A Linear Algebraic Framework for SIR Epidemic Modelling with Applications to Infectious Disease Dynamics and Healthcare Planning

Ahmed A.F. Osman[1,*], Aijaz Magray[2], Snehal Rathi[3], D. Raju[4], Madhulika Mishra[5], Aafaq A. Rather[5,*], Mohammed Ataelfadiel[1], Yasser Ayad Soliman[1], Ali Salem Bin Sama[6], Ala Abdullah[1]

[1]*Applied College, King Faisal University, P.O. Box 400, Al-Ahsa 31982, Saudi Arabia*

[2]*Department of Mathematics, JB Institute of Engineering and Technology, Hyderabad-500075, India*

[3]*Department of Computer Engineering, Vishwakarma Institute of Technology, Pune, India*

[4]*Department of Mathematics, Jawaharlal Nehru Technical University, Hyderabad-500085, India*

[5]*Symbiosis Statistical Institute, Symbiosis International (Deemed University), Pune, India*

[6]*Department of Management Information Systems (MIS), College of Business Administration, King Faisal University, Al-Ahsa, Eastern Province, Kingdom of Saudi Arabia*

Abstract: The Susceptible–Infected–Recovered (SIR) model is a fundamental framework in mathematical epidemiology used to analyse the spread and control of infectious disease within a population. Although classical SIR models provide valuable insights into epidemic dynamics, their nonlinear structure can make analytical investigation of local stability and epidemic thresholds challenging. In this study, a linear algebraic framework for the SIR model is developed by expressing the system in matrix form and analysing it through eigenvalues and eigenvectors. By linearizing the system around equilibrium points, the proposed approach enables systematic evaluation of local stability conditions and epidemic thresholds, providing insight into the early-stage growth or decay of an infection.

In addition to its theoretical advantages, the framework has important medical and public health applications. The eigenvalue-based analysis facilitates assessment of epidemic growth rates, local stability characteristics, and identification of critical control thresholds. These insights can support healthcare planning, including hospital resource allocation, intervention strategies, and epidemic preparedness. Overall, the study demonstrates that linear algebraic techniques provide a useful complementary framework for analysing local epidemic dynamics and supporting epidemiological decision-making. A comparison between the nonlinear SIR model and its linearized approximation is also presented to highlight the applicability and limitations of the linear algebraic approach.

Keywords: SIR model, Infectious disease modelling, Linear algebra, Eigenvalue analysis, Epidemic dynamics, Healthcare planning, Stability analysis, Basic reproduction number.

1. INTRODUCTION

The Susceptible–Infected–Recovered (SIR) model is one of the most fundamental and widely used mathematical models in epidemiology for describing the transmission dynamics of infectious disease. The model divides the population into three compartments: susceptible (S), infected (I), and recovered (R). By tracking the movement of individuals among these compartments, the SIR model provides a useful framework for understanding disease transmission, epidemic growth, and recovery processes within a population. It helps researchers and public health experts understand how infectious disease spread within a population over time. The SIR model provides a simplified yet powerful way to study how an infection starts, spreads, and eventually declines within a community. Although it is often expressed mathematically using differential equations, its main purpose is to capture the basic dynamics of disease transmission how people move from being susceptible to infected and finally to recovered [1,2]. in this model the population is divided into three main groups or compartments which are mentioned below.

- S(susceptible); an Individual who can catch the disease very easily'
- I (infectious); Individuals who have the disease and can transmit to the others
- R (Recovered): Individuals who have recovered from the disease and acquired immunity, or are otherwise removed from the transmission process and no longer contribute to disease transmission.

The model is usually represented using Differential equation

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

*Address correspondence to these authors at the Applied College, King Faisal University, P.O. Box 400, Al-Ahsa 31982, Saudi Arabia;
E-mail: afadol@kfu.edu.sa

Symbiosis Statistical Institute, Symbiosis International (Deemed University), Pune, India, E-mail: aafaq7741@gmail.com

$$\frac{dR}{dt} = \gamma I$$

where:

β (beta) is transmission rate (How easily the disease spread)

γ is Recovery Rate (How quickly people recover)

In recent years, the rapid spread of infectious disease such as COVID-19, influenza, and other emerging viral infections has emphasized the importance of reliable and interpretable epidemic models for supporting healthcare systems [3,4]. While classical SIR models provide valuable epidemiological insights, analytical investigation of local stability properties and epidemic thresholds can often benefit from linearization techniques and matrix-based analysis. There is a growing need for analytical approaches that can offer both mathematical clarity and practical usability in predicting disease dynamics. In this context, the present study introduces a linear algebraic framework that enables efficient analysis of epidemic behaviour, facilitating early detection of outbreak conditions and supporting healthcare planning through improved estimation of infection trends and system stability. The SIR model and its extensions have been extensively studied in mathematical epidemiology. Classical contributions by Kermack and McKendrick established the foundation of compartmental epidemic modelling, while subsequent works by Anderson and May and by Diekmann and Heesterbeek provided important developments in epidemic thresholds, reproduction numbers, and disease control strategies. These studies continue to serve as fundamental references for modern epidemiological research.

2. CONCEPTUAL EXPLANATION OF THE SIR MODEL

The SIR model is based on the assumption that the population can be divided into three distinct compartments according to disease status at a given time: susceptible (S), infected (I), and recovered (R). Together, these compartments represent the entire population under study and describe the progression of individuals through different stages of an infectious disease.These compartments are S(susceptible), I (infectious), R(Recovered) together these groups represent the entire population being studied [5, 6].

Susceptible(S)

In this group we include the individuals who are healthy but are vulnerable to the disease. they have not been yet infected but are at the risk of becoming infected if they come in the contact with infectious person the number of the susceptible persons or individuals usually decreases as the disease spreads, because some of them moves into the infectious category after get the infected.

Infectious(I)

In these types of group individuals currently carry the infection and can transmit to others through contact. they play a dynamic role to spread the disease. The number of infectious individuals changes over a time it increases because new infection occurs and decreases as the patients recover or die from the disease.

Recovered(R)

This compartment consists of individuals who have recovered from the infection and acquired immunity, or who have been removed from the transmission process. These individuals are no longer capable of transmitting the disease to susceptible members of the population. they are no longer capable to spread the infection. The size of this group increases while the number of the infection of the individuals decline [7,8].

The total population (N) is assumed to remain constant in the classical SIR model because births, natural deaths, and migration are not considered during the study period defined in equation (1). Mathematically it is mentioned below

Divide the total population into three groups

$$N = S(t) + I(t) + R(t) \quad (1)$$

Were

$S(t)$= number of susceptible individuals at time t

$I(t)$ = number of infectious individuals

$R(t)$ = number of recovered individuals

From a medical perspective, the three compartments of the SIR model correspond to critical stages in disease management and monitoring within a population. The susceptible group represents individuals at risk who may benefit from preventive measures such as vaccination and public health awareness [9]. The infectious group corresponds to actively diagnosed or undiagnosed patients who require medical attention, isolation, and treatment to prevent further transmission. The recovered group reflects individuals who have gained immunity or are no longer part of the transmission cycle, providing insight into the development of herd immunity within the population. Understanding the interaction between these compartments is essential for healthcare

authorities to design effective intervention strategies, allocate medical resources, and control the progression of infectious disease [10]. The interaction among these three compartments governs the transmission dynamics of an infectious disease and forms the basis for the mathematical formulation of the SIR model presented in the following section.

3. CHANGES AS THE DISEASE SPREADS THROUGH THE POPULATION

The SIR model describes how individuals transition among the susceptible (S), infected (I), and recovered (R) compartments over time. These transitions are governed by disease transmission and recovery processes, which determine the evolution of an epidemic within a population. The following explanation describes how each group changes as the disease's spreads through the population [11].

Rate of Change of Susceptible Individuals

The number of susceptible individuals decreases when they get contact with infected persons and contract the disease the more frequently susceptible and infectious persons interact, the faster new infection occurs this means that the rate at which the susceptible population declines depend on the two factors [12].

- How many individuals are susceptible.
- How many individuals are currently infectious.

When a susceptible person meets an infectious one, they may become infected the rate of new infections is proportion to both S & I Mathematically it is mentioned below equation (2).

$$\frac{dS}{dt} = -\beta SI \quad (2)$$

Were,

β is a transmission coefficient.

β measures the how fast the infection spreads.

Rate of Change of Infectious Individuals

The number of infectious persons in the population changes in two ways it increases when the new infections occur and decreases when infected persons recover or dies [13-18].

- The new infection adds the people to the infectious group as susceptible individuals becomes infected through contact.
- During the same time some infected persons recover and moves out of the infectious category.

Mathematically it is mentioned in below equation (3).

$$\frac{dI}{dt} = \beta SI - \gamma \quad (3)$$

Were $\gamma(gamma)$ is recovery rate or the fraction of infected people recovering per unit data.

Rate of Change of Recovered Individuals

Individuals who recover from the infection move into the recovered compartment. The size of this compartment increases as infected individuals recover at a rate proportional to the recovery parameter γ. In the classical SIR model, recovered individuals are assumed to acquire immunity and do not return to the susceptible class. the size of this category or group increases steadily as more people get recovered from this disease. The rate of recovery depends on how many individuals are currently infected and the average speed of their recovery. The recovery rate is denoted by γ once the persons enter in the recovered group, they are assumed to gain the permanent immunity means they cannot get infected again. Mathematically it is mentioned in below equation (4).

$$\frac{dR}{dt} = \gamma I \quad (4)$$

4. INTERPRETATION

The SIR model provides insight into how the numbers of susceptible, infected, and recovered individuals evolve during the course of an epidemic. By analysing the changes in these compartments over time, it is possible to identify different phases of disease transmission, epidemic growth, and eventual decline.by observing these changes we can interpret the different stages of disease spread and control.

Susceptible Population

- As time progresses, the number of susceptible individuals generally decreases because some individuals become infected through contact with infected individuals.
- According to Equation (2), the rate of decrease depends on both the susceptible population S and the infected population I.
- As more individuals become infected, fewer susceptible individuals remain available for disease transmission.

Infectious Population

The infected compartment is influenced by two competing processes. The first process increases the

number of infected individuals through new disease transmission events, while the second process decreases the number of infected individuals through recovery. Consequently, the epidemic grows when the rate of new infections exceeds the recovery rate and declines when recoveries dominate new infections. The equation (3) $\frac{dI}{dt} = \beta SI - \gamma I$ shows two effects.

I. The term $+\beta SI$ increases the number of infectious individuals (new infections).

II. The term $-\gamma I$decreases it (recoveries).

How to know the condition of the epidemic whether it grows or decline below mentioned mathematical equations defines the position of epidemic. These conditions are closely related to the basic reproduction number R_0. When $R_0 > 1$, each infected individual generates more than one secondary infection on average, leading to epidemic growth. Conversely, when $R_0 < 1$, disease transmission gradually declines and the epidemic eventually dies out

The epidemic grows when

$$\frac{dI}{dt} > 0 \; that \; is \; when \; \beta S > \gamma$$

The epidemic declines when

$$\frac{dI}{dt} < 0 \; that \; is \; when \; \beta S < \gamma$$

Recovered Population

The equation (4) $\frac{dR}{dt} = \gamma I$ clearly defines that the recovered group increases in proportion to the number of infectious individuals. the higher the recovery rate γ the faster this group grows.

Epidemic Threshold Condition

- The epidemic threshold represents the critical condition that determines whether an infectious disease will spread within a population. This threshold is commonly expressed in terms of the basic reproduction number R_0, which measures the average number of secondary infections generated by a single infected individual in a fully susceptible population. This ratio defines the basic reproduction number.

$$R_0 = \frac{\beta S}{\gamma}$$

- When $R_0 > 1$ the infection spreads rapidly (epidemic phase).
- When $R_0 < 1$ infection dies out the infections dies out over time.

These interpretations provide a qualitative understanding of epidemic behaviour and motivate the mathematical derivations presented in the subsequent sections.

5. DERIVATION OF THE MODEL

Population has been split into three compartments (functions of the time t) $S(t), I(t), R(t)$

Total population N is assumed constant.

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

The classical SIR model is based on the following assumptions:

- The total population is divided into three compartments: susceptible (S), infected (I), and recovered (R).
- The total population (N) is assumed to remain constant throughout the study period.
- Disease transmission occurs through contact between susceptible and infected individuals.
- The population is assumed to mix homogeneously, meaning that every individual has an equal probability of contacting any other individual.
- Recovered individuals acquire immunity and do not return to the susceptible compartment.
- Births, natural deaths, and migration are neglected in the classical SIR framework.

Law of Mass Action Equations

Under the homogeneous mixing assumption, the rate of new infections is proportional to the product of the susceptible and infected populations. This principle is known as the law of mass action and forms the basis for the transmission term in the SIR model. Let β denote the transmission rate and γ denote the recovery rate. The transmission rate β measures the frequency of effective contacts leading to infection, while γ represents the rate at which infected individuals recover. (new infection per unit time) IS Proportional to the product S.I introduce.

$\boldsymbol{\beta}$ Transmission contact rate per susceptible infected pair (units 1/person; time).

- γ recovery rate (units ;1/time) means infection $= \frac{1}{\gamma}$
- Susceptible decrease by new infections.

$$\frac{dS}{dt} = -\beta S\mathrm{I}$$

- Infection increases by new infections and decreases by recoveries.

$$\frac{dI}{dt} = \beta SI - \gamma\mathrm{I}$$

- Recovered increases by recoveries.

$$\frac{dR}{dt} = \gamma\mathrm{I}$$

Below Mentioned ODE'S Form the Classical SIR System

Non Dimensional/Fraction Form

Define proportion

$S(t) = \frac{S(T)}{N}$, $i(t) = \frac{i(t)}{N}$ and $r(t) = \frac{R(t)}{N}$

S0$s + i + r = 1$ the equation becomes.

$$\frac{dS}{dt} = -\beta si$$

$$\frac{di}{dt} = \beta si - ri$$

$$\frac{dr}{dt} = ri$$

Basic Reproduction Number $\boldsymbol{R_0}$

Consider a single infectious in other words susceptible population $(S \cong 1)$ the expected number of secondary infections produced by one infectious individual over their infectious period is.

$$R_0 = \frac{\beta}{\gamma}$$

Interpretation;

- If $\boldsymbol{R_0} > 1$ an introduced infection tends to grow (epidemic possible)
- If $\boldsymbol{R_0} < 1$ infection delay

Epidemic Growth Condition

From the equations we get.

$\frac{di}{dt} = i(\beta S - \gamma)$ At epidemic start $S \cong S(0) \cong 1$ So

$\frac{di}{dt}\Big|_{t=0} \cong i(0)(\beta - \gamma) = i(0)\gamma(\boldsymbol{R_0} - \mathbf{1})$.

Thus, initial exponential growth if $\boldsymbol{R_0} > 1$.

Peak Infection Condition

Peak i(t) occurs when $\frac{di}{dt} = 0\ and\ i > 0$

From $\frac{di}{dt} = i(\beta S - \gamma)$

We get at peak $(\beta S - \gamma) = 0$ and $s = \frac{\gamma}{\beta} = \frac{1}{\boldsymbol{R_0}}$

So, the fraction of susceptible at the infection peak equal to $\frac{1}{\boldsymbol{R_0}}$ this is useful to estimate when the epidemic turns around.

Conserved Relation between S & R – Integrate to Final Size

Divide $\frac{dS}{dt}\ by\ \frac{dR}{dt}$ To Eliminate t

$$\frac{dS}{dt} = \frac{\frac{dS}{dt}}{\frac{dR}{dt}} = \frac{-\beta si}{\gamma I} = \frac{-\beta}{\gamma}S$$

$$\frac{dS}{dR} = \frac{-\beta}{\gamma}S$$

This is a separable ODE for S as a function of R.

$$\frac{dS}{S} = \frac{-\beta}{\gamma}dr$$

Integrate from initial time (0) to t

$$in\frac{s(t)}{s(0)} = \frac{-\beta}{\gamma}\big(R(t) - R(0)\big)$$

Equivalently for fractions with $r = \frac{R}{N}$ & $s = \frac{S}{N}$

$$in(s(t) - in\ \big(s(0)\big) = -R_0(r(t) - r(0)$$

Final size equation (when $\boldsymbol{t \to \infty}$)

Let $S_\infty = S(\infty)\,, r_\infty = r(\infty)$

Usually, $r_0 = 0\ and\ s(0) = 1 - i(0) \cong 1$

From the integrated relation

$$inS_\infty - in\ s(0) = -\boldsymbol{R_0}(r_\infty - r(0)$$

Since $r_\infty = 1 - S(\infty)$ (all infections eventually leave I), assuming, $r_0 = 0$

$$inS_\infty - in\ s(0) = -R_0(1 - s_\infty)$$

If $S(0) \cong 1$ (linitially susceptibility near 1) this implies to

$$inS_\infty = -R_0(1 - s_\infty)$$

Rewrite in a more conventional final size form $(let\ z = 1 - nS_\infty = r_\infty)$ fraction ultimately infected

$$in(1 - z) = -R_0 z$$

Or equivalently equation for z gives the final epidemic size (fraction ever infected) it must be solved numerically for given R_0 important links.

Let C denote the constant of integration determined by the initial conditions. Thus,

$$\ln S + (\beta/\gamma)R = C,$$

were

$$C = ln(S_0) + (\beta/\gamma)R_0.$$

This relation establishes the connection between the susceptible and recovered populations throughout the epidemic and forms the basis for deriving the final size equation.

- If $R_0 \leq 1$ the non-trivial solution is z=0 (no out-break).
- If $R_0 > 1\,1$ there is a non-zero z some finite fraction infected.

6. EXTENDED SIR MODEL WITH DISEASE-INDUCED MORTALITY

In many real-world epidemics, disease-induced mortality may play an important role in shaping epidemic dynamics. To account for this effect, the classical SIR model can be extended by introducing an additional compartment D(t), representing individuals who have died as a result of the disease. Unlike the classical SIR model, the total living population is no longer constant because infected individuals may either recover or die [10].

$$\frac{dS}{dt} = -\beta SI \quad (5)$$

$$\frac{dI}{dt} = \beta SI - (\gamma + \alpha)I \quad (6)$$

$$\frac{dR}{dt} = \gamma I \quad (7)$$

$$\frac{dD}{dT} = \alpha I \quad (8)$$

where μ represents the disease-induced mortality rate. In this formulation, infected individuals leave the infected compartment either through recovery at rate γ or through disease-induced death at rate μ. Consequently, the total living population decreases over time as deaths accumulate in compartment D(t)

where:

$S(t)$= susceptible

$I(t)$ = infectious

$R(t)$ = recovered

D(t) = Deceased (due to the disease)

$\frac{dS}{dt} = -\beta SI$ Susceptible decrease only because they get infected no one new is born mentioned in equation (5).

$\frac{dI}{dt} = \beta SI - (\gamma + \alpha)I$ infected disease due to contact with S and decrease when they either recover (γI) or die from the disease (γI).defined in equation (6).

$\frac{dR}{dt} = \gamma I$ recovered people due to recovery from infection. defined in equation (7).

$\frac{dD}{dT} = \alpha I$ decreased count increases with the number of infected and death rate α .defined in equation (8).

(7) Relation between I and S; (trajectory in the S-I plane)

To obtain a direct relationship between the infected and susceptible populations, the differential equations for I and S are combined to eliminate the time variable t. Dividing $dI/dt\ by\ dS/dt$ yields a differential equation describing the trajectory of the epidemic in the S–I phase plane. $\frac{dI}{dt}\ by\ \frac{dS}{dt}$

$\frac{dI}{dt} = \frac{\beta SI - (\gamma+\alpha)I}{-\beta SI}$ = -1+$\frac{\gamma+\alpha}{\beta S}$

Integrate by separable

$$\int dI = \int -1 + \frac{\gamma + \alpha}{\beta S}\, ds$$

Integrating both sides with respect to S gives

$I = -S + ¥frac{¥gamma + ¥mu}{¥beta}¥ln(S) + C,$

where C is a constant of integration determined by the initial conditions.

Thus $Is =$ -S+$\frac{\gamma+\alpha}{\beta S}\ ins + c$

When integration constant C is set by initial conditions.

$$c = I_0 + S_0 - \frac{\gamma + \alpha}{\beta} inS_0$$

So the solution curve is the implicit relation.

$$I + S - \frac{\gamma + \alpha}{\beta} inS_0 = I_0 + S_0 - \frac{\gamma + \alpha}{\beta} inS_0$$

This expression generalizes the classical SIR trajectory by incorporating disease-induced mortality through the parameter μ. When μ = 0, the relation reduces to the corresponding expression for the standard SIR model.

The phase-plane relationship between S and I provide useful qualitative information about epidemic evolution and can be used to study infection trajectories without explicitly solving the full system of differential equations.

8. BASIC REPRODUCTION NUMBER R_0

The basic reproduction number, denoted by (R_0), is one of the most important quantities in mathematical epidemiology. It represents the average number of secondary infections generated by a single infected individual introduced into a completely susceptible population. The value of (R_0) determines whether an infectious disease can spread within a population or gradually disappear.

$$\frac{dI}{dt} = I(\beta S_0 - (\gamma + \alpha)$$

Define $\boldsymbol{R_0} = \frac{\beta S_0}{\gamma+\alpha} = \frac{\beta}{\gamma+\alpha}$

If S_0 is closed to N_0 one often writes

$$\boldsymbol{R_0} = \frac{\beta}{\gamma + \alpha}$$

Using fractions of population)

The basic reproduction number serves as an epidemic threshold parameter:

- If $(R\_0 > 1)$, each infected individual generates more than one secondary infection on average, and the epidemic can spread within the population.
- If $(R\_0 < 1)$, disease transmission declines over time and the epidemic eventually dies out.
- If $(R\_0 = 1)$, the disease remains at a critical threshold between growth and decline.

The reproduction number provides a direct measure of disease transmissibility and is widely used in public health planning to evaluate intervention strategies, vaccination requirements, and outbreak control measures.

9. RESULTS OF SIR EPIDEMIC MODEL USING A LINEAR ALGEBRAIC APPROACH

When analysed while using the linear algebraic approach, the system can be represented in a matrix form, which allows researchers to study the relationship between variables (S, I, R) more efficiently and gives clear situation about the epidemic condition at particular time.

This approach focuses on examining how small changes in one group affect the others and helps to determine the stability of the system for example, whether the disease will die out or continue spreading over time.

By treating the rates of change as a system of linear relationships, we can analyze the epidemic's behaviour using eigenvalues and eigenvectors (conceptually, without detailed calculation). These values indicate whether the infection grows (unstable system) or decays (stable system) as time progresses.

Introduction (No Birth/Death)

In this section, the classical SIR model is analysed under the assumption that the total population remains constant and no births or deaths occur during the observation period. The objective is to investigate the local stability of the disease-free equilibrium using linear algebraic techniques, including Jacobian matrices and eigenvalue analysis. because no birth or natural death or death due to disease during the period of the observation will not be considered. This situation represents a closed population, where person can move only the three states Suspectable(S), Infectious(I), and Recovered (R) without anyone entering or leaving the system mentioned below in equation (9), (10) and (11).

The model is based on the following conceptual ideas:

$$S = -\beta \frac{SI}{N} \quad (9)$$

$$I = \beta \frac{SI}{N} - \gamma I \quad (10)$$

$$R = \gamma I \quad (11)$$

Where $N = S + I + R$ constant

Step(A) Disease free Equilibrium (DFE)

DFE (S, I, R) = (N, 0, 0)

The disease-free equilibrium (DFE) corresponds to a state in which no infected individuals are present in the population. At this equilibrium, the entire population is susceptible and the infection has not yet established itself within the community.

Step (B): linearize; compute Jacobian and evaluate at DFE Jacobian J (S, I, R) (Compute partial derivatives)

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

Evaluate $DFS = N, and\ I = 0$

$$J_{DFE} = \begin{pmatrix} 0 & -\beta & 0 \\ 0 & \beta - \gamma & 0 \\ 0 & \gamma & 0 \end{pmatrix}$$

Step (C) Eigen values;

Characteristic polynomial yields eigenvalues

$\lambda_1 = \beta - \gamma$, and $\lambda_2 = 0$ and $\lambda_3 = 0$

Interpretation

- Define $R_0 = \frac{\beta}{\gamma}$.
- If $\beta - \gamma > 0 \quad (i, e\, R_0 > 1)$ one eigen value is positive ⇒ unstable ⟶ initial growth exponentially.
- If $\beta - \gamma < 0 \quad (i, e\, R_0 < 1)$ that eigen value is negative ⇒DFE Stable ⟶ small infection dies out.
- The other two zero eigen values reflect conservation of total N and the triangular structure; they don't change the epidemic threshold conclusion.

Early Time Solution (Linear Approximation)

For small $I(S_0, S \cong N)$

$I \cong (\beta - \gamma)I \;\Rightarrow\; I(T) \cong I(0)e^{(\beta-\gamma)t}$

Example $if \beta = 0.5$ and $\gamma = 0.2$

$\Longrightarrow \beta - \gamma = 0.3$, $R_0 = 2.5$ then $I(t) \cong I(0)e^{0.3t}$ exponential growth rate is 0.3

This approximation is valid only during the early phase of an epidemic when the susceptible population remains close to its initial value. As the epidemic progresses, nonlinear effects become significant and the full SIR model must be considered.

Introduction (Including Birth/Death at Rate μ)

In this subsection, the classical SIR model is extended by incorporating demographic effects through birth and death processes. The inclusion of the parameter μ allows the model to represent populations in which individuals enter and leave the system over time. Such an extension provides a more realistic description of long-term epidemic dynamics and permits the existence of endemic equilibria.in the population, represented by the symbol μ. This adjustment makes the model more realistic, as in most real-world situations, populations are not closed new individuals are born, and others die over time defined below in equation (12), (13) and (14).

This extended model is required for studying endemic equilibria, where the disease persists in the population over the long term. $I^* > 0$

The model is defined as

$$S = \mu N - \beta \frac{SI}{N} - \mu S \tag{12}$$

$$I = \beta \frac{SI}{N} - (\gamma + \mu)I \tag{13}$$

$$R = \gamma I - \mu R \tag{14}$$

Step (A) Disease free equilibrium (DFE)

DFE (S, I, R) = (N, 0, 0)

The disease-free equilibrium remains unchanged and corresponds to the absence of infection in the population. Stability of this equilibrium determines whether an introduced infection can invade the population.

Step (B): linearize at DFE- Infection subsystem;

Jacobian at DFE (full 3×3) becomes (substitute S=N and I=0)

$$J_{DFE} = \begin{pmatrix} -\mu & -\beta & 0 \\ 0 & \beta(-\gamma + \mu) & 0 \\ 0 & \gamma & -\mu \end{pmatrix}$$

To determine whether an epidemic can occur, it is sufficient to analyse the infected compartment because disease transmission is governed by the dynamics of infected individuals near the disease-free equilibrium. This approach leads naturally to the next-generation matrix formulation presented in the following subsection. (The block that drives I) Equivalently use the 2×2 subsystem for (S, I) or use the next generation matrix method (clean and Standard)

The inclusion of demographic processes modifies the epidemic threshold and allows the possibility of endemic disease persistence. These effects can be quantified using eigenvalue analysis and the next-generation matrix method.

Next Generation Matrix Method

The next-generation matrix (NGM) method provides a systematic approach for computing the basic reproduction number (R_0). The method separates the production of new infections from all other transitions involving infected individuals and evaluates these quantities at the disease-free equilibrium.

$F(I) = \beta \frac{SI}{N}$ linearized at DFE ,$F = \beta I$

$V(I) = (\gamma + \mu)I$

So, the next generation scale is

$$R_0 = \frac{New\ infection\ rate}{removal\ rate} = \frac{\beta}{(\gamma + \mu)}$$

Eigen Value Criterion

Linearized $I \cong (\beta - (\gamma + \mu))I$ the eigen value is

$\lambda = (\beta - (\gamma + \mu)$

- If $\lambda > 0 \Longrightarrow R_0 > 1 \Rightarrow DFE$ unstable infection grows
- If $\lambda < 0 \Longrightarrow R_0 < 1 \Rightarrow$ infection decays

Endemic equilibrium when $R_0 > 1$

With vital dynamics there is an endemic steady state $(S,^* I^*, R^*)$

With $I^* > 0$ solve equilibria.

At steady state

$0 = \mu N - \beta \frac{S^* I^*}{N} - \mu S^*$ And $0 = \beta \frac{S^* I^*}{N} - (\gamma + \mu)\, I^*$

From second equation if $(I^* > 0)$

$$\beta \left(\frac{S^*}{N}\right) = (\gamma + \mu) \Rightarrow S^* = \frac{(\gamma + \mu)}{\beta} N = \frac{N}{R_0}$$

Then use population conservation $S^* + I^* + R^* = N$ and first equation to find I^* algebra gives

$$I^* = N(1 - \frac{1}{R_0}) \frac{\mu}{\gamma + \mu}$$

Comparison: Standard (Nonlinear) SIR Model vs Linear-Algebraic Model

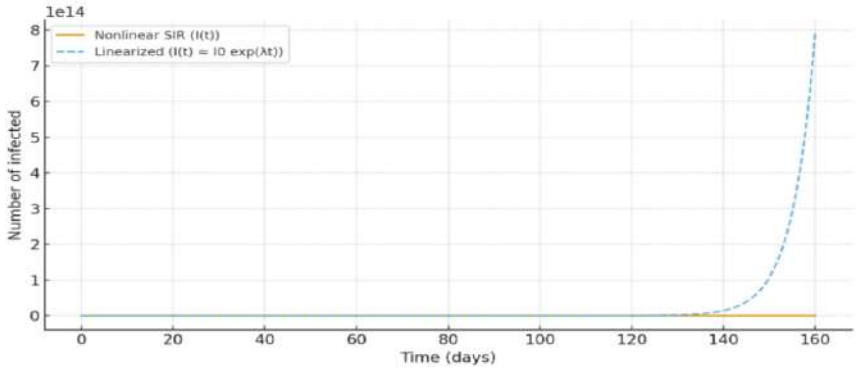
The classical SIR model describes epidemic dynamics through a system of nonlinear differential equations. These nonlinear interactions allow the model to capture the complete epidemic trajectory, including epidemic growth, peak infection levels, and final epidemic size. In contrast, the linear-algebraic approach is obtained by linearizing the system around an equilibrium point and analysing the resulting Jacobian matrix and its eigenvalues. This approach provides valuable information regarding local stability and early-stage epidemic behaviour among the different stages like susceptible, infected and recovered individuals, which often needs or requires calculus or simulations to understand how infection rise and fall over a period of time. The Linear algebraic approach restructured the above-mentioned model into a matrix form, allowing a clearer view of how each group influences the others and making it easier to study sensitivity, stability, and the effects of small changes in parameters. while the classical model focuses mainly in predicted infection curves, the new linear algebraic approach or method provides a more structured and transparent way to analyse the underlying behaviour of the epidemic, the comparison between the nonlinear approach and the algebraic approach is mentioned in below Table **1**.

The linearized approximation is valid primarily during the early phase of an epidemic when the susceptible population remains close to its disease-free equilibrium value. As the epidemic progresses and the susceptible population decreases substantially, nonlinear effects become increasingly important, causing the discrepancy between the nonlinear and linearized solutions to grow.

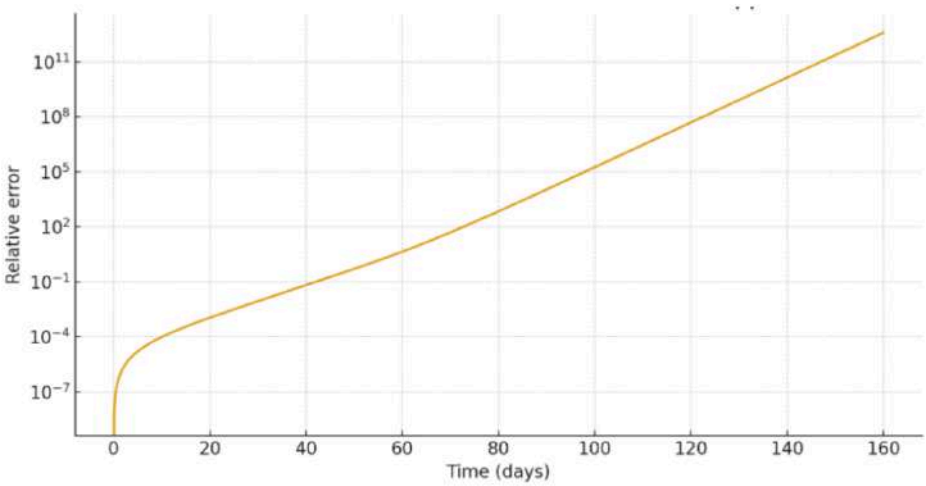
The nonlinear SIR model defines the full epidemic trajectory because of its SI infection term, whereas the linear algebraic approach studies the system by linearizing about equilibria. Linear methods (Jacobian, eigenvalues, and the next-generation matrix) efficiently yield the epidemic threshold R_0 and the early exponential growth rate λ. These results correctly predict whether small introductions of infection will grow or decay, and they produce analytic expressions for the endemic equilibria when vital dynamics are included. However, because linearization is valid near

Table 1: Comparison: Standard (Nonlinear) SIR Model vs Linear-Algebraic Approach

Aspect	Classical nonlinear SIR	Linear Algebraic Approach
Equations	Nonlinear ODE with SI coupling	Linear Approximation of ODEs near an equilibrium (Jacobians)
Exact global dynamics	Requires phase-plane conserved N or numerical simulation	Local dynamics only valid near the dynamic equilibrium use
Stability Criterion (DFE)	Threshold $\boldsymbol{R_0 = \frac{\beta}{\gamma}}$ (no vital dynamics) decides fate	Same threshold appears via eigen value of Jacobian $\beta - \gamma$
Early time growth	Nonlinear model→ for I Small, exponential growth $I(t) \approx I_0 e^{(\beta-\gamma)t}$ derived by approximation.	Directly given by linearized eigen values $\lambda = \beta - \gamma$
Information from eigenvectors	Not directly used in many epidemiological interpretations.	Eigen vectors show model directions (which linear combinations of S, I, R grow/decay useful for Moc reduction
Global vs local	Can describe entire epidemic cum a (peak, size final size relation)	Only local behaviour (near DFE or epidemic)- cannot predict final size without nonlinear analysis.



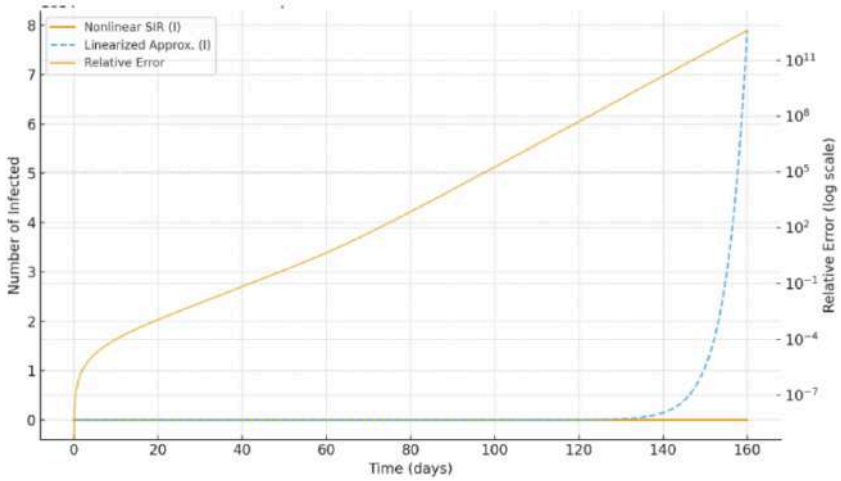
Graph 1: Nonlinear vs Linearized SIR Infection Curve.



Graph 2: Relative Error of Linearized SIR Approximation.

equilibria which is mentioned in the below Graphs **1** and **2**. the conclusions about peak incidence and final size require retaining nonlinear effects or numerical simulation.

The combined Graph **3** presents the three important views of how an epidemic behaves under the different assumptions. The first curve clearly defines the actual infection pattern predicted by the SIR model, where the spread slows down naturally as more the people become infected and gets recover. The second curve, also defines the linearized approximation, assumes the conditions remain the same as at the beginning of the outbreak, so it predicts a much faster and continuously growing number of infections. This helps us understand how the epidemic would grow if nothing changed over time. The third curve, the relative error line, highlights the increasing difference between the realistic model and the simplified one as time passes. Together, these curves show that the linearized approach is useful only in the early stage of an outbreak, while the nonlinear



Graph 3: SIR Dynamics vs Linear Algebraic Approximation.

SIR model gives a more accurate picture as the epidemic progresses.

Graph **1** compares the infection curve obtained from the nonlinear SIR model with the corresponding linearized approximation. The results demonstrate close agreement during the early stages of the epidemic. However, as time increases, the nonlinear model exhibits saturation effects due to the depletion of susceptible individuals, whereas the linearized solution continues to predict exponential growth.

Graph **2** illustrates the relative error between the nonlinear and linearized infection trajectories. The error remains small during the initial phase of disease transmission but increases over time as nonlinear effects become dominant. This behaviour confirms that the linear approximation is most accurate near the disease-free equilibrium.

Graph **3** presents a combined comparison of the nonlinear infection curve, the linearized approximation, and the corresponding relative error. Together, these results demonstrate that linear algebraic methods provide accurate information regarding local stability and early epidemic growth, while the full nonlinear model is required for predicting long-term epidemic behaviour, peak prevalence, and final epidemic size.

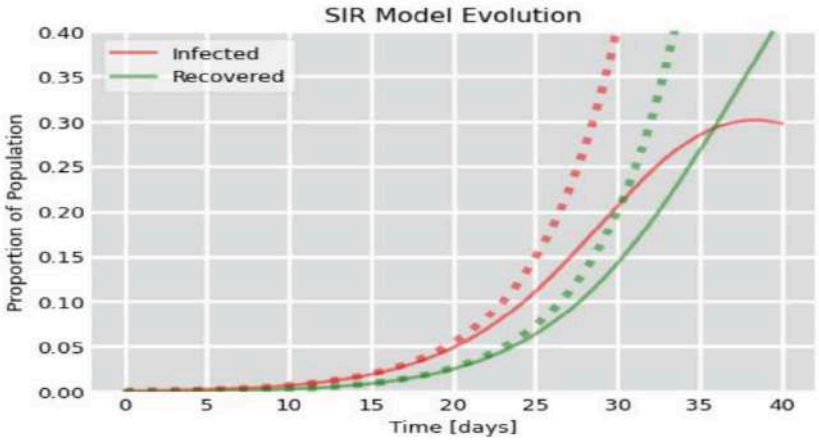
10. MEDICAL APPLICATIONS AND HEALTHCARE PLANNING IMPLICATIONS"

The linear algebraic formulation of the SIR model provides a useful framework for analysing epidemic thresholds, local stability properties, and early-stage disease transmission dynamics. The eigenvalue-based approach developed in this study allows rapid assessment of whether an infectious disease is likely to grow or decline following its introduction into a susceptible population. Such information can support public health decision-making and epidemic preparedness planning that extends beyond theoretical analysis to practical medical and public health applications. By utilizing eigenvalue-based stability analysis, the model enables early identification of epidemic growth or decline, which is crucial for timely intervention in infectious disease outbreaks. In particular, the sign and magnitude of the dominant eigenvalue offer a direct indication of whether an infection will spread exponentially or gradually diminish, thereby serving as an early warning indicator for emerging epidemics.

From a healthcare planning perspective, the ability to estimate infection growth rates and stability conditions has significant implications for resource management. During the early stages of an outbreak, healthcare systems often face uncertainty in predicting patient load. The proposed framework allows for approximate estimation of peak infection levels and growth trends, which can assist in planning hospital capacity, including the allocation of beds, intensive care units (ICUs), medical staff, and essential supplies such as oxygen and medications. This predictive capability is particularly valuable in preventing healthcare system overload and ensuring timely medical response.

Furthermore, the model provides insights into epidemic threshold conditions, particularly through the interpretation of the basic reproduction number. This information can be effectively used to design and evaluate intervention strategies such as vaccination programs, social distancing measures, and quarantine policies. By identifying the critical thresholds required to control disease spread, public health authorities can implement targeted strategies to reduce transmission rates and stabilize the epidemic.

In addition, the matrix-based structure of the model offers flexibility for integration with real-world epidemiological data and computational tools. It can be extended to incorporate demographic variations,



Graph 4: Comparison between the nonlinear SIR infection curve and the linearized exponential growth approximation in the early stage of an epidemic.

Table 2: Medical Interpretation of SIR Model Parameters and their Relevance in Healthcare Planning and Epidemic Control

Parameter /Concept	Mathematical Meaning	Medical Interpretation	Practical Health Care Applications
S(t) (Susceptible)	Individuals at risk of infection	Healthy population vulnerable to disease	Target group for vaccination and prevention
I(t) (Infectious)	Number of infected individuals	Active patients capable of transmission	Isolation, treatment, and monitoring
R(t) (Recovered)	Recovered or removed individuals	Immune or non-infectious population	Indicator of herd immunity development
β (Transmission Rate)	Rate of disease spread	Contact rate between individuals	Control via social distancing, masks, lockdown
γ (Recovery Rate)	Rate of recovery	Speed of patient recovery	Improve treatment protocols, reduce hospital stay
$R_0 = \beta/\gamma$	Basic reproduction number	Average secondary infections caused by one patient	Determine vaccination coverage and control strategies
Eigenvalues	Stability of the system	Growth or decline of infection	Early outbreak detection and warning system
Peak of I(t)	Maximum infection level	Maximum patient load	Hospital capacity and ICU planning

contact patterns, and disease-specific parameters, thereby enhancing its applicability in diverse medical scenarios. This opens opportunities for combining the proposed framework with modern data-driven approaches, including machine learning techniques, for real-time epidemic forecasting and decision support systems.

Overall, the linear algebraic approach to the SIR model serves as a bridge between mathematical theory and practical healthcare applications. It provides a simplified yet powerful analytical tool that supports disease prediction, intervention planning, and healthcare system preparedness, thereby contributing to more effective management of infectious disease outbreaks which is described in Graph **4** and Table **2**.

CONCLUSION

In conclusion, the extended SIR model formulated through the linear algebraic approach which presents a more realistic understanding of epidemic dynamics by incorporating natural birth and death rates. Unlike the basic SIR model, which assumes a fixed population, this version acknowledges the continuous addition of new susceptible individuals through births and the removal of individuals through deaths. As a result, the model better reflects real-world population behaviour and provides a deeper understanding of how infections can persist or stabilize over time rather than fade out completely.

The use of a linear algebraic framework offers a simplified yet powerful way to analyse the interdependence between the susceptible, infected, and recovered groups. It enables the identification of equilibrium points and helps in predicting long-term disease trends. Overall, this approach strengthens the theoretical foundation of epidemiological modelling and offers a valuable tool for studying and controlling infectious disease in populations undergoing demographic changes.

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