Vaccination Dynamics and Stability Insights: A SIR Model Approach to Epidemic Control

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ABSTRACT
In this paper, the standard susceptible, infectious, recovered (SIR) model of epidemic dynamics is considered to examine how vaccination in newborn affects equilibrium stability and infectious spread dynamics. This work includes the analysis of local and global stability at disease-free and endemic equilibrium points. Analytical and numerical methods are employed to demonstrate the increase in vaccination rate reduce infectious disease transmission. Our work addresses a literature gap and leads to better epidemic control methods. An in-depth investigation of the model's local and global stability will help policymakers and health workers build immunization plans by revealing how the illness behaves in different situations. In summary, this work provides a straightforward, brief, and informative analysis of epidemic mobility with vaccination, essential for optimizing vaccination tactics to manage epidemics.

Keywords : Vaccination, Stability analysis, Disease transmission dynamics, Newborn vaccination impact, Equilibrium point analysis, Local stability and Global stability.

1. INTRODUCTION

1.1 Background and Motivation

The main strategy for containing, or managing epidemics has been vaccination, which has been based on the burden of infectious diseases. An epidemiological model assists us in forecasting epidemic dynamics and developing immunization programs when combined with the introduction of new illnesses (and the re-emergence of established ones). The SIR (containing populations susceptible, infectious, and recovered) model provides mathematical evidence for this. It can assist in tracking the spread of infectious diseases throughout the community based on these basic populations.

This study motivated us to refine these models in light of new challenges and the increasing need for predictive tools that span the spectrum of human disease transmission from basic infection mechanisms through the influence of laboratory intervention technology and behavioural interventions to develop informed public health policies. Despite the considerable successes of vaccination technologies and widespread implementation, many essential aspects of how vaccination strengthens or destabilizes the dynamics of a disease within a population remain hidden in the black box of disease transmission, recovery rates, and immunity levels, the components of all models.

Public health crises have long been the driving force behind infectious disease epidemiology. Classical models, such as those first proposed by W. O. Kermack and A. G. McKendrick [1] in 1927 and described by their SIR model, have greatly aided in our understanding of disease transmission and control. These models form the foundation of modern epidemiological modelling.

Consequently, there is a widespread recognition that we should pair vaccination strategies with epidemic models to experimentally verify their effects. This could apply to diseases like measles, polio, and influenza. As disease dynamics became more complex, such as variable vaccination coverage, nonlinear incidence rates, and stochastic effects [6], mathematical modelling became more and more sophisticated, eventually becoming critical for guiding public health responses to the COVID-19 pandemic.. We use stability analysis to determine the eventual elimination of the infectious disease in the presence of vaccination, provided the disease-free equilibrium remains stable. Even more important, it's how we make vaccine dosage decisions to maximize their effectiveness and predict the long-term results of vaccination programs.

We accomplish this by incorporating vaccination into the SIR model, which provides a local and global stability at the equilibrium points of infection dynamics. Additional encouragement comes from recent

worldwide outbreaks of diseases, which have underscored the necessity of predicting the evolution of an epidemic and the effectiveness of vaccination coverage in attempting to achieve herd immunity. A better appreciation of the stability of the SIR model under vaccination strategies could lead to constructive public health policies that can flexibly adapt and adapt to emerging and existing natural phenomena such as infectious diseases. This study contributes to the global effort of mitigating and preventing diseases by characterizing the impact of vaccination coverage on the evolution of disease spread and the asymptotic states of the population using mathematical modelling and simulation.

1.2 Literature Review

Studying the basic dynamics of infectious disease using mathematical models has a long history, going back to 1927 paper by the British mathematicians William Kermack and Anderson Grey McKendrick [1], who first described the classic SIR model of epidemics as a system of ordinary differential equations to understand the basic mechanisms of epidemic spread and control. Bayley [2] later applied mathematical theory to a broad range of infectious diseases, resulting in a framework known as the modelling of infectious diseases. Anderson and May [3] rounded out the topic with a two-part series exploring the population biology of infectious diseases, exhibiting the impact of various epidemiological parameters on disease dynamics. The framework for using mathematics in biology was formed by Kapoor [4], who provided an overview of the integration of mathematical modelling into biology and medicine

More recently, Appa Rao D, Kalesha Vali S et.al [9, 10, 14] worked extensively on epidemic models, discussed and analyzed the stability at the equilibrium points. Divya Kumari G, Kalesha Vali et.al, discussed stability analysis of SIR epidemic model under vaccination coverage on newborns with time delay on susceptible and infected individuals [15], Many researchers contributed their work on various epidemic models like SIRS and SIRI etc. Appa Rao D, Kalesha Vali et.al [11, 12, 13] worked on SIRS epidemic models with non linear incidence rates and Kanaka Mahalakshmi E, Kalesha Vali et.al, discussed stability analysis of SIRI model with reintroduced susceptible [16].

1.3 Objectives of the Study

The primary objective of this study is to analyse the stability of the SIR epidemic model under varying levels of vaccination coverage on newborns. By examining both local and global stability at equilibrium points, we aim to determine the conditions under which vaccination on newborns can effectively control the spread of the disease and potentially eradicate it specifically, we seek to:

1. Determine the disease-free equilibrium (DFE) and endemic equilibrium (EE) points.
2. Analyse the local stability at the DFE and EE points using the Jacobian matrix and eigen
3. value analysis.
4. Investigate the global stability of the endemic equilibrium using Lyapunov functions.
5. Carry out numerical simulations to support the analytical findings and explore the impact of different vaccination rates on disease dynamics.
6. Compare disease dynamics with and without vaccination interventions

2. Mathematical Formulation of the SIR Model with Vaccination

The SIR model is a foundational framework in epidemiology, widely utilized to comprehend and forecast the dynamics of infectious disease spread within a population. The model developed by Kermack and McKendrick in 1927, categorizes the population into three compartments: Susceptible (S), Infective (I), and Recovered (R). The susceptible compartment includes individuals who have not yet contracted the disease but are at risk of infection. These individuals transition to the infective compartment upon exposure to the pathogen. The infective compartment comprises individuals who have been infected and are capable of transmitting the disease to susceptible individuals. The size of this compartment varies based on the number of new infections and the rate at which infected individuals recover or die. The recovered compartment consists of individuals who have either recovered from the infection or gained immunity due to vaccination to the newborns. These individuals are no longer susceptible to the disease and cannot transmit it to other.

In this section, various steps which are followed to build mathematical formulation of SIR epidemic model under vaccination coverage on newborns. These steps include (1) Model assumptions (2) Model diagram or flowchart, description of model variables and parameters (3) Formulation of model equations.

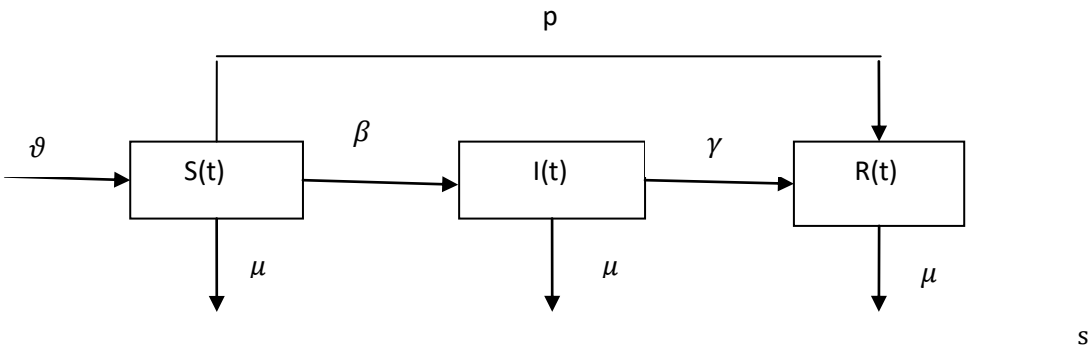
2.1 Model Assumptions

In this section, the following assumptions were made to develop the model with vaccination on newborns.

(a) The total human population is divided into three compartments susceptible (S), infectious (I), and removable / recovered (R)
(b) The total population N is constant with respect to births and deaths over time, that is, the sum of the individuals in the three compartments does not change
(c) Humans are recruited into susceptible class with birth rate.
(d) Humans of all classes will die with death rate due to unrelated background mortality
(e) Susceptible humans if interacted with infected humans will become infected and will go to infected class.
(f) Newborn susceptible humans if vaccinated will go to recovered/removable class.
(g) Some exposed humans having sufficient natural immunity will recover from the infection naturally and will go to recovered class.
(h) Humans of infected will die with disease induced death rate.

2.2 Model diagram

Based on the model assumptions listed in section 2.1, the model diagram or model flowchart is drawn as shown in Figure 1. This flow chart describes the flow of humans among the model compartments.



s

Figure 1. Flowchart showing flow of humans among the model compartments

The model variables and parameters description is detailed in the tables 1 and 2.

Table 1. Description of model variables

Variable	Description
S(t)	Susceptible population size at time t
I(t)	Infected population size at time t
R(t)	Recovered population size at time t

Table 2. Description of model parameters

Parameter	Description
ϑ	Rate of individuals joining susceptible class or birth rate
β	Rate of transmission of infection or infection rate
γ	Rate of the infected individual recover or recovery rate
μ	Rate of death which is the loss of the individual due to unrelated background mortality or death rate
p	Proportion of vaccination on newborn or vaccination rate

2.3 Model equations

Based on the model assumptions, model flow chart and description of model variables and parameters, the system of model equations is constructed and presented in (1). The system is a group of three nonlinear ordinary differential equations.

$$\frac{dS}{dt} = \vartheta - p\vartheta - \frac{\beta SI}{N} - \mu S,$$
$$\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I - \mu I,$$
$$\frac{dR}{dt} = \gamma I - \mu R + \vartheta p \qquad \text{for } 0 \leq p \leq 1 \qquad (1)$$

The equation representing the rate of change in the susceptible population is obtained as ϑ represents the birth rate, introducing new individuals into the susceptible population, the term $p\,\vartheta$ represents the fraction of new-borns that are vaccinated immediately after birth and thus move directly to the recovered compartment, β being the transmission rate, the term $\frac{\beta SI}{N}$ models the rate at which susceptible individuals become infected through contact with infective individuals and μS represents the natural death of susceptible individuals.

The equation representing the rate of change in the infected population is due to $\frac{\beta SI}{N}$, the rate at which susceptible individuals become infected, γI, the infective individuals recover and move to the recovered compartment and μI, the natural death of infective individuals.

The equation representing the rate of change in the removed class is because of γI, the individuals recover from infection, μR, the natural death of recovered individuals and pϑ, the fraction of new-borns that are vaccinated and thus enter directly into the recovered compartment, by passing the susceptible stage.

3. Equilibrium points and Stability analysis

The model equations show how the population transitions between compartments over time, influenced by the rates of infection, recovery, and vaccination. By examining these equations, researchers can gain insights into how different factors affect the spread of the disease and the effectiveness of vaccination programs in controlling epidemics. Understanding the equilibrium points of the model is crucial for determining the long-term behaviour of the disease within the population. An equilibrium point is a state where the number of individuals in each compartment remains constant over time. The disease-free equilibrium (DFE) occurs when there are no infective individual in the population, while the endemic equilibrium (EE) represents a state where the disease persists at a constant level. Local stability analysis assesses the behaviour of the system in the vicinity of the equilibrium points. This is done by linearizing the system around the equilibrium points and analyzing the Jacobian matrix. Global stability analysis ensures that the system will return to the equilibrium state from any initial condition within the state space. This is typically assessed using Lyapunov functions, which are scalar functions that decrease over time and serve as an energy-like measure for the system.

In this section we identify equilibrium points viz, disease free and endemic equilibrium points and discuss local stability at its equilibrium points and global stability at endemic equilibrium point.

3.1 Steady state / Equilibrium points

Disease-free and endemic equilibrium points are obtained by solving the equations in (1) after equating the equations individually to zero that is,

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

The equilibrium points are,

E_1: Disease-free equilibrium point $E_1(S^*, I^*, R^*)$ is $\left(\frac{\vartheta - p\vartheta}{\mu}, 0, \frac{\vartheta p}{\mu}\right)$ (2)

E_2: Endemic equilibrium point $E_2(S^*, I^*, R^*)$ is $\left(\frac{(\mu+\gamma)N}{\beta}, \frac{(\vartheta - p\vartheta - \mu S^*)N}{\beta S^*}, \frac{\gamma I^* + \vartheta p}{\mu}\right)$ (3)

Here the basic reproduction rate (the number of new infections that an each infected individual generates on an average) R_0 is $\frac{\beta}{(\mu+\gamma)}$ and the growth of the population is $N^* = \frac{\vartheta}{\mu}$.

If the basic reproduction rate, less than unity, the disease cannot spread in the population and therefore only the susceptible population remains. And if the basic reproduction rate is greater than unity, infection increases and confine with endemic equilibrium point [11].

In the steady state, the growth of population is $N^* = \frac{\vartheta}{\mu}$, then the equilibrium point is,

$$E_2(S^*, I^*, R^*) = \left(\frac{\vartheta}{\mu R_0}, \left(\frac{\vartheta}{\mu}\right)\left(\frac{\mu}{(\mu+\gamma)}\right)\left(1 - p - \frac{1}{R_0}\right), \frac{\vartheta}{\mu} - S^* - I^*\right)$$

The fractions of the populations (susceptible, infected, and recovered) derived from the endemic equilibrium point are

$$\frac{S^*}{N^*} = \frac{1}{R_0}, \frac{I^*}{N^*} = \left(\frac{\mu}{(\mu+\gamma)}\right)\left(1 - p - \frac{1}{R_0}\right), \frac{R^*}{N^*} = 1 - \frac{S^*}{N^*} - \frac{I^*}{N^*}$$

The fraction of the susceptible individuals $\left(\frac{S^*}{N^*}\right)$ is independent of vaccination coverage. But the fraction of infected individuals $\left(\frac{I^*}{N^*}\right)$ depends on 'p' which is inversely proportional and reaches a point of elimination with increasing vaccination. The point at which the threshold coverage of vaccination needed to eliminate infection is called 'Critical vaccination coverage' p_c and is $1 - \frac{1}{R_0}$. The percentage of the population that

must receive vaccinations in order to eradicate infection rises with the basic reproduction number. Even if not every member of the public receives vaccinations, an epidemic disease can be contained. Herd immunity refers to a population's ability to fade off infection as a result of increased immunity or immunization rates.

3.2 Local stability at disease free equilibrium (DFE) point

Theorem: At the DFE point E_1, the system is locally asymptotically stable provided, $\frac{\beta S^*}{N} < (\mu + \gamma)$

Proof: The variational matrix for the system (1) at the DFE $E_1(S^*, I^*, R^*)$ is given by

$$J = \begin{bmatrix} -\mu & -\beta\frac{\vartheta(1-p)}{N\mu} & 0 \\ 0 & \beta\frac{\vartheta(1-p)}{N\mu} - (\mu+\gamma) & 0 \\ 0 & \gamma & -\mu \end{bmatrix} \qquad (4)$$

The characteristic equation of (4) given by $|J - \lambda I| = 0$, where λ is a parameter is

$(\mu + \lambda)^2 \left[\beta\frac{\upsilon(1-p)}{N\mu} - (\mu+\gamma) - \lambda\right]=0$

The roots of the characteristic equation are $\lambda_1 = -\mu, \lambda_2 = -\mu, \lambda_3 = \frac{\beta S^*}{N} - (\mu+\gamma)$.

Here, the roots λ_1, λ_2 are negative and the root λ_3 is negative if $\frac{\beta S^*}{N} < (\mu+\gamma)$.

Thus, at DFE point $E_1(S^*, I^*, R^*)$, the system is asymptotically stable provided $\frac{\beta S^*}{N} < (\mu+\gamma)$.

3.3 Local stability at endemic equilibrium (EE) point

Theorem: At the EE point $E_2(S^*, I^*, R^*)$, the system is locally asymptotically stable provided, $I^* > S^*$

Proof: The variational matrix for the system (1) at EE point $E_2(S^*, I^*, R^*)$ is

$$J = \begin{bmatrix} -\frac{\beta I^*}{N} - \mu & -\beta\frac{S^*}{N} & 0 \\ \beta\frac{I^*}{N} & \beta\frac{S^*}{N} - (\mu+\gamma) & 0 \\ 0 & \gamma & -\mu \end{bmatrix} \qquad (5)$$

The characteristic equation of (5) is given by $|J - \lambda I| = 0$, where λ is a parameter is,

$$(\mu+\lambda)\left(\lambda^2 - \lambda\left(\frac{\beta S^*}{N} - \frac{\beta I^*}{N} - 2\mu - \gamma\right) - \left(\frac{\beta S^*\mu}{N} - \frac{\beta I^*\mu}{N} - \frac{\beta I^*\gamma}{N} - \mu^2 - \mu\gamma\right)\right) = 0 \qquad (6)$$

Which implies $(\lambda + \mu) = 0$ and

$$\left(\lambda^2 - \lambda\left(\frac{\beta S^*}{N} - \frac{\beta I^*}{N} - 2\mu - \gamma\right) - \left(\frac{\beta S^*\mu}{N} - \frac{\beta I^*\mu}{N} - \frac{\beta I^*\gamma}{N} - \mu^2 - \mu\gamma\right)\right) = 0$$

From equation (6) we have $\lambda = -\mu$ and

$$\left(\lambda^2 + \lambda\left(\frac{\beta}{N}(I^* - S^*) + 2\mu + \gamma\right) + \left(\frac{\beta\mu}{N}(I^* - S^*) + \frac{\beta I^*\gamma}{N} + \mu(\mu+\gamma)\right)\right) = 0 \qquad (7)$$

It is clear that one of the roots of the equation (6) is negative and two of the roots of the equation (7) are negative if sum of the roots of (7) (trace of the matrix) is negative and product of the roots of (7) (determinant of the matrix) is positive.

Here the trace $-(\frac{\beta}{N}(I^* - S^*) + 2\mu + \gamma)$ is negative if $I^* > S^*$and the

determinant, $(\frac{\beta\mu}{N}(I^* - S^*) + \frac{\beta\gamma I^*}{N} + \mu(\mu+\gamma))$ is positive if $I^* > S^*$

Thus, at the endemic equilibrium point $E_2(S^*, I^*, R^*)$, the system is asymptotically stable provided $I^* > S^*$. This implies that the disease will persist in the population at a constant level if the number of infective individuals is greater than the number of susceptible individuals at equilibrium.

3.4. Global stability at endemic equilibrium (EE) point:

Theorem: At the EE point $E_2(S^*, I^*, R^*)$, the system is globally asymptotically stable.

Proof: Let the Lyapunov function be

$$V(t) = (S - S^*)^2 + (I - I^*)^2 + (R - R^*)^2 \qquad (8)$$

Then, $V'(t) = 2(S - S^*)\frac{dS}{dt} + 2(I - I^*)\frac{dI}{dt} + 2(R - R^*)\frac{dR}{dt}$ (9)

By proper choice of

$(1-p)\vartheta - \beta\frac{SI}{N} = \mu S^*, \beta\frac{SI}{N} = (\mu+\gamma)I^*$ and $\gamma I + p\upsilon = \mu R^*$from (1), $V'(t)$

Becomes,

$V'(t) = 2(S - S^*)\mu(S^* - S) + 2(I - I^*)(\mu+\gamma)(I^* - I) + 2(R - R^*)\mu(R^* - R)$

i.e., $V'(t) = -2(S - S^*)^2\mu - 2(I - I^*)^2(\mu + \gamma) - 2(R - R^*)^2\mu < 0$.
Hence, at its EE point, the system is globally asymptotically stable.

4. Numerical Simulation

Numerical simulations are performed to validate the analytical results and to explore the dynamics of the model under various parameter settings. Using software tools like MATLAB, the model equations are simulated with different values of β, γ, p to observe the impact of vaccination on disease spread. Simulations provide visual insights into how changes in vaccination coverage affect the susceptible, infective, and recovered populations over time. Phase portraits and time series plots help illustrate the convergence to equilibrium points and the stability of the system under different scenarios. Despite the extensive research on epidemic models and vaccination strategies, there remains a significant gap in the quantitative understanding of how vaccination affects the temporal stability of disease transmission. Most of the previous studies focused on the basic reproduction number R_0 and the long-term behaviour of the epidemic without delving into the stability conditions under varying vaccination rates. This study addresses this gap by providing a comprehensive analysis of the stability of the SIR model with vaccination, offering insights into the critical vaccination coverage required to achieve herd immunity and control the epidemic. To emphasise the importance of vaccination, numerical examples are considered. Comparative analysis of disease dynamics with and without vaccination interventions also presented for different examples tabulated in Table 3. From the table one can observe that the disease reduces as the proportion of vaccination rate increases.
The simulations aim to observe changes in the susceptible (S), infective (I), and removable (R) populations in response to variations in specific parameters, while keeping other parameters constant. The primary focus is on examining how recovery rate (γ), transmission rate (β), vaccination rate (p) influence the dynamics of the populations. Total of nine examples (labelled as 4.1 to 4.9) are considered to study these effects under vaccination coverage. Each example consists of two types of graphical representations: Time series responses (Figure A) and Phase Portraits (Figure B). These plots show how the populations susceptible, infective, and recovered individuals changes over time and phase portraits provide a phase-space representation of the dynamics, illustrating the trajectories of the system in the (S, I) or (I, R) planes. Time series responses and phase portraits help to visualize the stability and convergence behaviour of the model. For all the examples, S, I, R values are fixed and considered S=50, I=30, R=20 to observe the change in population by varying one at a time of β, γ, p and keeping remaining parameters fixed. The graphs are

Example 4.1: For $\vartheta = 10, \beta = 1, \gamma = 0.5, \mu = 0.1, N = 100, p = 0.1$.

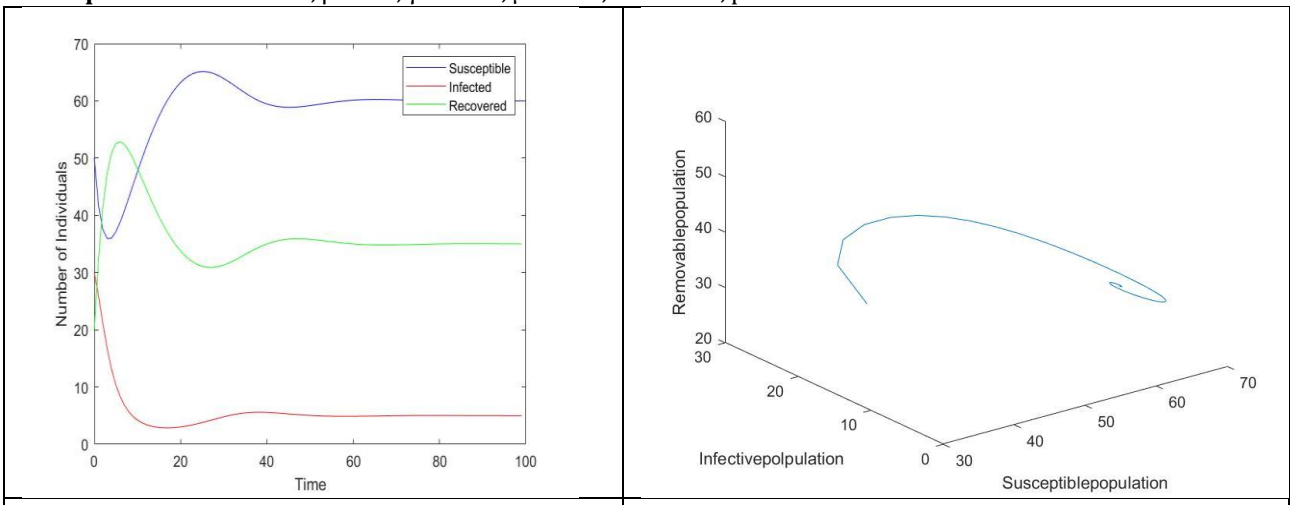


Fig 4.1 - (A) Fig 4.1 - (B)

The system is stable asymptotically and converges to E(60, 5, 35).

Example 4.2: For $\vartheta = 10, \beta = 2, \gamma = 0.5, \mu = 0.1, N = 100, p = 0.1$.

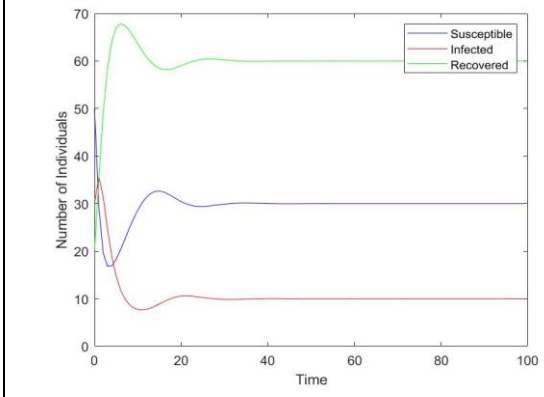


Fig 4.2 - (A)

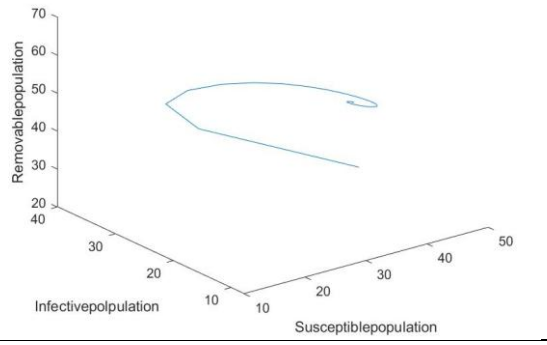


Fig 4.2 - (B)

The system is stable asymptotically and converges to E(30, 10, 60).

Example 4.3: For $\vartheta = 10, \beta = 3, \gamma = 0.5, \mu = 0.1, N = 100, p = 0.1$.

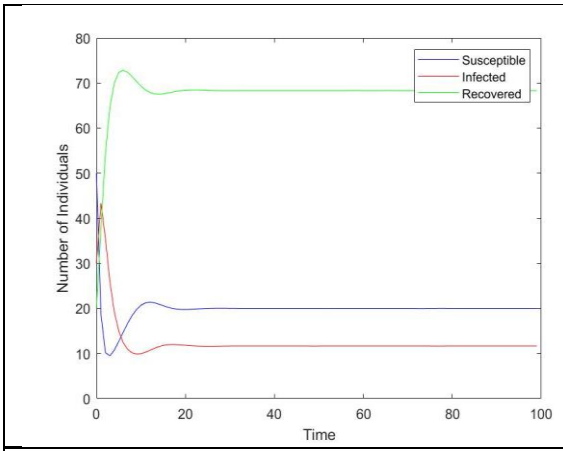


Fig 4.3 - (A)

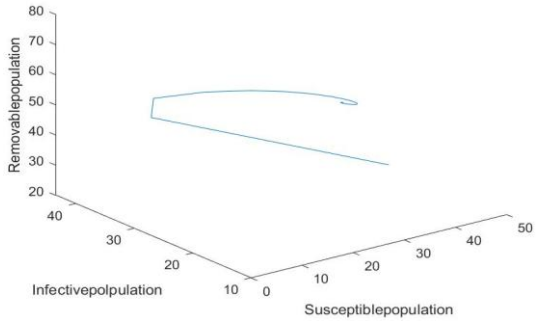


Fig 4.3 - (B)

The system is stable asymptotically and converges to E(20, 12, 68).
Examples 4.1 to 4. 3, illustrates that the system converges to the equilibrium points. Also observed that, infective and removable individuals increases when there is an increase in the transmission rate (β) and remaining parameters are fixed constant.

Example 4.4: For $\vartheta = 10, \beta = 4, \gamma = 1, \mu = 0.1, N = 100, p = 0.1$.

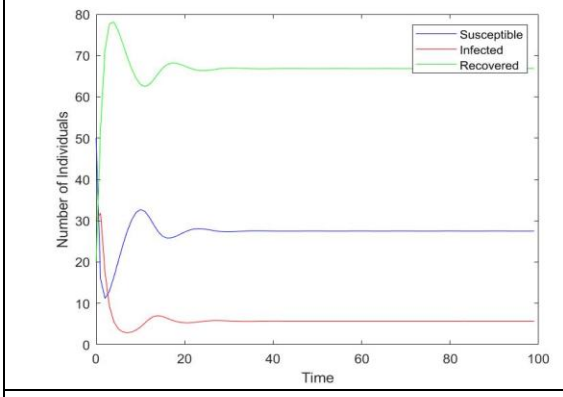


Fig 4.4 - (A)

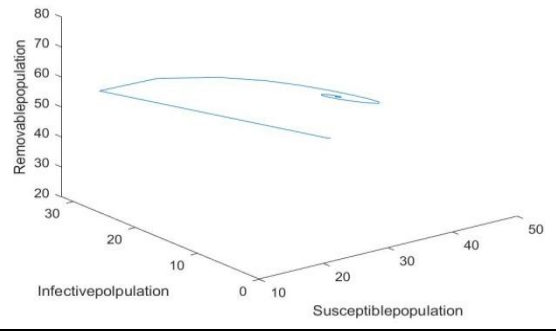


Fig 4.4 - (B)

The system is stable asymptotically and converges to E(27, 6, 67).

Example 4.5: For $\vartheta = 10, \beta = 4, \gamma = 2, \mu = 0.1, N = 100, p = 0.1$.

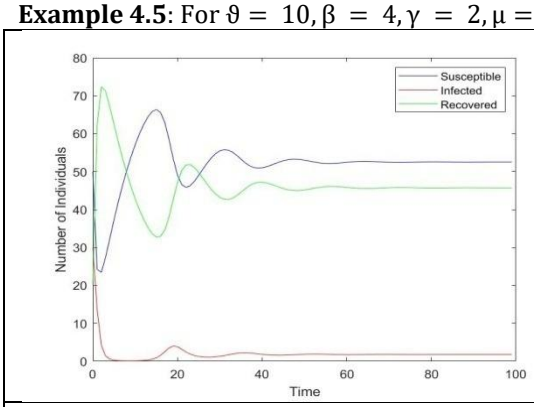


Fig 4.5 - (A)

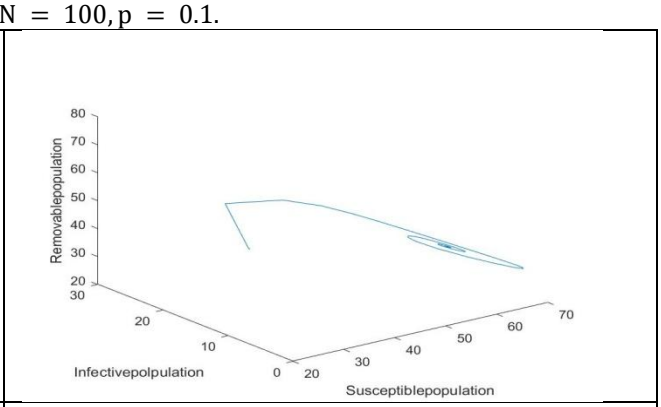


Fig 4.5 - (B)

The system is stable asymptotically and converges to E(52, 2, 46).

Example 4.6: For $\vartheta = 10, \beta = 4, \gamma = 4, \mu = 0.1, N = 100, p = 0.1$.

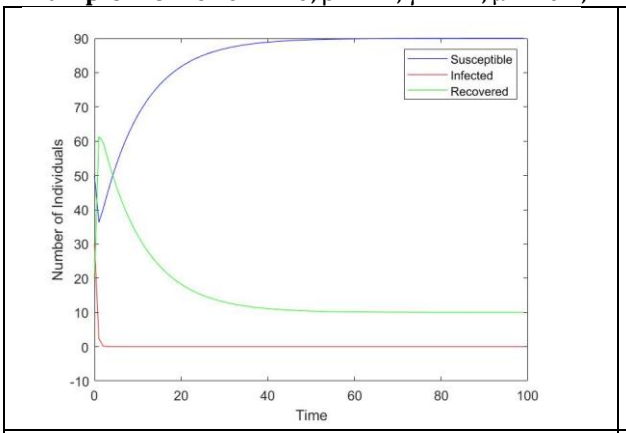


Fig 4.6 - (A)

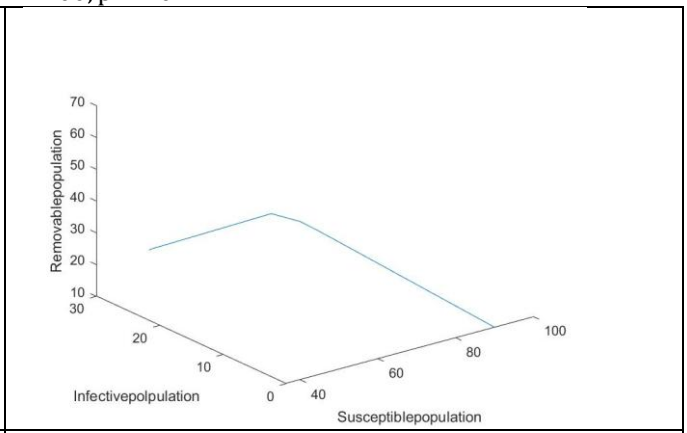


Fig 4.6 – (B)

The system is stable asymptotically and converges to E(90,0,10).
Examples 4.4 to 4.6, illustrates that the system converges to the equilibrium points and also observed that, susceptible individuals increases with the increase in the recovery rate (γ) and the remaining parameters are fixed constant.

Example 4.7: For $\vartheta = 10, \beta = 2, \gamma = 1, \mu = 0.1, N = 100, p = 0.1$.

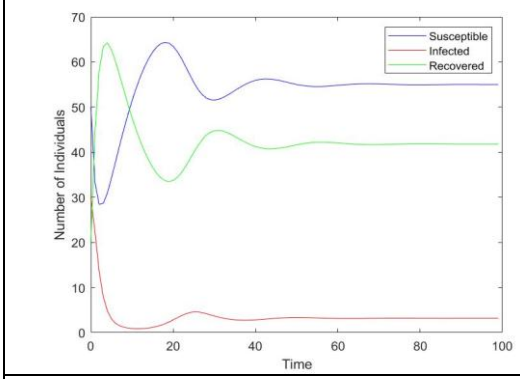


Fig 4.7 - (A)

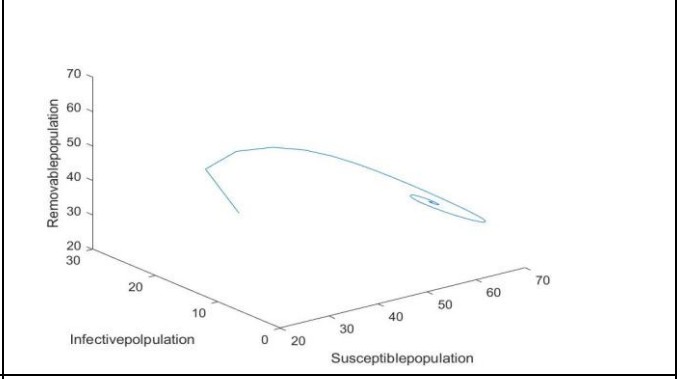


Fig 4.7- (B)

The system is stable asymptotically and converges to E(55, 3, 42).

Example 4.8: For $\vartheta = 10, \beta = 2, \gamma = 1, \mu = 0.1, N = 100, p = 0.4$.

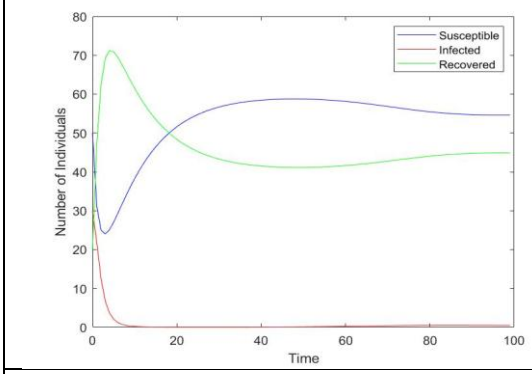


Fig 4.8 - (A)

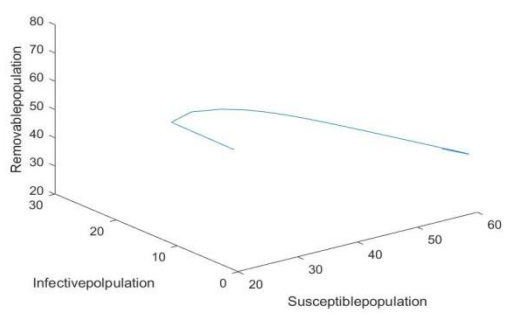


Fig 4.8 - (B)

The system is stable asymptotically and converges to E(55, 0, 45).

Example 4.9: For $\vartheta = 10, \beta = 2, \gamma = 1, \mu = 0.1, N = 100, p = 0.6$.

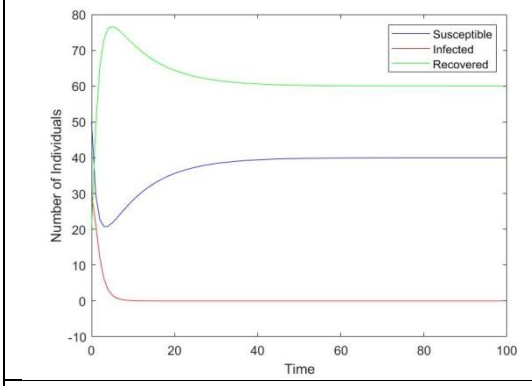


Fig 4.9 - (A)

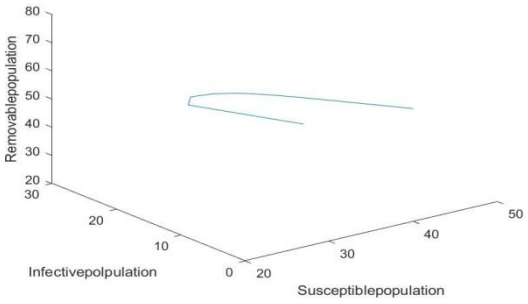


Fig 4.9 - (B)

The system is stable asymptotically and converges to E(40, 0, 60).
Examples 4.7 to 4.9, illustrates that the system converges to the equilibrium points and also observed that, infective individuals almost vanish with the increase in the proportion of vaccination on newborns (p) keeping all the remaining parameters as fixed constant. To compare the disease dynamics with vaccination intervention same numerical examples are considered and numerical simulation is carried out for the model without vaccination (Examples 4.10 - 4.16).

Example 4.10: For $\vartheta = 10, \beta = 1, \gamma = 0.5, \mu = 0.1, N = 100, p = 0$.

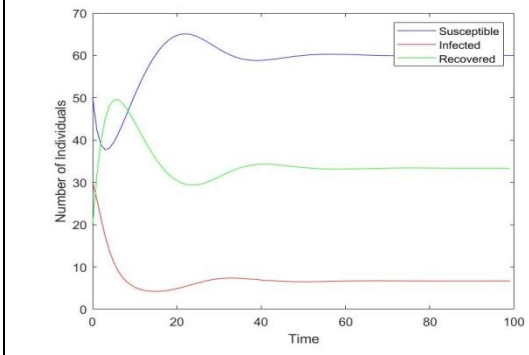


Fig 4.10 - (A)

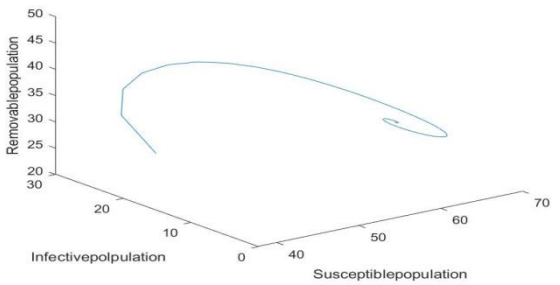


Fig 4.10 - (B)

The system is stable asymptotically and converges to E(60, 7, 33).

Example 4.11: For $\vartheta = 10, \beta = 2, \gamma = 0.5, \mu = 0.1, N = 100, p = 0$.

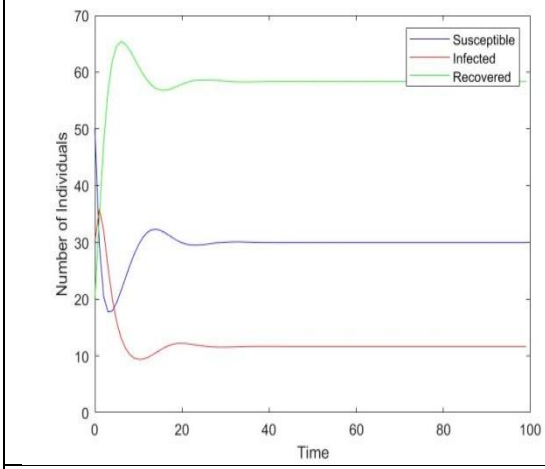


Fig 4.11 - (A)

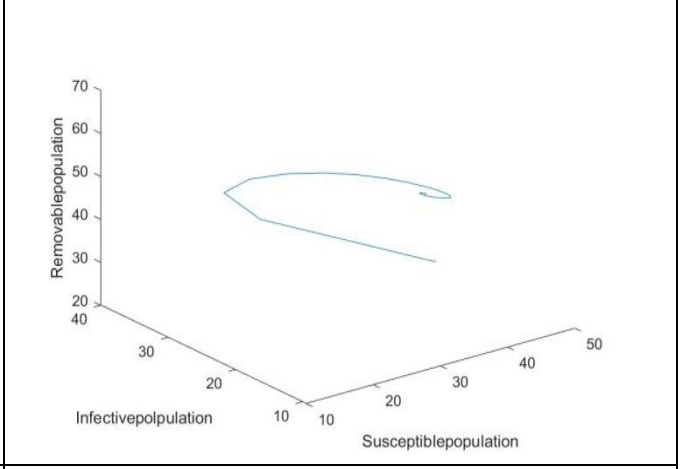


Fig 4.11- (B)

The system is stable asymptotically and converges to E(30, 12, 58).

Example 4.12: For $\vartheta = 10, \beta = 3, \gamma = 0.5, \mu = 0.1, N = 100, p = 0$.

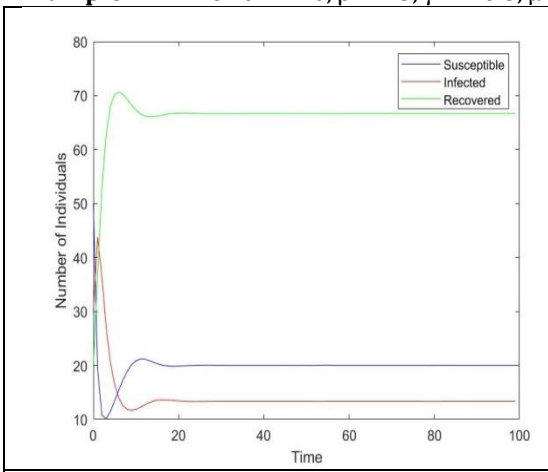


Fig 4.12 - (A)

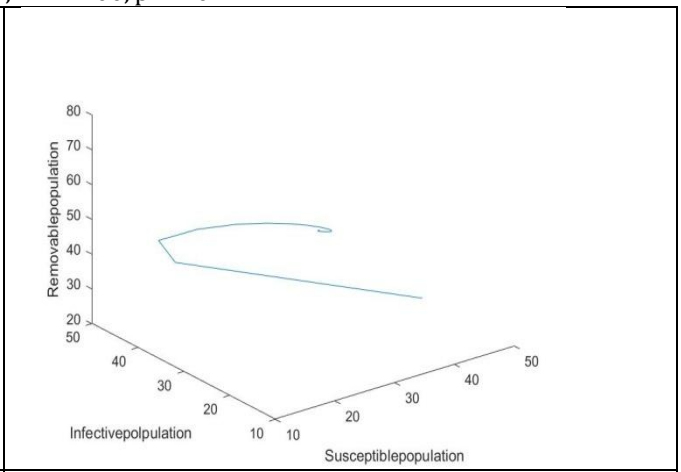


Fig 4.12- (B)

The system is stable asymptotically and converges to E(20, 13, 67).

Example 4.13: For $\vartheta = 10, \beta = 4, \gamma = 1, \mu = 0.1, N = 100, p = 0$.

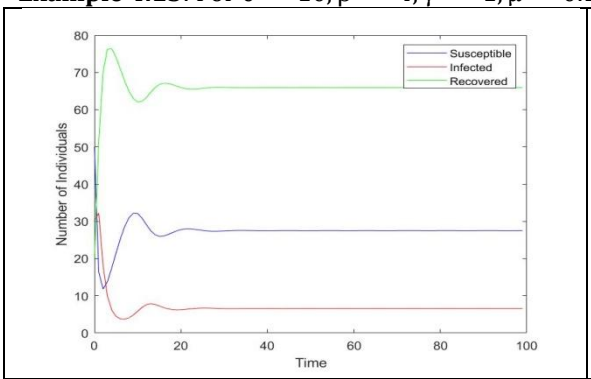


Fig 4.13 - (A)

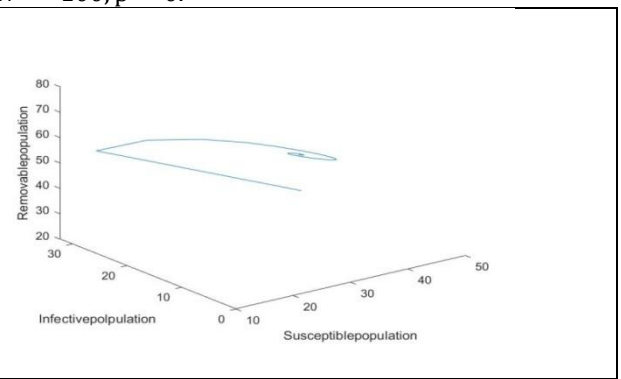


Fig 4.13 - (B)

The system is stable asymptotically and converges to E(27, 7, 66).

Example 4.14: For $\vartheta = 10, \beta = 4, \gamma = 2, \mu = 0.1, N = 100, p = 0$.

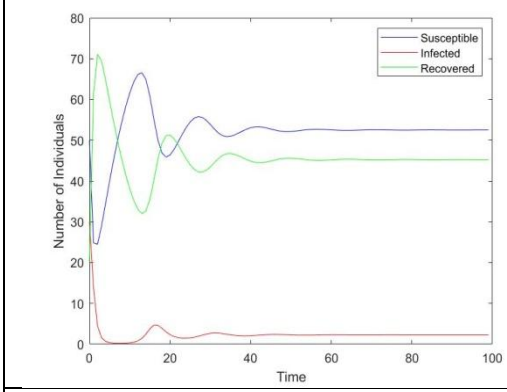


Fig 4.14 - (A)

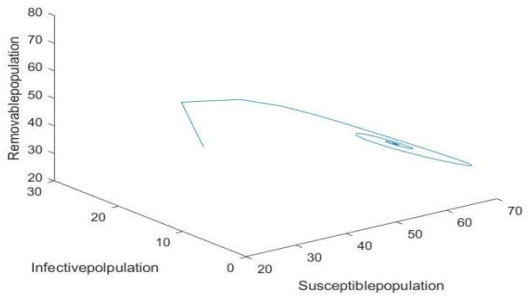


Fig 4.14 - (B)

The system is stable asymptotically and converges to E(52, 3, 45).

Example 4.15: For $\vartheta = 10, \beta = 4, \gamma = 4, \mu = 0.1, N = 100, p = 0$.

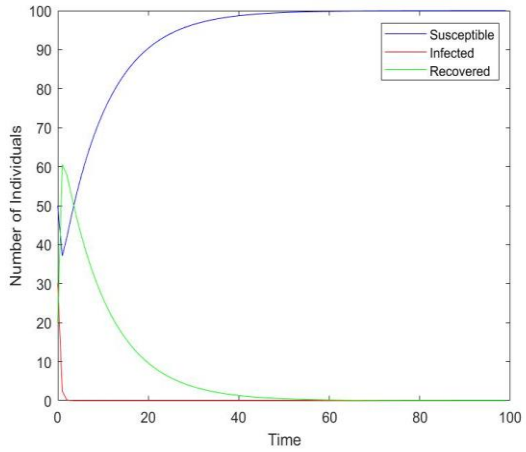


Fig 4.15 - (A)

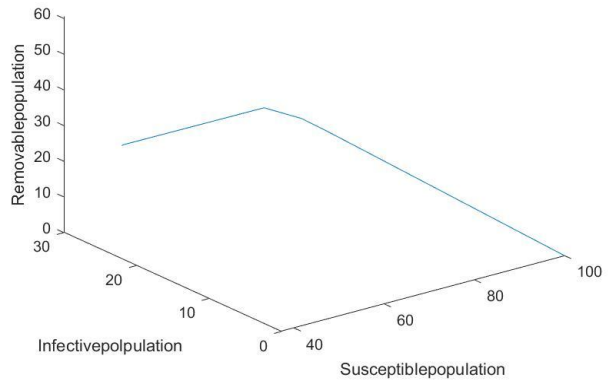


Fig 4.15 - (B)

The system is stable asymptotically and converges to E(100, 0, 0).

Example 4.16: For $\vartheta = 10, \beta = 2, \gamma = 1, \mu = 0.1, N = 100, p = 0$.

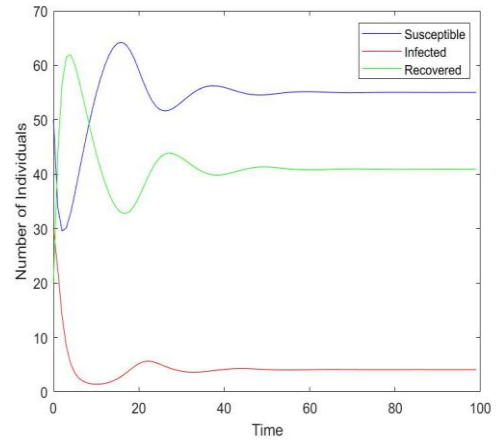


Fig 4.16 - (A)

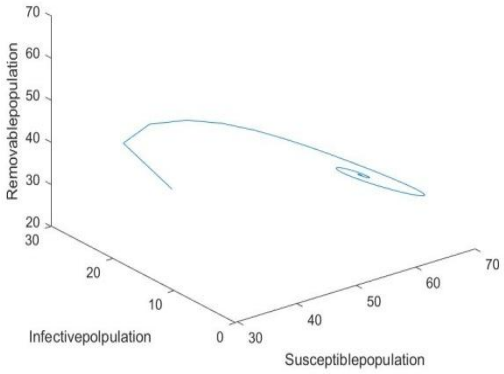


Fig 4.16 - (B)

The system is stable asymptotically and converges to E(55, 4, 41).
Comparative analysis of disease dynamics with and without vaccination interventions presented for different examples are tabulated in Table 3.

Table 3. Comparative analysis of disease dynamics with and without vaccination interventions

S. No	Parameters	Without Vaccination (p=0)	With Vaccination
1	ϑ=10, β = 1, γ = 0.5, μ = 0.1	E(60,07,33)	E(60,05,35) when p=0.1
2	ϑ=10, β = 2, γ = 0.5, μ= 0.1	E(30,12,58)	E(30,10,60) when p=0.1
3	ϑ=10, β = 3, γ = 0.5, μ= 0.1	E(20,13,67)	E(20,12,68) when p=0.1
4	ϑ=10, β = 4, γ = 01, μ= 0.1	E(27,07,66)	E(27,06,67) when p=0.1
5	ϑ=10, β = 4, γ = 02, μ= 0.1	E(52 ,03,45)	E(52,02,46) when p=0.1
6	ϑ=10, β = 4, γ =04, μ= 0.1	E(100,00,0)	E(90,00,10) when p=0.1
7	ϑ=10, β =2, γ = 01, μ= 0.1	E(55,04,41)	E(55,03,42) when p=0.1
8	ϑ=10, β = 2, γ = 01, μ= 0.1	E(55,04,41)	E(55,00,45) when p=0.4
9	ϑ=10, β = 2, γ = 01, μ= 0.1	E(55,04,41)	E(40,00,60) when p=0.6

It is understood from the tabulated values that the increase in the proportion of vaccination, there is a significant downfall in infective population.

5. RESULTS AND DISCUSSIONS

Examined the SIR (containing susceptible, infectious, and removable populations) epidemic model's stability under vaccination coverage with an assumption of total population (N) is constant with respect to births and deaths. The model is represented by nonlinear ordinary differential equations. It is observed that if the basic reproduction rate R_0 is more than one, the system possesses endemic equilibrium point and a disease-free equilibrium point if it is less than one. It is established that the system is globally asymptotically stable at the endemic equilibrium point, and it is locally asymptotically stable if $I^* > S^*$.Whereas the system is locally asymptotically stable at the disease free equilibrium point if $\frac{\beta S^*}{N} < (\mu + \gamma)$. It is observed by the way of numerical simulation that due to increase in the transmission rate (β) , there is an increase in the infective and removable populations .Also due to increase in the recovery rate of the infected individuals (γ) there is an increase in susceptible populations. Also, because of increase in proportion of vaccination of newborn (p) the infective population almost vanish. The system is consistent if $\vartheta = \mu N^*$. If the rate of transmission of the infection β is equal to the recovery rate of the infected individuals γ i.e., $\beta = \gamma$, then the infective population almost vanish. If the rate of transmission of the infection β is less than the recovery rate of the infected individuals γ i.e., $\beta < \gamma$ then there is a significant growth in susceptible population. If the rate of transmission of the infection β is greater than the recovery rate of the infected individual γ i.e., $\beta > \gamma$ then there is a significant growth in infective and removable populations.

6. CONCLUSIONS

The paper examines the SIR model's stability under vaccination coverage to emphasise the importance of vaccination as one of the control methods in infectious disease management. Under certain circumstances, the model demonstrates local and global stability, emphasising the need to reduce transmission or increase recovery in order to limit outbreaks. High vaccination rates could eradicate the disease, demonstrating that vaccination reduces infective cases proportionally.

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