfies condition (3.2).

Analogous to the ordinary case, we can formulate a theorem on existence and uniqueness for the systems with delay (see[13][40]).

**Theorem 3.1.** *Let $D \subseteq \mathbb{R} \times C$ be an open set and suppose that $f\colon D \to \mathbb{R}^n$ be continuous and $f(t, \varphi)$ be Lipschitzian with respect to the second variable in every compact subset of $D$. If $(t_0, \phi) \in D$, then the initial value problem* (3.1)– (3.2) *has a unique solution on $[t_0 - r, t_0 + a]$ for some $a > 0$.*

## 3.3 LOCAL STABILITY OF DDE

Let us consider an autonomous non-linear system

$$\mathbf{x}'(t) = f(\mathbf{x}_t), \quad t \in \mathbb{R} \tag{3.3}$$

and $\mathbf{x}^* \in \mathbb{R}^n$ be an equilibrium point of the system (3.3), i.e. $f(\mathbf{x}^*) = \mathbf{0}$.

**Definition 3.1.** The equilibrium $\mathbf{x}^*$ of the system (3.3) is *(locally) stable* if for any $\varepsilon > 0$, there exists $\delta = \delta(t_0, \varepsilon)$ such that $||\mathbf{x}^* - \mathbf{x}(t_0, \psi)||_\infty < \varepsilon$ for any $\psi \in C$ satisfying $||\phi - \psi||_{\mathrm{C}} < \delta$.
The equilibrium $\mathbf{x}^*$ of the system (3.3) is *asymptotically stable* if it is stable and if there exists $\delta = \delta(t_0) > 0$ such that $||\mathbf{x}^* - \mathbf{x}(t_0, \psi)||_\infty \to 0$ for any $\psi \in C$ satisfying $||\phi - \psi||_{\mathrm{C}} < \delta$, where $||\cdot||_\infty$ and $||\cdot||_{\mathrm{C}}$ are usual norms.
The equilibrium $\mathbf{x}^*$ of the system (3.3) is *unstable* if it is not stable.

Investigating the local stability of a delayed systems is similar to those without delay. A linearized system to (3.3) is also studied. For a single delay $\tau$ at equilibrium $\mathbf{x}^*$, the linearized system of (3.3) has the form

$$\mathbf{x}'(t) = A_0\mathbf{x} + A_1\mathbf{x}(t - \tau), \tag{3.4}$$

where $A_0 = Df(\mathbf{x}^*)$ is $n \times n$ is the Jacobian matrix and $A_1$ is the Jacobian matrix with respect to $\mathbf{x}(t - \tau)$ evaluated at an equilibrium point $\mathbf{x}^*$. The characteristic equation of the system (3.4) is not polynomial but takes the form

$$\det\left(A_0 + A_1 \mathrm{e}^{-\lambda\tau} - \lambda E\right) = 0, \tag{3.5}$$

where $E$ represents $n \times n$ identity matrix. Let $\Lambda$ be the set of all roots of the characteristic equation (3.5). Then the following theorem holds (see [13]).

**Theorem 3.2.** *Let $\Re(\lambda_i) < 0$ for all $\lambda_i \in \Lambda$. Then the equilibrium point $\mathbf{x}^*$ of the system (3.3) is asymptotically stable. If $\Re(\bar{\lambda}) > 0$ for some characteristic root $\bar{\lambda} \in \Lambda$, then $\mathbf{x}^*$ is unstable.*

# 4 EPIDEMIC MODELS

## 4.1 INTRODUCTION

In this chapter, we expatiate on Epidemic dynamics and epidemic models,which are mostly based on compartment structures.Researches on communicable diseases or infectious diseases can be classified under descriptive,analytic,experimental and theoretic.

When it comes to the epidemic dynamics, the study is an important approach to investigate the transmission dynamics of an infectious disease. We formulate mathematical models to analyze the transmission dynamics of these infectious diseases.

These models are based on the population dynamics,behavior of the disease transmission,features of the infectious agents and the connections with other social and psychological factors.

Epidemic dynamic models were created under the assumption that the specific population under study can be divided into compartments.The compartmental model was proposed by W.O. Kermack and A.G.McKendrick in the years 1927,1932 and 1933.It was then developed over the years by other biomathematicians.

The Kermack-McKendrick model(KM model) was based on relatively simple assumptions like the rate of flow from one compartment to the other and uses the latency period of the disease .It also used the general mode of transmission of the infectious disease (see[43][20][4]).

Using epidemic dynamical models, we discover general principles governing the transmission dynamics of the disease and identify important parameters to provide useful prevention and control strategies of the disease.

## 4.2 EPIDEMIC MODELS

Diseases especially communicable diseases, from time immemorial have been an important part of the human history.
Worst case scenarios of diseases are pandemics.This is when the infectious disease spreads from one border to other borders of countries.
Epidemics have invaded many populations, causing many deaths before dying out or reoccurring in the future.On the economic growth, it causes economic damages like short-term fiscal shocks and long -term negative shocks.

In the 14th century, about a third of Europe's population was wiped out by a bubonic plague called the Black Death. It also raged through Asia and Africa .The Black Death is said to have claim 75 to 200 million lives between 1346 and 1350.

In 1918, about 50 million people died from the Spanish flu. Against popular opinion, it derived its name from the place it was first identified: Spain. The period of discovery was 1918, towards of the First World War. The powers of the world at the time were more committed to warfare than they were to epidemics. As a result of this diverted attention,

more deaths were recorded.

The Human Immunodeficiency Virus (HIV) responsible for causing Acquired Immunodeficiency Syndrome (AIDS) was discovered in 1980. It has since then claimed about 38 million lives. In 2008, The United Nations estimated that there were 14 million AIDS orphans and the number would go up by more than 80 percent in 2010.

In December 2019, a novel disease, the Coronavirus disease, was discovered in Wuhan China. Since that time up to this moment, the world has registered 3,283,422 deaths. see([8])

It is then obvious that to prevent and control infectious diseases more effectively, it is important to fully understand the mechanism of the spread and the transmission dynamics of the disease and then provide useful predictions and guidance so that better strategies can be established. Quantitative and qualitative analysis, sensitivity analysis and numeric simulations make a mathematical model give us a good understanding of how infectious diseases spread. Equipped with this, we can make reliable predictions and obtain useful information on how to prevent and control the spread of these infectious diseases.
Epidemic models are not exactly novel; they can be traced back to the time of Bernoulli. In 1760, he used a mathematical model to study the rate of spread of smallpox. Proper research into the field of using mathematical models to study infectious diseases did not kick off until the 20th century. It was Hamer , in 1906, that came up with a discrete time model to study the spread of measles. The physician, Dr. Ross, used differential equation models to describe the transmission of malaria between the vectors and hosts in 1911. "He determined that there is a threshold of the size of mosquitoes below which the spread of malaria can be controlled."

Between 1927 and 1933, W.O. Kermack and A.G. McKendrick formulated the SIR compartmental model (Susceptible-Infected-Recovered). This model was used to study the outbreak of Black death in London, that broke out in 1665 and ravaged on till 1666. They also formulated the SIS compartment model. This model introduced the concept of thresholds; the determiner of whether a disease spreads in a population. This concept established the fundamental of the theory of epidemic dynamics see([20][4]).

Mathematical models can be categorized as linear, nonlinear, autonomous, non-autonomous based on the described diseases, population and the environment.

A deterministic mathematical model is a mathematical model which does not allow for randomness. It is necessary that the results (output) does not change for given initial and final states. Analysis of deterministic models have been focused on the wellposedness of the models and their solutions, persistence of the diseases, stability of their steady states and the existence. These let us know whether the disease is pestilent or being endemic. The existence of periodic solutions describe the oscillatory movement of disease transmissions and occurrence of bifurcation and chaotic behaviour.

## 4.3 SOME TYPES OF EPIDEMIC MODELS

Epidemic models can be broadly classified into two:

- Stochastic models
- Deterministic models

**Stochastic models**: these rely on chance variation in risk exposure, and this gives better insight into an individual level modelling. It incorporates large amount of complexity and heterogeneity making it more insightful for monitoring. Parameters in a stochastic model gives different outputs

**Deterministic models**:also known as compartmental models, depend less on high quality data. They are easier to set up as opposed to stochastic models. Under the same initial and final conditions, the same behaviour is expected in the population.The best way to model real life problems is by the use of Deterministic models.Here, the specific population under observation is divided into compartments which constitutes different levels of an epidemic.Transitions from one compartment to the other are illustrated in differential equations with each compartment being differentiable with respect to time see([5]).

Some types of deterministic models are:

- SIR model (Susceptible-Infected-Removed)
- SIS model (Susceptible-Infected-Susceptible)
- SIRS model (Susceptible-Infected-Removed-Susceptible)
- SEIR model (Susceptible-Exposed-Infected-Recovered)

**SIS Model**

This model is made up of two compartments: the Susceptible compartment and the Infected compartment. In this model, the infected members of the population become susceptible to the disease after they have recovered from their infection. This is because the disease gives no immunity against reinfection.

Examples of these diseases are Influenza,chickenpox and measles.Hence there is a probability individuals who have suffered from the disease before would suffer from that same disease in the near future.

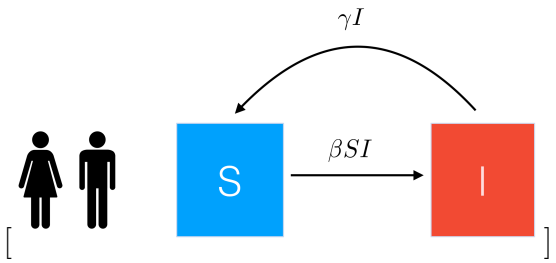



Figure 1: Image of the Susceptible-Infected-Susceptible model

**SIRS Model**

This model is made up of three compartments: the Susceptible compartment, the Infected

compartment and the Removed Compartment. In this model, transmission of individuals from the infected compartment to the removed compartment means either they are recovered or they are dead. Recovered individuals are not susceptible to the same disease until they lose their immunity.

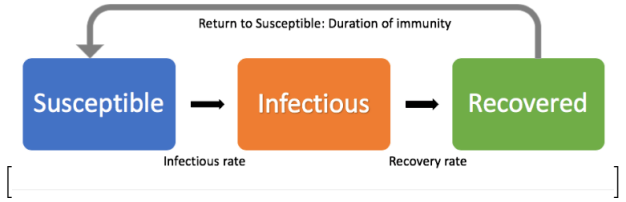



Figure 2: Image of the Susceptible-Infected-Removed-Susceptible model

**SEIR Model**

The SEIR model has four compartments: Susceptible, Exposed, Infected, and Recovered compartments. The transmission of individuals from the susceptible to the exposed compartment entails that individuals enter a latent period where the disease is contagious. This means they carry the infection but cannot transmit it.

After a period of time, there is a transmission of these individuals from the exposed to

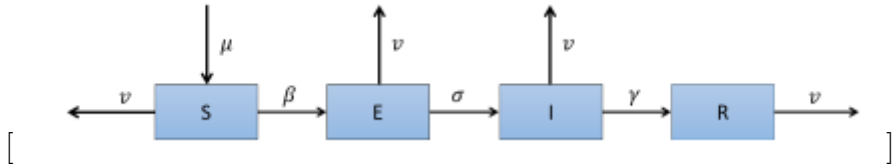



Figure 3: Image of the Susceptible-Infected-Removed model

the infected compartment. They finally get recovered: they either get healed or they die.

**SEIRS Model**

The SEIR model has five compartments. Like in SEIR,transmission of individuals is from the susceptible to the exposed compartment.Individuals enter a latent period where the disease is contagious. This means they carry the infection but cannot transmit it.After leaving the removed compartment, they move back into the susceptible state when they lose their immunity

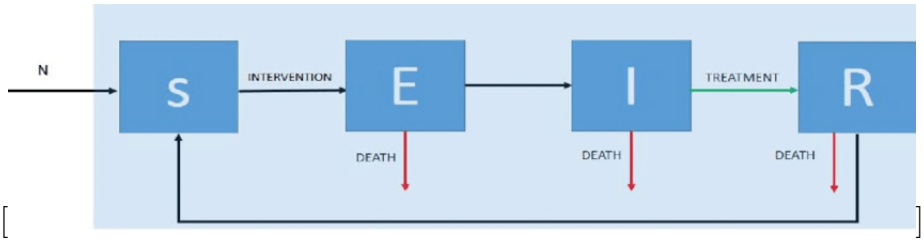



Figure 4: Image of the Susceptible-Exposed-Infected-Removed-Susceptible model

### 4.3.1 BASIC OF A COMPARTMENTAL MODEL

- Vital dynamic factor such as natural death and natural birth are neglected in the model
- the latent period of the model
- the population under observation is considered to be of constant size in epidemic periods.Hence, we assume that natural birth rate and death rate equal.
- The flow transmission of the compartmental model
  - Models with vertical transmission flow has most disease origins from natural birth.Example is HIV/AIDS
  - Models without vertical transmission means that the disease was not from birth and everyone no matter their age can get infected.Example is SARs-COV 2

### 4.3.2 BASIC REPRODUCTIVE NUMBER ($\mathcal{R}_0$)

In the study of disease modelling , the basic reproduction number, $\mathcal{R}_0$ plays a very important role. Its value tells one if the specific population under observation is at risk or not.

The basic reproduction number is defined as the average number of secondary infections produced by the primary infection into the total susceptible population.An infected person infects others at a rate of $\beta$ during an expected infection period of $\frac{1}{\alpha}$ see[17].

### 4.3.3 FACTORS AFFECTING BASIC REPRODUCTIVE NUMBER

- Rate of contact in the specific population under observation
- Duration of infection and
- Probability of transmission per contact

Being a dimensionless parameter, it determines the threshold condition for a Disease-Free Equilibrium. When $\mathcal{R}_0 < 1$, the Disease-Free Equilibrium is said to be locally asymptotically stable This means the disease cannot invade the population and will die out.This depends on how small $\mathcal{R}_0$ is .

When $\mathcal{R}_0 > 1$, the disease is difficult to contain and the Disease -free Equilibrium is unstable but the we get an Endemic Equilibrium.At this point, the value of $\mathcal{R}_0$ is very large see ([43][17]).

For example, if $\mathcal{R}_0 > 1$ for influenza cases in a specific hostel in a university, then that management of that hostel should expect more cases more cases of susceptible students infected with influenza.But when $\mathcal{R}_0 < 1$, the management of the hostel is assured that the influenza disease would die out. They can then know steps to take to curb the situation.

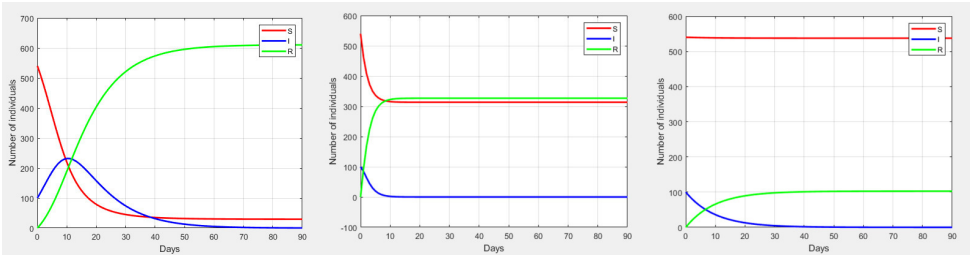



Figure 5: Figure:Above is an image of Epidemic curves of an SIR model with different basic reproductive numbers,$\mathcal{R}_0 > 1$ ,$\mathcal{R}_0 = 1$ and $\mathcal{R}_0 < 1$ respectively .

The disease becomes difficult to contain at $\mathcal{R}_0 > 1$. This mostly occurs at the beginning of the epidemic when it invades the susceptible population.$\mathcal{R}_0 > 1$ implies that new infections caused by an infected individual is greater than one and leads to the continuous growth of the infection.At this point the Disease-Free Equilibrium is unstable

When $\mathcal{R}_0 = 1$, the disease becomes endemic. It is seen on the second plot that the infection rate reduces as compared to when $\mathcal{R}_0 > 1$.$\mathcal{R}_0 = 1$ determines if a disease would persist or die out.

At $\mathcal{R}_0 < 1$ the disease dies out and infection rate decreases monotonically to zero.Infections caused by an infected individual become less than one and this shows that,we have a stable Disease-Free Equilibrium.

Epidemic mathematical models were created with the assumption that the population under study can be divided into compartments. They capture the dynamics of an infection which gives an individual permanent immunity after they have recovered.Examples of these diseases are typhoid fever,measles,small pox amongst others see([28]).

The compartmental model proposed by Kermack and McKendrick was based on that assumption of the rate of flow from one compartment to the other. It uses the latency period or incubation period of the disease, represented by time delay($\tau$) in the model, and the general mode of transmission during the spread of the communicable disease.

This model had the specific population under observation divided into compartments namely: the Susceptible compartment, the Infected Compartment and the Removed or Resisted compartment see([20]).

## 4.4 SUSCEPTIBLE-INFECTED-REMOVED(SIR)MODELS

The SIR model is in 3 compartments.The Susceptible compartment,Infected compartment and Removed compartment.

An assumption of the SIR model is that there is a homogeneous mixing of the Infected (I) and Susceptible(S) populations and that the total population (N) is constant in time.

The Susceptible population decreases monotonically towards zero in the model while the population of infected people increases.
And as the hosts transition from the Infected compartment to the Removed compartment,the population in the Infected compartment reduces as the population in the Removed increases

The SIR model simply implies that:

- Susceptible:during a pandemic, all individuals in this compartment stand the risk of contacting the disease or remain infected over a period of time (t).This includes passively immune individuals,once the loose their immunity . The Susceptible population would decrease as the virus spreads from one person to the other(one source to the other).

  In an SIR model with no vertical transmission,individuals,no matter the gender, age and size of the person, they are susceptible to getting infected.This compartment is denoted by *S(t)* at time $t$

- Infected: Every Individual in this compartment has been infected by the disease .The level of parasite is sufficiently large within the host and they can transmit to others in the Susceptible compartment. These people either recover of die.This compartment is denoted by *I(t)* at time $t$ see[28].

- Removed:Individuals in this compartment are either healed or died from the disease.Removed compartment is denoted by *R(t)* at time $t$

### 4.4.1 SOME BASIC ASSUMPTIONS OF THE SIR MODEL

- Specific population under observation is a closed environment.It is assumed that during this period there is no migration nor emigration into this population.
  There is neither a natural death or birth in this population, so the total population, $S(t) + I(t) + R(t) \equiv N$

- There is a homogeneous mix of the Susceptible population and the Infected population.

- The number of Susceptible individuals who get infected by an individual in the Infected compartment per unit of time, at time t, is proportional to the total number of Susceptible individuals with the proportional coefficient (transmission coefficient) , so that the total number of newly infected at time t, is S(t)I(t).

- The number of Removed individuals from the infected compartment per unit time is $\alpha I(t)$ at time t, where $\alpha$ is the rate of recovery Recovered individuals gain permanent immunity.

- All infections are assumed to end with Removed Compartment.

- Infection does not depend on age,gender nor social status

- Recovery rate $\alpha$ of individuals in the Infected region to the Removed or Recovered region is constant with time.

- The dynamical equations are of first order see([43]):

$$\begin{aligned} \frac{dS}{dt} &= - \text{ New infection rate} \\ \frac{dI}{dt} &= \text{ New infection rate - Recovery rate} \\ \frac{dR}{dt} &= \text{ Recovery rate} \end{aligned}$$

### 4.4.2 TIME DELAY IN SIR MODEL

The Susceptible individual, after coming in contact with an infected person, does not immediately transition into the Infected state.There is a latent period of incubation, which takes a period of about 5 to 14 days.
This is from the time of exposure to the virus till symptoms onset.After this period,the susceptible individual becomes infectious hosts to other susceptible people.

About 97% of people in the populations shows symptoms of SARS-COV2 within 11.5 days from the time of exposure.

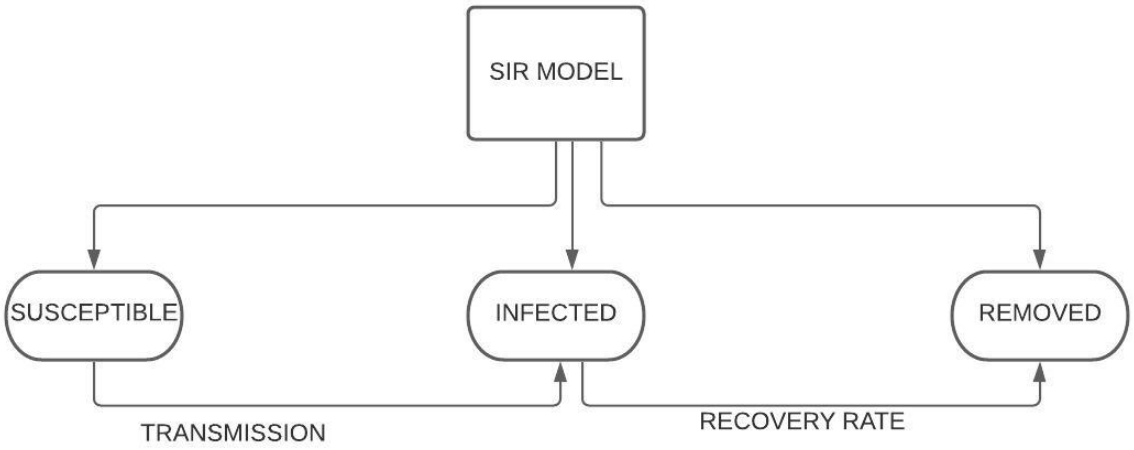
Inculcating time delays in epidemic models bring to bear the fact that the transmission dynamic behavior of the disease at time t depends not on only the present state of the time t but also on the state of the previous time.

We have have two types of time model:

- Discrete delay: in this time delay the dynamic behavior of time $t$ depends on the state of the previous time at $t - \tau$, where $\tau$ is a fixed constant. In this thesis, $\tau$ is the latent period (see[20]).
- Continuous delay: this delay depends on the whole period before the time t.

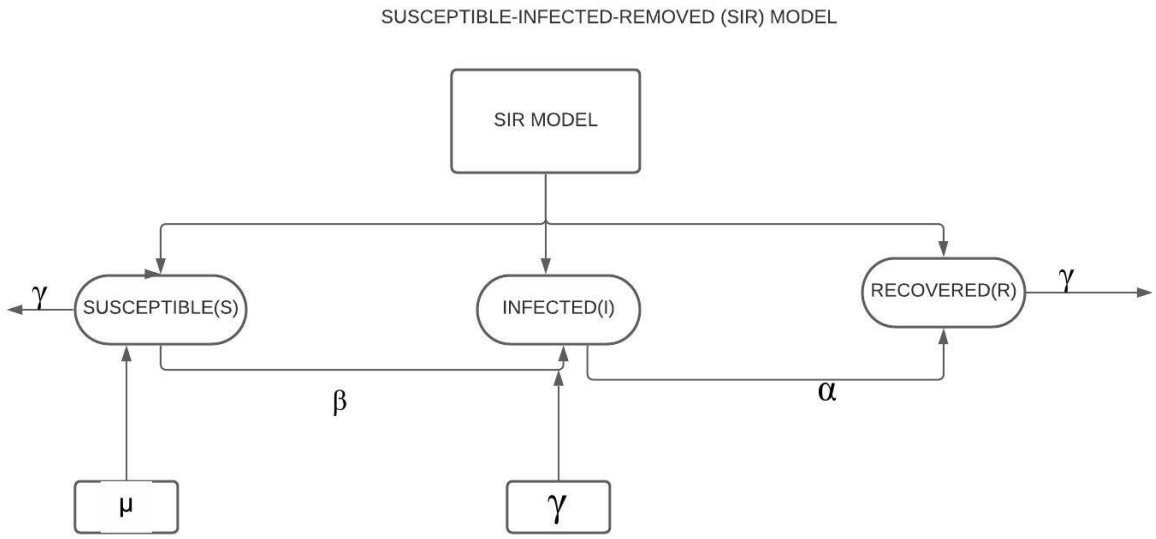
When there is time-delay in a mathematical model, periodic solutions may occur for various time delays.

### 4.4.3 THE SIR MODEL




In vital demographical statistics we have natural deaths $\gamma$ and natural rates of births $\mu$. Deaths caused by coronavirus cannot be considered as a natural death and so it is excluded ($\gamma = \mu = 0$ ).

For transmission from the susceptible compartment to the infected compartment, we represent by $\beta$ (is the average number of contacts per infected individual per day).$\alpha$ represents the rate of outflow into the removed compartment from the infected compartment.




The total population is represented as $N = S(t) + I(t) + R(t)$

We derive three non-linear ordinary differential equations from the compartments.

## 4.5 GENERALIZED SYSTEM

$\frac{dS}{dt} = \mu\ N - \beta S(t)I(t) - \gamma_1\ S(t)$

$\frac{dI}{dt} = \beta\ S(t)\ I(t) - (\gamma_2\ +\ \alpha)\ I(t)$

$\frac{dR}{dt} = \alpha\ I(t) - \gamma_1\ R(t)$

During the pandemic it was observed that natural deaths and births are very low and so they counterbalance each other, making $\mu$=$\gamma$=0
The SIR model is now simplified to the form

$\frac{dS}{dt} = -\beta\ S(t)\ I(t)$

$\frac{dI}{dt} = \beta\ S(t)\ I(t) - (\alpha)\ I(t)$

$\frac{dR}{dt} = \alpha\ I(t)$

As time goes the infection rate of the virus changes and the record of infected persons in the beginning of the pandemic would vary from the number of infected persons in the present.

There is also a behavioural change of the Susceptible individuals and the crowding effect of the infective individuals due to introduction of safety measures and this prevents the unboundedness of the contact rate.

$\beta\ S\ I$ is the bi-linear incidence rate which is suitable for communicable diseases such as influenza but not for sexually transmitted diseases. It measures the infection force of the disease.

The model above has no time delays which means it did not include the

- the latent state of the infection in the vector and
- latent state of the infection in an infected host.

There is a need for time to elapse before the host or the vector reaches a sufficiently high level to transmit from one person to the other.
Considering the above description we have our model with time delays to be as follows:

## 4.6 SIR MODEL WITH DELAY

$\frac{dS}{dt} = -\beta\ S(t)\ I\ (t-\tau)$

$\frac{dI}{dt} = \beta\ S(t)\ I(t-\tau) - (\alpha)\ I(t)$

$\frac{dR}{dt} = \alpha\ I(t)$

where $\tau > 0$ is the time delay incorporated in the model to represents the latent period or incubation period of the disease.

## 4.7 GENERALISED SYSTEM WITH DELAYS

$\frac{dS}{dt} = \mu\ N - \beta\ S(t)\ I(t-\tau) - \gamma_1\ S(t)$

$\frac{dI}{dt} = \beta\ S(t)\ I(t-\tau) - (\gamma_2\ +\ \alpha)\ I(t)$

$\frac{dR}{dt} = \alpha\ I(t) - \gamma_1\ R(t)$

where $S(0) > 0, I(0) > 0, R(0) > 0$ and parameter $\mu, \beta, \gamma, \alpha$ are of positive values.$S(0)+ I(0) + R(0) = N(t)$.

| Parameters | Definitions |
|---|---|
| $\mu$ | Natural birth |
| $\gamma$ | Natural death |
| $\alpha$ | Rate of outflow from the Infectious compartment to the Removed compartment |
| $\beta$ | Rate of outflow from the Susceptible compartment to the Infected Compartment |

# 5 Simulation of results

In this section we find and discuss on the stability and equilibrium of the system with and without time delays.We then use assume values to show the population dynamics in a our model with delay and without delays.

### 5.0.1 Generalized SIR model without delay

$$\begin{aligned} S' &= \mu N - \beta S(t)I(t) - \gamma_1 S(t) \\ I' &= \beta S(t)I(t) - (\gamma_2 + \alpha)\, I(t) \\ R' &= \alpha I(t) - \gamma_1 R(t) \end{aligned}$$

Finding the equilibria of the above system we equate the left side of the equation to 0. This forms the steady-state of the model.Solving the steady -state equations gives us the equilibria of the system.We denote equilibrium of the generalized system as follows: $E[S^*, I^*, R^*]$

To find $E[S^*, I^*, R^*]$ we have to solve systems

$$\begin{aligned} 0 &= \mu N - \beta S^* I^* - \gamma_1 S^* \\ 0 &= \beta S^* I^* - (\gamma_2 + \alpha)\, I^* \\ 0 &= \alpha I^* - \gamma_1 R^* \end{aligned}$$

Introducing the basic reproductive number, denoted by $\mathcal{R}_0 = \frac{\mu\, N\, \beta}{\gamma_1(\gamma_2+\alpha)}$. The equilibria of the generalized system without delay is given as:

- If $\mathcal{R}_0 < 1$ then generalized system is always equilibrium of $E_1[\frac{\mu\, N}{\gamma_1}, 0, 0]$ and it is locally stable
- If $\mathcal{R}_0 > 1$ then the generalized has two equilibria given as $E_1[\frac{\mu\, N}{\gamma_1}, 0, 0]$, which is unstable and $E_2[\frac{\gamma_2+\alpha}{\beta}; \frac{\mu\, N\, \beta - \gamma_1(\gamma_2+\alpha)}{(\gamma_2+\alpha)\beta};\ \frac{\alpha(\mu\, N\beta - \gamma_1(\gamma_2+\alpha))}{\beta\, \gamma_1(\gamma_2+\alpha)}]$ which is locally asymptotically stable.

We will show in details the local stability of these equilibrium in the following section

### 5.0.2 LOCAL STABILITY OF EQUILIBRIUM OF GENERALIZED SIR MODEL WITHOUT DELAY

We are interested in the sign of the real part of the characteristic equation of the linearized system, when finding the local stability of the equilibrium.

Our linearized system at equilibrium, for $x_0 \in \mathbb{R}_n$ is given as $(\mathbf{x}\prime) = \mathbb{A}\mathbf{x}$ where $\mathbb{A}$ is the Jacobi matrix $Df(x_0)$ .

The Jacobi matrix of the generalized SIR model is given as:

$$A = \begin{pmatrix} -\beta I - \gamma_1 & -\beta S & 0 \\ \beta I & \beta S - (\gamma_2 + \alpha) & 0 \\ 0 & \alpha & -\gamma_1 \end{pmatrix}$$

Then the CHARACTERISTIC EQUATION is $\det(A - \lambda E) = 0$, at equilibrium $E[S^*, I^*, R^*]$.

$$0 = \det \begin{pmatrix} -\beta I^* - \gamma_1 - \lambda & -\beta S^* & 0 \\ \beta I^* & \beta S^* - (\gamma_2 + \alpha) - \lambda & 0 \\ 0 & \alpha & -\gamma_1 - \lambda \end{pmatrix}$$

This implies that, $-(\lambda + \gamma_1)(\lambda^2 + \lambda(k_1 + e_1) + (k_0 + e_0)) = 0$.
The roots of the characteristic equation are:

- $\lambda = -\gamma < 0$
- and $(\lambda^2 + \lambda(k_1 + e_1) + (k_0 + e_0) = 0$

Local stability then depends on the equation

$$\lambda^2 + \lambda(k_1 + e_1) + (k_0 + e_0) = 0 \tag{5.1}$$

where

$$\begin{aligned} k_1 &= (\gamma_2 + \alpha) + \beta I^* + \gamma_1 \\ k_0 &= (\beta I^* + \gamma_1)(\gamma_2 + \alpha) \\ e_1 &= -\beta S^* \\ e_0 &= -\beta S^* \gamma_1 \end{aligned}$$

Equation 5.1 is a second degree transcendental polynomial at $\tau = 0$ with $k_1, k_0, e_1, e_0$ being real numbers. The steady state is asymptotically stable if the roots of the characteristic equation has negative real parts (see[29]).

The roots have negative real parts if and only if ;

- $k_1 + e_1 > 0$ and
- $k_0 + e_0 > 0$

We see that:

1. $k_1 + e_1 = \gamma_2 + \alpha + \beta I^* + \gamma_1 + (-\beta S^*)$ and

2. $k_0 + e_0 = (\beta I^* + \gamma_1)(\gamma_2 + \alpha) - \beta S^* \gamma_1$

Placing the new values of $k_1 + e_1$ and $k_0 + e_0$ into equation (5.1) , we get;
$(\lambda^2 + \lambda(\lambda^2 + \lambda\gamma_2 + \alpha + \beta I^* + \gamma_1 + (-\beta S^*)) + ((\beta I^* + \gamma_1)(\gamma_2 + \alpha) - \beta S^* \gamma_1) = 0$
I. If $\mathcal{R}_0 = \frac{\mu\, N\, \beta}{\gamma_1(\gamma_2+\alpha)} < 1$, then for $E_1[\frac{\mu N}{\gamma_1}, 0, 0]$

$k_1 + e_1 = \gamma_2 + \alpha + \beta 0 + \gamma_1 + (-\beta\frac{\mu N}{\gamma_1}) = (\gamma_2 + \alpha)(\frac{\gamma_1}{\gamma_2\alpha} + (1 - \frac{\beta\mu N}{\gamma_1})) > 0$

$k_0 + e_0 = (\beta 0 + \gamma_1)(\gamma_2 + \alpha) - \beta(\frac{\mu N}{\gamma_1})\gamma_1 = \gamma_1(\gamma_2 + \alpha) - \beta\mu N > 0$

Therefore,

$(\lambda^2 + \lambda(k_1 + e_1) + k_0 + e_0) = (\lambda^2 + \lambda(\gamma_2 + \alpha)(\frac{\gamma_1}{\gamma_2\alpha} + (1 - \frac{\beta\mu N}{\gamma_1})) + \gamma_1(\gamma_2 + \gamma) - \beta\mu N) = 0$

Hence, equation $(\lambda^2 + \lambda(k_1 + e_1) + k_0 + e_0) = 0$ has roots with negative real parts. Consequently, if $\mathcal{R}_0 < 1$, then $E_1$ is locally asymptotically stable.

II.(a) If $\mathcal{R}_0 = \frac{\mu N\beta}{\gamma_1(\gamma_2+\alpha)} > 1$, then for $E_1[\frac{\mu N}{\gamma_1}, 0, 0]$ , $k_1 + e_1 < 0$
Hence, equation $(\lambda^2 + \lambda(k_1 + e_1) + k_0 + e_0) = 0$ has roots with positive real parts and this implies that $E_1$ is unstable.

(b) If $\mathcal{R}_0 = \frac{\mu\, N\, \beta}{\gamma_1(\gamma_2+\alpha)} > 1$ and then $E_2[\frac{\gamma_2+\alpha}{\beta}; \frac{\mu\, N\, \beta - \gamma_1(\gamma_2+\alpha)}{(\gamma_2+\alpha)\beta}; \frac{\alpha((\mu N\beta) - \gamma_1(\gamma_2+\alpha)}{\beta\gamma_1(\gamma_2+\alpha))}]$

$k_1 + e_1 = \gamma_2 + \alpha + \beta I^* + \gamma_1 + (-\beta S^*) = \gamma_2 + \alpha + \beta I^* + \gamma_1 - \beta(\frac{\gamma_2+\alpha}{\beta}) = \beta I^* + \gamma_1 > 0$

$k_0 + e_0 = (\beta I^* + \gamma_1)(\gamma_2 + \alpha) - \beta S^* \gamma_1 = (\beta I^* + \gamma_1)(\gamma_2 + \alpha) - \beta(\frac{\gamma_2+\alpha}{\beta})\gamma_1 = \beta I^*(\gamma_2 + \alpha) > 0$

The quadratic equation $(\lambda^2 + \lambda(k_1 + k_0) + (\lambda e_1 + e_0)) = 0$ has $k_1 + e_1$ and $k_0 + e_0$ all greater than zero.

The value of the roots of are $\lambda_{1,2} = \frac{-(k_1+e_1) \pm \sqrt{D}}{2}$ where $D = (k_1 + e_1)^2 + 4(k_0 + e_0)$. For $D \geq 0$ D is less than $(k_1 + e_1)$ and for $D < 0$,D has complex values. Either case the value for $\lambda$ has negative real parts.As in I, we obtain roots with negative real parts which implies that $E_2$ is locally asymptotically stable.

### 5.0.3 GENERALIZED SIR MODEL WITH DELAY

Given our generalized system with delay,

$$\begin{aligned} S' &= \mu N - \beta S(t) I(t-\tau) - \gamma_1 S(t) \\ I' &= \beta S(t) I(t-\tau) - (\gamma_2 + \alpha)\, I(t) \\ R' &= \alpha I(t) - \gamma_1 R(t) \end{aligned}$$

We find the equilibrium $E[S^*, I^*, R^*]$ by solving the systems

$$\begin{aligned} 0 &= \mu N - \beta S^* I^* - \gamma_1 S^* \\ 0 &= \beta S^* I^* - (\gamma_2 + \alpha) I^* \\ 0 &= \alpha I^* - \gamma_1 R^* \end{aligned}$$

Equilibrium calculation and points are the same as for the Generalized SIR model without delay.

- If $\mathcal{R}_0 < 1$ then generalized system is always equilibrium of $E_1[\frac{\mu N}{\gamma_1}, 0, 0]$ and it is locally stable

- If $\mathcal{R}_0 > 1$ then the generalized has two equilibria given as $E_1[\frac{\mu N}{\gamma_1}, 0, 0]$, which is unstable and $E_2[\frac{\gamma_2+\alpha}{\beta}; \frac{\mu N \beta - \gamma_1(\gamma_2+\alpha)}{(\gamma_2+\alpha)\beta}; \frac{\alpha(\mu N\beta - \gamma_1(\gamma_2+\alpha))}{\beta \gamma_1(\gamma_2+\alpha)}]$ which is locally asymptotically stable. $E_2$ would only exists if $\mathcal{R}_0 > 1$

### 5.0.4 LOCAL STABILITY OF EQUILIBRIUM OF GENERALIZED SIR MODEL WITH DELAY

Our linearized system at equilibrium, for $x_0 \in \mathbb{R}^n$ is given as $\mathbf{x\prime} = \mathbf{A_0 x(t)} + \mathbf{A_1 x(t-\tau)}$ where $A_0$ is the Jacobi matrix $Df(x_0)$ with respect to $x(t)$ and $A_1$ is the Jacobi matrix $Df(x_0)$ with respect to $x(t-\tau)$.

We would differentiate column-wise ,the Jacobi matrix $A_0$ with respect to $\frac{d}{dS}, \frac{d}{dI}, \frac{d}{dR}$ and Jacobi matrix $A_1$ with respect to $\frac{d}{dS(t-\tau)}, \frac{d}{dI(t-\tau)}, \frac{d}{dR(t-\tau)}$

Our Jacobi matrices would become :

$$A_0 = \begin{pmatrix} -\beta\tau(t-\tau) - \gamma_1 & 0 & 0 \\ \beta I(t-\tau) & -(\gamma_2+\alpha) & 0 \\ 0 & \alpha & -\gamma_1 \end{pmatrix}$$

$$A_1 = \begin{pmatrix} 0 & -\beta S(t) & 0 \\ 0 & -\beta S(t) & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

Placing both matrices into $\mathbf{x\prime} = \mathbf{A_0 x(t)} + \mathbf{A_1 x(t-\tau)}$ we solve to find the solutions of the linear system.

We consider the solution of the linear system of a delay differential equation in the form, $x(t) = \exp(-\lambda\tau)k$. This means that with respect to the previous time, $x(t-\tau) = \exp(-\lambda(t-\tau))k$.

$\lambda$ is a root of the characteristic equation $\det(A_0 + A_1 \exp(-\lambda(t-\tau)) - \lambda E) = 0$ where $E$ is a unit matrix. The characteristic equation is , at equilibrium $E[S^*, I^*, R^*]$ is given as

$$0 = \det \begin{pmatrix} -\beta I^* - \gamma_1 - \lambda & -\beta S^* \exp(-\lambda\tau) & 0 \\ \beta I^* & \beta S^* \exp(-\lambda\tau) - (\gamma_2+\alpha) - \lambda & 0 \\ 0 & \alpha & -\gamma_1 - \lambda \end{pmatrix}$$

This means $-(\lambda+\gamma_1)(\lambda^2+\lambda(k_1+k_0)+(\lambda e_1+e_0)\exp(-\lambda\tau))=0$ and we have one root is $\lambda=-\gamma<0$

This implies that the local stability depends on the roots of

$$(\lambda^2+\lambda(k_1+k_0)+(\lambda e_1+e_0)\exp(-\lambda\tau))=0 \tag{5.2}$$

To find the local stability of $E_2$ we need to show that equation (5.2) has no pure imaginary roots.Assuming that as $\lambda=i\omega,\omega>0$ is a root of the second degree transcendental polynomial function equation (5.2) and $i$ is a complex number.We use the Euler equation $\exp(i\omega\tau)$ in equation (5.2) to obtain

$$\omega^4+(k_1^2-2k_0-e_1^2)\omega^2+k_0^2-e_0^2=0 \tag{5.3}$$

Substituting $z=\omega$ in the quadratic equation (5.3) we obtain

$$z^4+(k_1^2-2k_0-e_1^2)z^2+k_0^2-e_0^2=0 \tag{5.4}$$

$E_2[\frac{\gamma_2+\alpha}{\beta};\frac{\mu\ N\ \beta-\gamma_1(\gamma_2+\alpha)}{(\gamma_2+\alpha)\beta};\ \frac{\alpha(\mu\ N\beta-\ \gamma_1(\gamma_2+\ \alpha))}{\beta\gamma_1(\gamma_2+\alpha)}]$ for $S^*,I^*andR^*$ respectively, if

$\mathcal{R}_0=\frac{\mu\ N\ \beta}{\gamma_1(\gamma_2+\alpha)}>1$, (necessary condition for $E$ to exist).

Using Equation 5.4, we get $k_0^2-e_0^2=(k_0-e_0)(k_0-e_0)>0$

where

$$\begin{aligned}k_1&=(\gamma_2+\alpha)+\beta I^*+\gamma_1\\ k_0&=(\beta I^*+\gamma_1)(\gamma_2+\alpha)\\ e_1&=-\beta S^*\\ e_0&=-\beta S^*\gamma_1\end{aligned}$$

In varying time,

- if $(k_0-e_0)>0$ and
- $(k_1^2-2k_0+e_1^2)<0$

then $\omega_+^2$ and $\omega_-^2$ is not positive and the roots are also not positive. Hence the characteristic equation would not have a purely imaginary root see ([29]).

$k_0+e_0=(\beta I^*+\gamma_1)(\gamma_2+\alpha)-\beta S^*\gamma_1>0$

$k_0-e_0=(\beta I^*+\gamma_1)(\gamma_2+\alpha)-\beta S^*\gamma_1>0$

Hence $(k_0-e_0)^2=(k_0-e_0)(k_0-e_0)>0$ when $\mathcal{R}_0=\frac{\mu\ N\ \beta}{\gamma_1(\gamma_2+\alpha)}>1$. We have $(k_1^2-2k_0+e_1^2)=(\gamma_1+\beta I^*)^2<0$ . Consequently equations (5.2) and (5.4) have no positive roots and hence has no pure imaginary roots.

Moreover, as shown in II.(b) the quadratic equation $(\lambda^2+\lambda(k_1+k_0)+(e_1+e_0))=0$ has $k_1+e_1$ and $k_0+e_0$ all greater than zero.

The value of the roots of are $\lambda_{1,2} = \frac{-(k_1+e_1)\pm\sqrt{D}}{2}$ where $D = (k_1 + e_1)^2 + 4(k_0 + e_0)$. For $D \geq 0$, D is less than $(k_1 + e_1)$ and for $D < 0$,D has complex values. Either case the value for $\lambda$ has negative real parts.And so we obtain roots with negative real parts which implies that $E_2$ is locally asymptotically stable.

For the local stability of the characteristic equation at equilibrium $E_1[\frac{\mu N}{\gamma_1}, 0, 0]$,it is given as:

$(\lambda^2 + \lambda(k_1 + k_0) + (\lambda e_1 + e_0)\exp(-\lambda\tau)) = 0$, which becomes

$(\lambda^2 + \lambda(\gamma_2 + \alpha + \gamma_1) + \gamma_1(\gamma_2 + \alpha) - (\frac{\mu N}{\gamma_1}(\lambda + \gamma_1)\exp(-\lambda\tau)) = 0 = \mathbb{F}(\lambda)$

$\mathbb{F}(0) = (0^2 + 0(\gamma_2 + \alpha + \gamma_1) + \gamma_1(\gamma_2 + \alpha) - (\frac{\mu N}{\gamma_1}(0 + \gamma_1)\exp 0\tau) = \gamma_1(\gamma_2 + \alpha) - \mu N\beta < 0$ if $\mathbb{R}_0 > 1$
On the other hand, $\lambda = \infty$ when $\mathcal{R}_0 > 1$, $\mathbb{F}(\infty) \to \infty$ hence $\mathbb{F}(\lambda)$ has positive roots which implies $E_1$ is unstable for $\mathcal{R}_0 < 1$.

The above analysis of the basic reproductive number at the equilibrium points of the system corresponds to the average number of infections caused by an infected person on susceptible population. When $R_0 > 1$, the disease would be difficult to contain and might become endemic to the specific population under observation. The disease goes extinct when $\mathcal{R}_0 < 1$.

## 5.1 EPIDEMIC CURVES FOR SIR MODEL

Using a projected population of $N = 624,404$ with an initial number of infected individuals of COVID-19 to be, $I(0) = 600$ and $R(0) = 0$,to be initial recovered individuals. We deduce our $S(0) = N - I(0) - R(0) = 623504$.The time length $\tau$, from exposure to symptoms is commonly between 5 to 14 days. We used an average time length of 11.5 days.About 97 percent of people show symptoms in that time length . Our $\alpha = \frac{1}{timelength} = 0.105$ and assumed rate of transmission from the susceptible region to the infected region is given as $\beta = 0.3$ and 0.03.We want to examine the dynamics of two basic reproductive number for the system, when $\mathcal{R}_0 > 1$ and when $\mathcal{R}_0 < 1$, to know the evolution of the Epidemic SIR model curves and also determine the local stability of the population.Below are images of the SIR model with and without delay produced using our assumed values in MATLAB.

### 5.1.1 SIR MODEL WITHOUT TIME DELAY

In Figure 6, we compare the epidemic curves of an SIR model with a basic reproductive number of $\mathcal{R}_0 > 1$ set at 3 and $\mathcal{R}_0 < 1$ set at 0.3 respectively.

Each figure was generated over a time period of 365 days. Incubation period or latency period was not considered in the generation of these epidemic curves.This means that Susceptible individuals would get infected immediately. When $\mathcal{R}_0 > 1$, infection rate rises exponentially to a peak of morbidity, which falls on 49 days and reaches 175

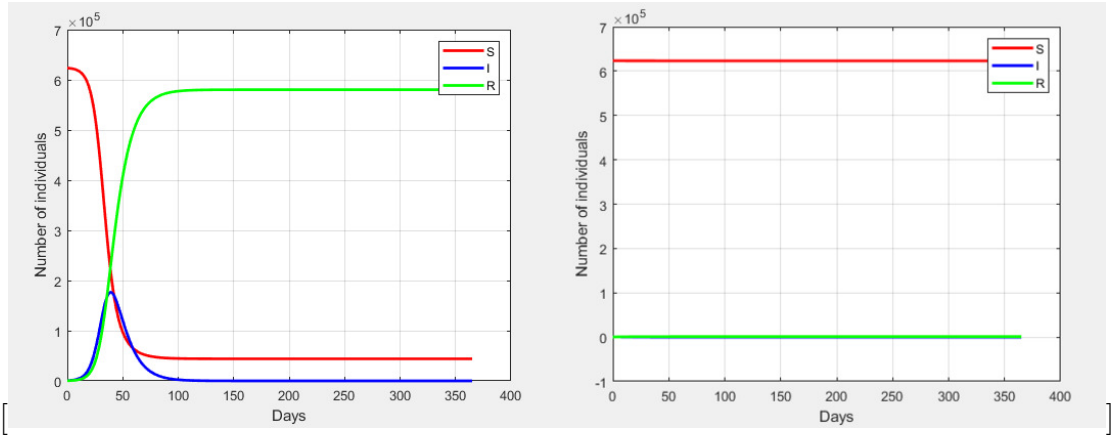



Figure 6: Epidemic curves of an SIR model without delay with basic reproductive numbers, $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively .

individuals per a 1000 population.It then decays into an endemic stage. The pandemic lasts in about 100 days . Fewer people may get infected but the disease would take quite some time to go extinct.
When $\mathcal{R}_0 < 1$, the rate of infection dies out and the susceptible population remains constant. At this point, the population or system is said to have a disease- free equilibrium.

### 5.1.2 SIR MODEL WITH TIME DELAY

Figure 7 included the incubation period before the host becomes infectious. The Epidemic curves of the SIR model with delay has a basic reproductive number of $\mathcal{R}_0 > 1$ set at 3 and $\mathcal{R}_0 < 1$ set at 0.3.

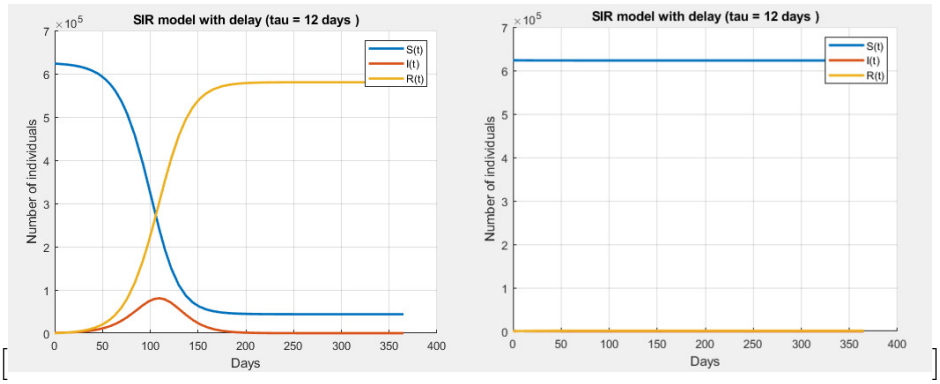



Figure 7: Epidemic curves of an SIR model with delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

The average incubation period is $\tau = 11.5$. Susceptible individuals who get infected show symptoms within this period and are infectious. When $\mathcal{R}_0 > 1$ , rate of infection grows steadily to a peak of morbidity,which falls on 110 days and reaches 80 individuals per a 1000 population.It decays into an endemic stage. Here the pandemic lasts in about

177 days. When $\mathcal{R}_0 < 1$ , the disease dies out and the Susceptible population remains constant and disease-free.

### 5.1.3 GENERALIZED SIR MODEL WITHOUT TIME DELAY

In Figure 8, we compare the epidemic curves of Generalized SIR model without delay with a basic reproductive number of $\mathcal{R}_0 > 1$ set at 3 and $\mathcal{R}_0 < 1$ set at 0.3

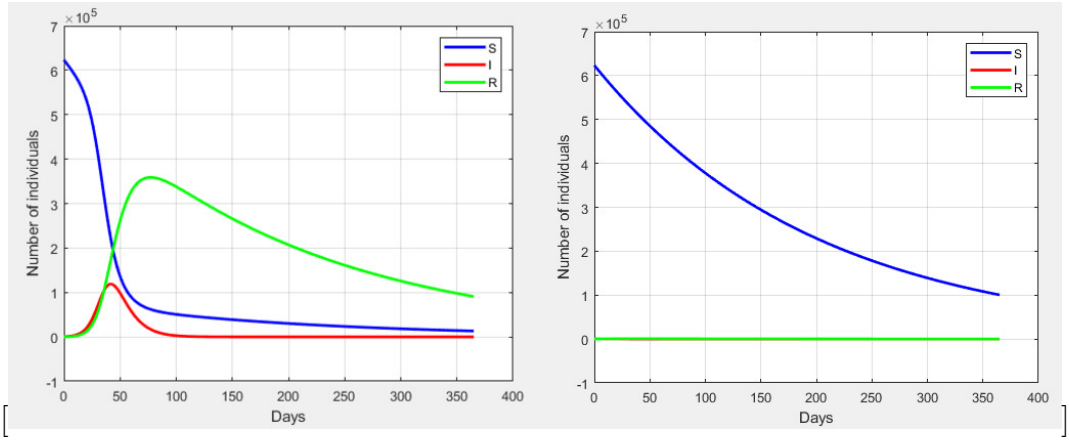



Figure 8: Epidemic curves of the Generalized SIR model without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

In the generalized SIR model without delay, pandemic lasts for about 93 days.The susceptible region decreases sharply not to zero.The infected region rises exponentially to a peak of morbidity ,which falls on 43 days and reaches 119 individuals per a 1000 population.It decays to the endemic stage after 96 days. At $\mathcal{R}_0 > 1$ , it stays in its endemic stage and would take some time to go extinct.

### 5.1.4 GENERALIZED SIR MODEL WITH TIME DELAY

Figure 9, we compares the epidemic curves of Generalized SIR model with delay with a basic reproductive number of $\mathcal{R}_0 > 1$ set at 3 and $\mathcal{R}_0 < 1$ set at 0.3

The susceptible region decreases at a steady rate.This makes the infected region to increase slowly .It decays to an endemic state,with approximately no peak of morbidity.It can happen that safety measures are put in place after the pandemic started and this slows down infection rate.The pandemic takes 180 days and may take a while for the disease to die out. At $\mathcal{R}_0 < 1$, the population has a disease-free equilibrium.

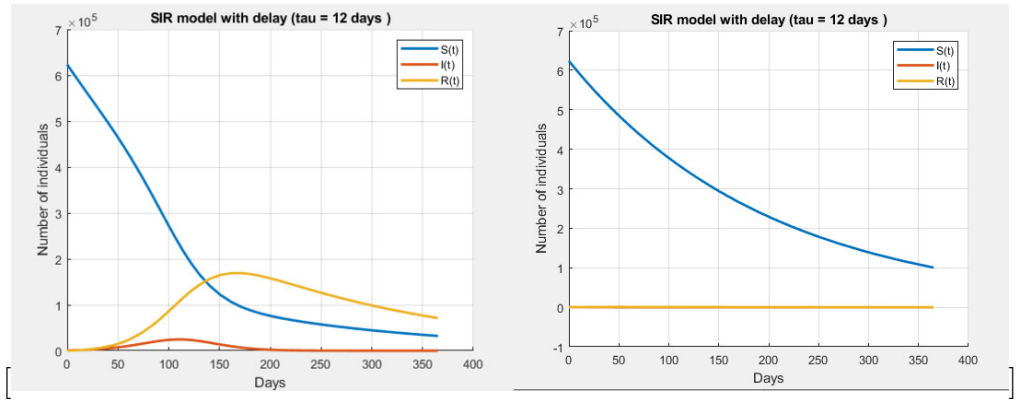



Figure 9: Epidemic curves of the Generalized SIR model with delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

### 5.1.5 SIR MODEL WITH AND WITHOUT TIME DELAY

We compare the SIR model without to the generalized SIR model with delay. We set the basic reproductive number $\mathcal{R}_0 > 1$ at 3 and $\mathcal{R}_0 < 1$ at 0.3.

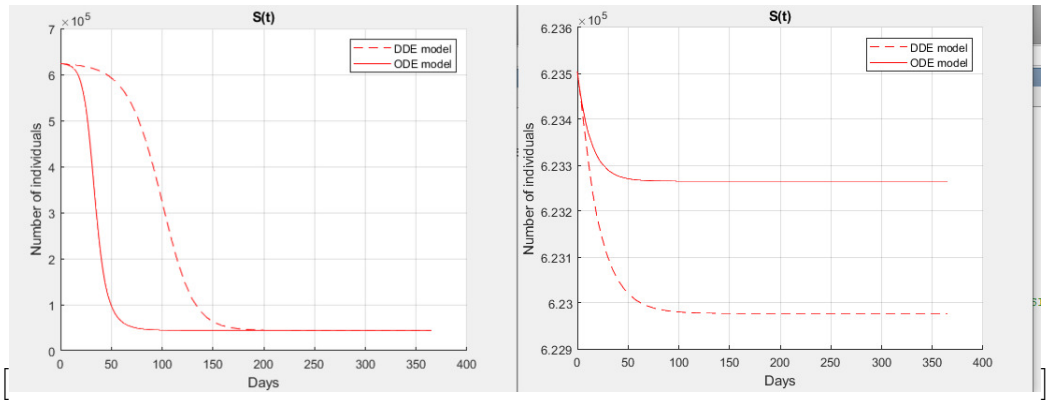



Figure 10: Epidemic curves of the Susceptible Compartment of an SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

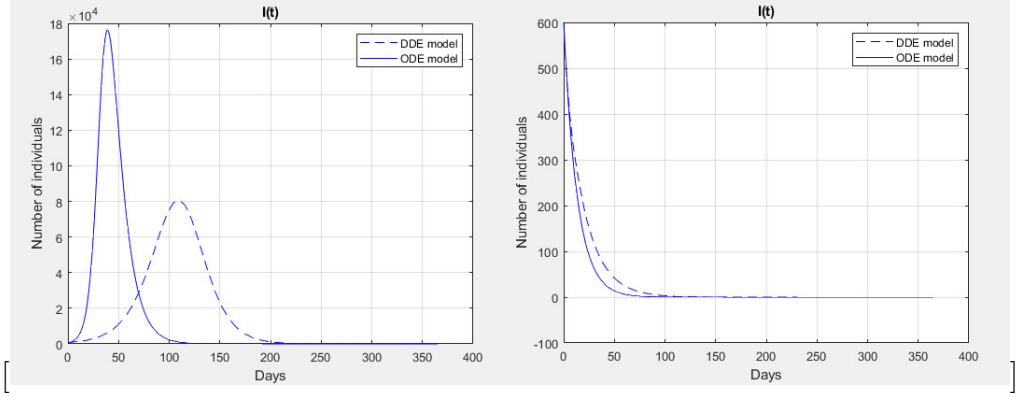



Figure 11: Epidemic curves of the Infected Compartment an SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

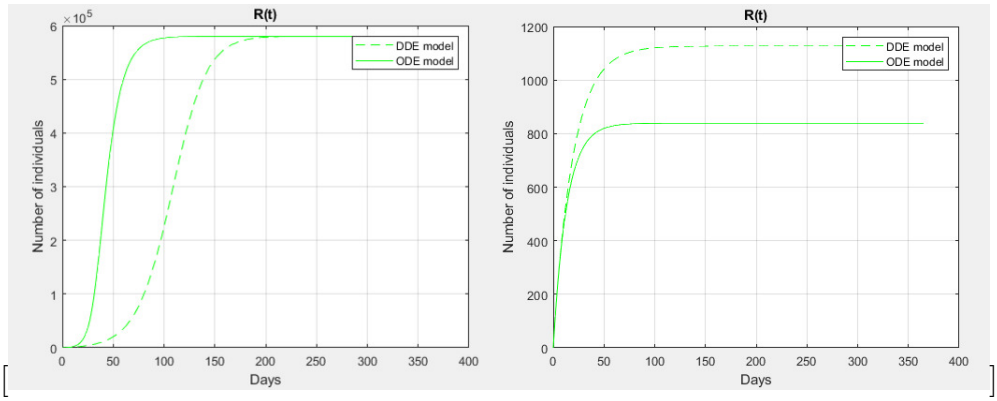



Figure 12: Epidemic curves of the Removed Compartment of an SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

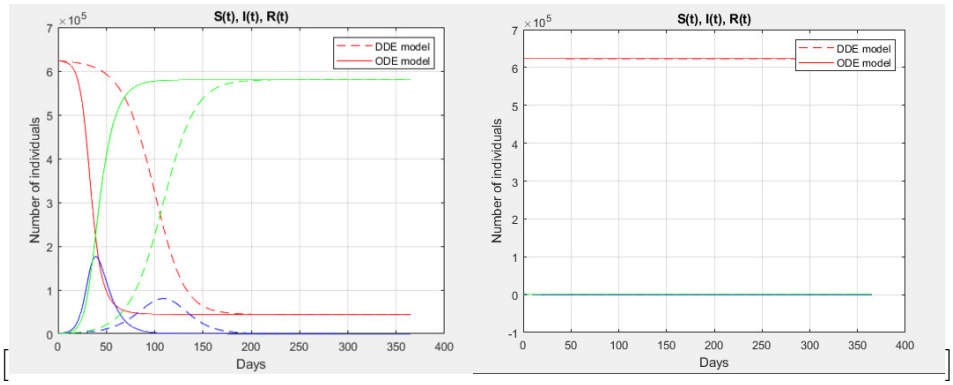



Figure 13: Epidemic curves of the SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively . Susceptible(red),Infected(blue) and Removed (Green)

When $\mathcal{R}_0 > 1$ ,the SIR model without delay has pandemic lasting 100 days.There is a sharp decrease in the susceptible population and the rate of infection grows exponentially to a peak of morbidity, which falls on 40 days, and reaches 175 individuals per 1000 population.Due to increase in infection of the population,many would recover (either heal or die). Comparing to the SIR model without delay, the SIR model with delay has infection rate growing gradually at a steady pace .It reaches a peak of morbidity, which falls on 110 days and reaches 80 individuals per 1000 population. The pandemic lasts for 200days.
SIR model with delay is more realistic than the SIR model without delay because it involves the latency period. The Susceptible population decreases at a slower pace .Individuals, after coming in contact with an infected individual, might not be infected (the individual might be immune),unless symptoms of disease show after incubation period.Hence,the infected population increases at a slower pace which is more realistic.At $\mathcal{R}_0 < 1$, the disease dies out,making the system disease free.

### 5.1.6 GENERALIZED SIR MODEL WITH AND WITHOUT TIME DELAY

We compare the generalized SIR model without delay to the generalized SIR model with delay at $\mathcal{R}_0 < 1$ set at 0.3 and $\mathcal{R}_0 > 1$ set at 3.
In $\mathcal{R}_0 > 1$, Susceptible region decreases but never to zero in each model. In the SIR

model with delay, the infected region rises exponentially to a peak of morbidity which falls on 43 days reaching 119 individuals per 1000 population.The pandemic in the model without delay lasts 96 days.In the model with delay, there is a gradual decrease in the susceptible population which makes transmission into the infected compartment low.There is approximately no peak of morbidity.

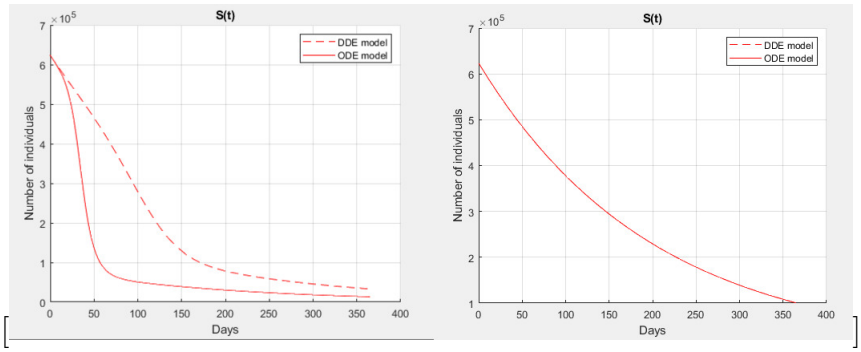



Figure 14: Epidemic curves of the Susceptible compartment of the Generalized SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

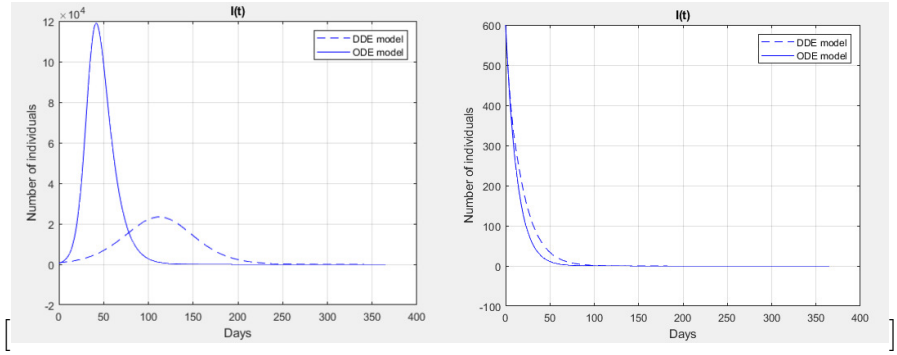



Figure 15: Epidemic curves of the Infected compartment of the Generalized SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

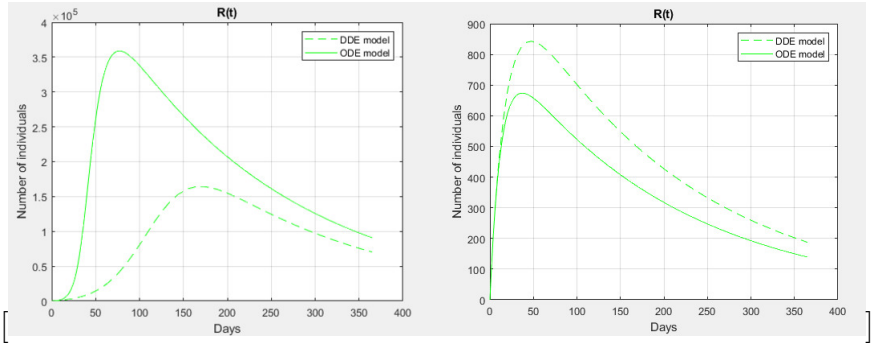



Figure 16: Epidemic curves of the Removed compartment of the Generalized SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

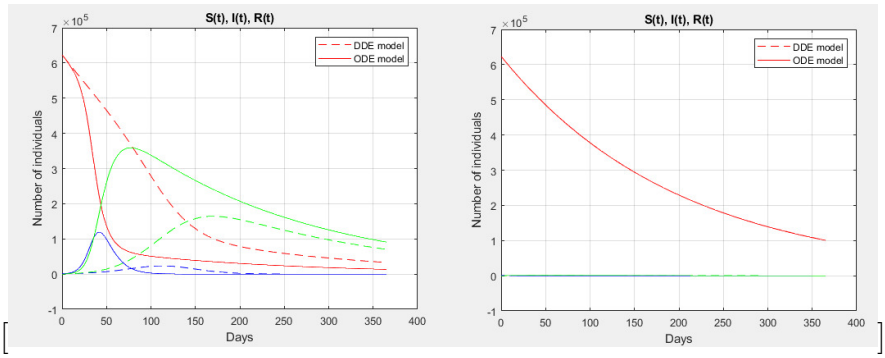



Figure 17: Epidemic curves of the Generalized SIR model with and without delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively .Susceptible(red),Infected(blue) and Removed (Green)

### 5.1.7 SIR MODEL WITH DIFFERENT TIME DELAY

We describe the evolution of the epidemic curve at different time delays. The incubation period of COVID-19 is from 5 to 14 days. We chose time variations of 5, 8.5, 11.5 and 14. *Susceptible without delay*, $Infected\ without\ delay\ and$ *Removed without delay*$(blueshortdashes), \tau_1 = 5(bluecurve), \tau_2 = 9.5(yellowcurve), \tau_3 = 11.5(greencurve) and \tau_4 = 14(redcurve)$. We set $\mathcal{R}_0 < 1$ at 0.3 and $\mathcal{R}_0 > 1$ set 3

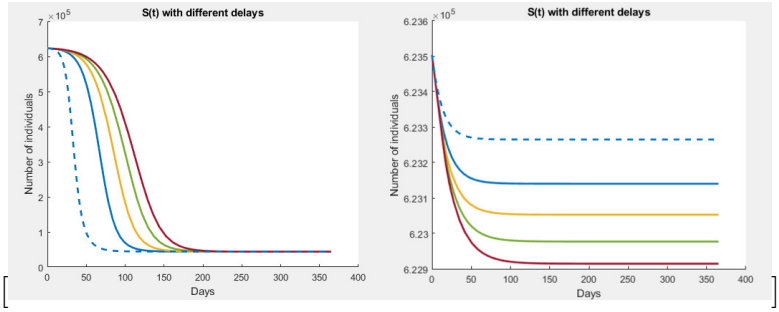



Figure 18: Figure:Epidemic curves of the Susceptible compartment with Different time delays and basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

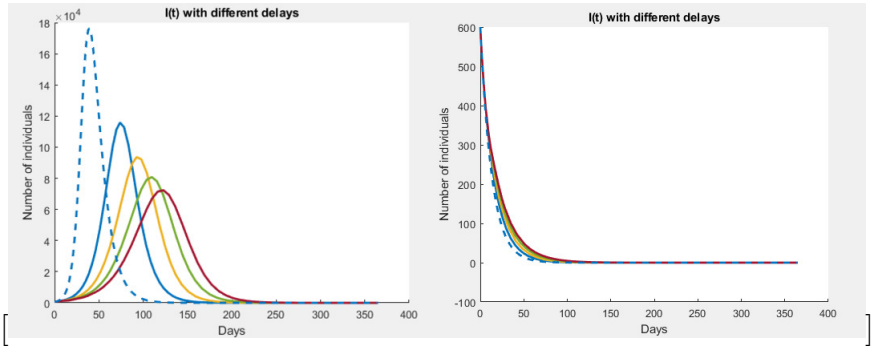



Figure 19: Figure:Epidemic curves of the Infected compartment with Different time delays and basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

We compare the SIR model with different time delays.The SIR curve without time delay was fixed into it to show the dynamics of the disease in those two cases.

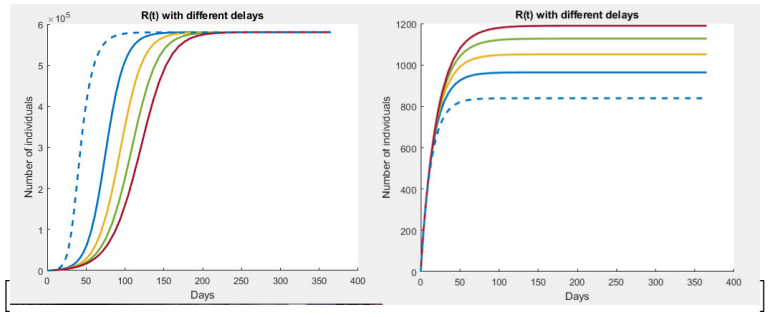



Figure 20: Epidemic curves of the Removed Compartment with Different time delay with basic reproductive numbers, $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

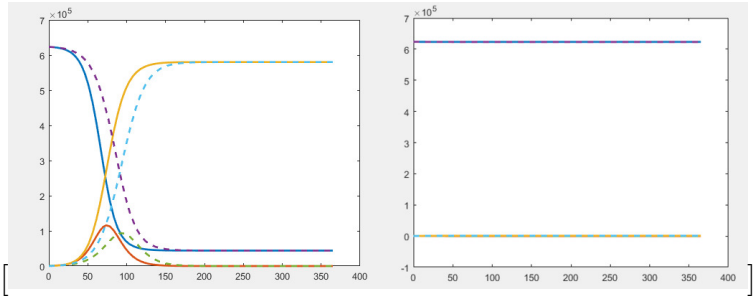


Figure 21: Epidemic curves of the SIR model with Different time delay with basic reproductive numbers, $\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively .

When $\mathcal{R}_0 > 1$, the individuals in the Susceptible compartment get the infection immediately.IT causes an increase in the Infected region in earlier times ($\tau < 14$) within a short pandemic period and more recoveries.
As the latency period increases, it would take a longer time for a susceptible individual to get infectious.The latency period elongates the timeline of the epidemic.

### 5.1.8 GENERALIZED SIR MODEL WITH DIFFERENT TIME DELAY

Describing the evolution of the Generalized epidemic SIR model curve at different time delays. The incubation period of COVID-19 is from 5 to 14 days. We choose time variations of 5, 8.5, 11.5 and 14. *Susceptible without delay*, *Infected without delay and Removed without delay*$(blueshortdashes), \tau_1 = 5(bluecurve), \tau_2 = 9.5(yellowcurve), \tau_3 = 11.5(greencurve) and \tau_4 = 14(redcurve)$.We set $\mathcal{R}_0 < 1$ at 0.3 and $\mathcal{R}_0 > 1$ set 3

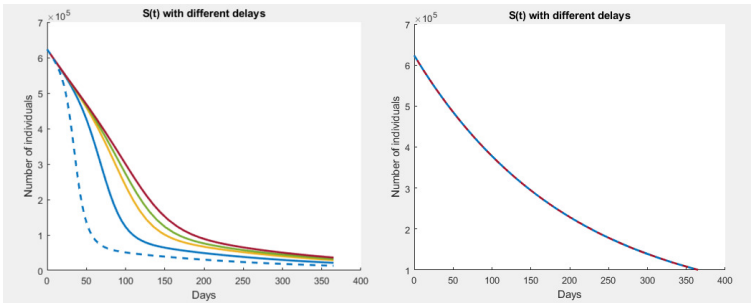



Figure 22: Epidemic curves of the Susceptible compartment of the Generalized SIR model with Different time delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

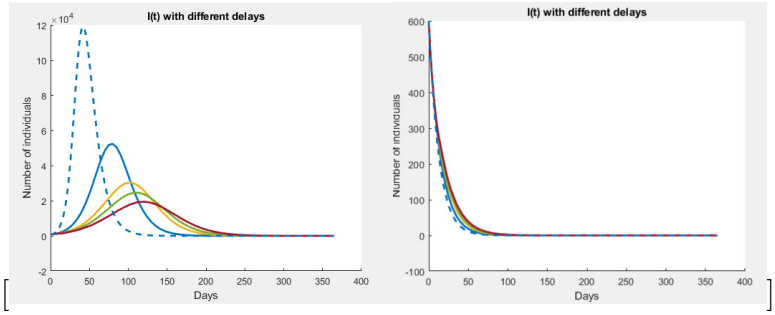



Figure 23: Epidemic curves of the Infected compartment of the Generalized SIR model with Different time delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

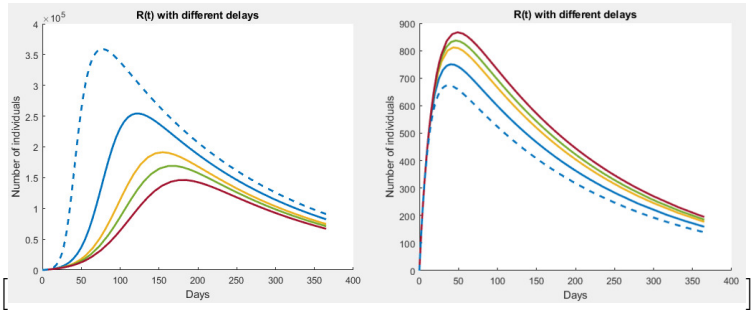



Figure 24: Epidemic curves of the Removed compartment of the Generalized SIR model with Different time delay with basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively

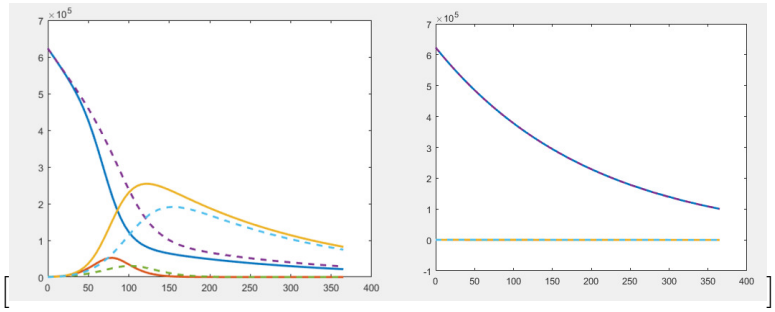


Figure 25: Epidemic curves of a Generalized SIR model with Different time delay. We included the basic reproductive numbers,$\mathcal{R}_0 > 1$ and $\mathcal{R}_0 < 1$ respectively to show,if it was disease-free or endemic of at different time delays .

# 6 Conclusion

Time delays have been used over many years to solve problems in various fields; from biology,physics,engineering and other fields. Time delays in a dynamical system system yields more realistic results than the ordinary differential equation.
In this thesis, we give an insight on delay differential equations in a dynamical system,especially in the view of the stability of their solution. In chapter one, we gave a theoretical background of delay differential, some examples and general principle surrounding the Delay Differential Equation.
In Chapter 2, we discussed about dynamical systems and the fundamental theories necessary for stability of a system.In Chapter 3 we discuss more on delay differential equations and it theories that accounts for stability.

In Chapter 4, we discussed epidemic dynamic models, types of the epidemic mathematical models and basic reproductive number.We formulated a mathematical model to solve an epidemiological dynamical problem.This model described the spread of a disease in a specific population under investigation.The vital demographic quantities were included in the formulation of this model.We chose the SIR model to analyze the spread of the Novel Coronavirus disease.We formulated the model without delays and then involved time delays.Epidemic problems are first written in Ordinary differential equations when solving.Because we want a more realistic result ,we involve time delays.

In Chapter 5, we assumed data was to analyze the dynamics of the disease using the SIR with delay and without delay. We observed how parameters like the basic reproductive number $\mathcal{R}_0$ affects the dynamics of the disease using the SIR model.

It is observed that, when the SIR model is without delay, it does not involve the latency period of the disease and hence gives unrealistic results.With time delays involved, the results of the SIR turns out to be more realistic compared to the SIR model without delay. Analysis of the basic reproductive number,$\mathcal{R}_0$ using the SIR model, shows that the disease is driven out at $\mathcal{R}_0 < 1$.This gives a disease-free equilibrium and the system, asymptotically stable. At $\mathcal{R}_0 > 1$, the disease is in an endemic state and would take a while to be die out.

Due to how realistic the results of the SIR model with delay is, it helps the right stakeholders to take steps that would curbed the disease spread. Using the model figures to analyze, stakeholders can rules for everyone in the susceptible population to observe social distancing and use nose masks. As the disease rises exponentially to a peak of morbidity, social distancing will not be enough. Public gatherings of more than two people is then cancelled. The disease decreases gradually into an endemic state. This shows that the steps taken were good. To wipe the disease out completely, stakeholders introduce vaccines and regular checkups.
Stakeholders are able to know using the SIR model with delay that, the disease may or may not be difficult to contain.

We resolve that ,ordinary differential equations,although it gives good approximations of a situation, fails to show the dynamics of the system well.This is done better by delay differential equation in the chosen dynamical system.It gives realistic results, although it

is difficult to analyze.

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# 7 APPENDIX

```
{SIR MODEL FUNCTION}
% This function solves SIR model.
    function [t,S] = modelSIR(S0,I0,R0,beta,alpha, t,N)
x0 = [S0 I0 R0];
     [T,Sv] = ode15s(@DifEq, t, x0);

        function dS = DifEq(t, x)
            xdot = zeros(3,1);
            xdot(1) = -beta.* x(1).* x(2)/N ;
            xdot(2) =  beta.* x(1).* x(2)/N - alpha.*x(2);
            xdot(3) =  alpha.*x(2);
            dS = xdot;
        end

%  S = Sv(:,1);
S = Sv;
end



GENERALIZED SIR MODEL FUNCTION

    function [t,S] = gen_modelSIR(S0,I0,R0,beta,alpha, t,N,mu,gamma1,gamma2)
% This function solves generalized SIR model.
x0 = [S0 I0 R0];
     [T,Sv] = ode15s(@DifEq, t, x0);

        function dS = DifEq(t, x)
           xdot = zeros(3,1);
            xdot(1) = mu-beta.* x(1).* x(2)/N-gamma1.* x(1) ;
            xdot(2) =  beta.* x(1).* x(2)/N - (gamma2+alpha).*x(2);
            xdot(3) =  alpha.*x(2)-gamma1.* x(3);
            dS = xdot;
        end

%  S = Sv(:,1);
S = Sv;
end

SIR MODEL WITHOUT DELAY

N = 624404;\\
S0 = 623504;\\ % initial susceptible
I0 = 600;\\ % initial infections
R0 = 0;\\ % initial removed
beta = 0.3;\\ % rate of infection
alpha = 0.1052;\\ % rate of recovery
```

```
time = 0:1:365;\\ % time interval
tv = linspace(time(1), time(end),365); \\

SIR = model(S0,I0,R0,beta,alpha,tv,N);\\
figure(1)\\
plot(tv,SIR(:,1),'r',tv,SIR(:,2),'b',tv,SIR(:,3),'g','LineWidth',2); grid on;\\
xlabel('Days');ylabel('Number of individuals');\\
legend('S','I','R');\\
function S = model(S0,I0,R0,beta,alpha, t,N)\\
x0 = [S0 I0 R0];\\
     [T,Sv] = ode15s(@DifEq, t, x0);\\

        function dS = DifEq(t, x)\\
            xdot = zeros(3,1);\\
            xdot(1) = -beta.* x(1).* x(2)/N ;\\
            xdot(2) =  beta.* x(1).* x(2)/N - alpha.*x(2);\\
            xdot(3) =  alpha.*x(2);\\
            dS = xdot;\\
        end\\

%  S = Sv(:,1);
S = Sv;\\
end\\


SIR MODEL WITH DELAY
 clear all; close all;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;
tau= 11.5; % delay
sol = dde23(@kmf,[tau],[S0;I0 ;R0 ],[0, 365]);

hold on;
figure(1)
plot(sol.x,sol.y,'LineWidth',2);
legend('S(t)','I(t)','R(t)')
name=sprintf('SIR model with delay (tau = %0.0f days )',tau); %title of graph
title(name)
xlabel('Days');ylabel('Number of individuals');
grid on;

function v = kmf(t,y,Z)
```

```
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
v(1) = - beta*y(1)*ylag1(2)/N;
v(2) = beta*y(1)*ylag1(2)/N -  alpha*y(2);
v(3) =  alpha*y(2);
end
```

GENERAL SIR MODEL WITHOUT DELAY

```
% This Matlab script solve generalized SIR model

close all;
clear all;
N = 624400;
S0 = 623500; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
time = 0:1:365; % time interval
tv = linspace(time(1), time(end),365);
mu=0.004;
gamma1=0.005;
gamma2=0.001;
tv = 0:1:365;

SIR = model(S0,I0,R0,beta,alpha, tv,N,mu,gamma1,gamma2);

figure(1)
plot(tv,SIR(:,1),'b',tv,SIR(:,2),'r',tv,SIR(:,3),'g','LineWidth',2); grid on;
xlabel('Days');ylabel('Number of individuals');
legend('S','I','R');

function [S] = model(S0,I0,R0,beta,alpha, t,N,mu,gamma1,gamma2)
% generalized SIR model.
x0 = [S0 I0 R0];
     [T,Sv] = ode15s(@DifEq, t, x0);

        function dS = DifEq(t, x)
           xdot = zeros(3,1);
            xdot(1) = mu-beta.* x(1).* x(2)/N-gamma1.* x(1) ;
            xdot(2) =  beta.* x(1).* x(2)/N - (gamma2+alpha).*x(2);
            xdot(3) =  alpha.*x(2)-gamma1.* x(3);
            dS = xdot;
        end
```

```
%  S = Sv(:,1);
S = Sv;

end
```

GENERAL SIR MODEL WITH DELAY

```
% This script solves generalized SIR model with delay
clear all; close all;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;
tau= 11.5; % delay
sol = dde23(@kmf,[tau],[S0;I0 ;R0 ],[0, 365]);

hold on;
figure(1)
plot(sol.x,sol.y,'LineWidth',2);
legend('S(t)','I(t)','R(t)')
name=sprintf('SIR model with delay (tau = %0.0f days )',tau); %title of graph
title(name)
xlabel('Days');ylabel('Number of individuals');
grid on;

% generalized system with delay
function v = kmf(t,y,Z)
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
mu=0.004;
gamma1=0.005;
gamma2=0.001;
v(1) = mu- beta*y(1)*ylag1(2)/N-gamma1.*y(1);
v(2) = beta*y(1)*ylag1(2)/N -  (gamma2+alpha)*y(2);
v(3) =  alpha*y(2)-gamma1*y(3);
end
```

COMPARING SIR MODEL WITH AND WITHOUT DELAY

```
 clear all; close all;
```

```
%comparison of SIR delay with and without time delay
%global beta;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;

[t,SIR] = modelSIR(S0,I0,R0,beta,alpha,tv,N); % solution of SIR model without
delay - calling function  modelSIR.m

sol = dde23(@kmf,[11.5],[S0;I0 ;R0 ],[0, 365]);

hold on;
figure(1)
plot(sol.x,sol.y(1,:),'r--',tv,SIR(:,1),'r');
legend('DDE model','ODE model')
title('S(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
figure
plot(sol.x,sol.y(2,:),'b--',tv,SIR(:,2),'b');
legend('DDE model','ODE model')
title('I(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
figure
plot(sol.x,sol.y(3,:),'g--',tv,SIR(:,3),'g');
legend('DDE model','ODE model')
title('R(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
figure
plot(sol.x,sol.y(1,:),'r--',tv,SIR(:,1),'r',sol.x,sol.y(2,:),
'b--',tv,SIR(:,2),'b',
sol.x,sol.y(3,:),'g--',
tv,SIR(:,3),'g');
legend('DDE model','ODE model')
title('S(t), I(t), R(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
function v = kmf(t,y,Z)
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
```

```
alpha = 0.1052; % rate of recovery
v(1) = - beta*y(1)*ylag1(2)/N;
v(2) = beta*y(1)*ylag1(2)/N -  alpha*y(2);
v(3) =  alpha*y(2);
end


COMPARING GENERALIZED SIR MODEL WITH AND WITHOUT DELAY
%This script compares generalized SIR model with and without delay

clear all; close all;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;
mu=0.004;
gamma1=0.005;
gamma2=0.001;
tau= 11.5; % delay
sol = dde23(@kmf,[tau],[S0;I0 ;R0 ],[0, 365]);


% solution of generalized SIR model without delay - calling function gen_modelSIR.m
[t,SIR] = gen_modelSIR(S0,I0,R0,beta,alpha, tv,N,mu,gamma1,gamma2);

sol = dde23(@kmf,[11.5],[S0;I0 ;R0 ],[0, 365]);

hold on;
figure(1)
plot(sol.x,sol.y(1,:),'r--',tv,SIR(:,1),'r');
legend('DDE model','ODE model')
title('S(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
figure
plot(sol.x,sol.y(2,:),'b--',tv,SIR(:,2),'b');
legend('DDE model','ODE model')
title('I(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;
figure
plot(sol.x,sol.y(3,:),'g--',tv,SIR(:,3),'g');
legend('DDE model','ODE model')
title('R(t)')
xlabel('Days');ylabel('Number of individuals');
```

```
grid on;
figure
plot(sol.x,sol.y(1,:),'r--',tv,SIR(:,1),'r',sol.x,sol.y(2,:),
'b--',tv,SIR(:,2),'b',sol.x,sol.y(3,:),'g--',tv,SIR(:,3),'g');
legend('DDE model','ODE model')
title('S(t), I(t), R(t)')
xlabel('Days');ylabel('Number of individuals');
grid on;

% generalized system with delay
function v = kmf(t,y,Z)
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
mu=0.004;
gamma1=0.005;
gamma2=0.001;
v(1) = mu- beta*y(1)*ylag1(2)/N-gamma1.*y(1);
v(2) = beta*y(1)*ylag1(2)/N -  (gamma2+alpha)*y(2);
v(3) =  alpha*y(2)-gamma1*y(3);
end
```

```
COMPARING SIR MODEL WITH DIFFERENT DELAY
clear all;
close all;
%comparing model with different time delays
%global beta;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;

[t,SIR] = modelSIR(S0,I0,R0,beta,alpha,tv,N); % solution of SIR model without
delay - calling function modelSIR.m

tau= [5,9.5,11.5,14];  % vector of different delays,
there can be also more or less delay
for i= 1 : length(tau)
sol(i) = dde23(@kmf,[tau(i)],[S0;I0 ;R0 ],[0, 365]);
end
hold on;
```

```
% comparison S(t) with different delay
for i=1:length(tau)
plot(sol(i).x,sol(i).y(1,:),'LineWidth',2);
plot(tv,SIR(:,1),'--','LineWidth',2) % S(t) without delay
title('S(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;
figure;

% comparison I(t) with different delay
for i=1:length(tau)
hold on;
plot(sol(i).x,sol(i).y(2,:),'LineWidth',2);
plot(tv,SIR(:,2),'--','LineWidth',2) % I(t) without delay
title('I(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;
figure;

% comparison R(t) with different delay
for i=1:length(tau)
hold on;
plot(sol(i).x,sol(i).y(3,:),'LineWidth',2);
plot(tv,SIR(:,3),'--','LineWidth',2) % R(t) without delay
title('R(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;

figure;



plot(sol(1).x,sol(1).y,sol(2).x,sol(2).y,'--','LineWidth',2);

function v = kmf(t,y,Z)
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
v(1) = - beta*y(1)*ylag1(2)/N;
v(2) = beta*y(1)*ylag1(2)/N -  alpha*y(2);
v(3) =  alpha*y(2);
end
```

```
COMPARING GENERALIZED SIR MODEL WITH DIFFERENT DELAYS
%This script shows the change of generalized SIR model
with different  delays and also compare with generalized SIR model without delay

clear all; close all;
N = 624404; % population
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
S0 = 623504; % initial susceptible
I0 = 600; % initial infections
R0 = 0; % initial removed
tv = 0:1:365;
mu=0.004;
gamma1=0.005;
gamma2=0.001;

% solution of generalized SIR model without delay - calling function gen_modelSIR.m
[t,SIR] = gen_modelSIR(S0,I0,R0,beta,alpha, tv,N,mu,gamma1,gamma2);

 % vector of different delays, there can be also more or less delay
tau= [5,9.5,11.5,14];
for i= 1 : length(tau)
sol(i) = dde23(@kmf,[tau(i)],[S0;I0 ;R0 ],[0, 365]);
end
hold on;


% comparison S(t) with different delays
for i=1:length(tau)
plot(sol(i).x,sol(i).y(1,:),'LineWidth',2);
plot(tv,SIR(:,1),'--','LineWidth',2) % S(t) without delay
title('S(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;
figure;

% comparison I(t) with different delays
for i=1:length(tau)
hold on;
plot(sol(i).x,sol(i).y(2,:),'LineWidth',2);
plot(tv,SIR(:,2),'--','LineWidth',2) % I(t) without delay
title('I(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;
figure;

% comparison R(t) with different delays
for i=1:length(tau)
```

```
hold on;
plot(sol(i).x,sol(i).y(3,:),'LineWidth',2);
plot(tv,SIR(:,3),'--','LineWidth',2) % R(t) without delay
title('R(t) with different delays')
xlabel('Days');ylabel('Number of individuals');
end;

figure;

plot(sol(1).x,sol(1).y,sol(2).x,sol(2).y,'--','LineWidth',2);

% generalized system with delays
function v = kmf(t,y,Z)
ylag1 = Z(:,1);
v = zeros(3,1);
N = 624404;
beta = 0.3; % rate of infection
alpha = 0.1052; % rate of recovery
mu=0.004;
gamma1=0.005;
gamma2=0.001;
v(1) = mu- beta*y(1)*ylag1(2)/N-gamma1.*y(1);
v(2) = beta*y(1)*ylag1(2)/N -  (gamma2+alpha)*y(2);
v(3) =  alpha*y(2)-gamma1*y(3);
end
```