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MATHEMATICAL INVESTIGATION OF A MASS ACTION EPIDEMIC MODEL

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ABSTRACT

A mass action epidemic model incorporating vital dynamics was developed and examined. This type of model is particularly applicable to several childhood illnesses such as Mumps, Rubella, as well as highly contagious diseases like influenza. We demonstrated that the biologically meaningful region—where solutions are feasible—is positively invariant, meaning that any solution starting within this region remains there for all time. The model features two equilibrium points: the disease-free equilibrium (DFE) and the endemic equilibrium. By utilizing the basic reproduction number, R_0, we established that the DFE is locally asymptotically stable when $R_0 < 1$. Additionally, we proved the global stability of the DFE through an appropriately chosen Lyapunov function. We also found that the endemic equilibrium exists only when $R_0 > 1$. Numerical simulations highlighted the critical role of the contact rate in influencing the transmission dynamics within mass action models.

1 INTRODUCTION

The analysis of infectious diseases increasingly relies on mathematical modeling as an essential tool. Its powerful capacity for testing hypotheses and conducting simulations that closely mirror real-world outcomes has made it indispensable in epidemiological research. Infectious disease models have played a significant role in designing and analyzing epidemiological surveys, identifying critical data to be collected, detecting trends, making broad predictions, and assessing the uncertainty associated with forecasts. Despite advances, infectious diseases remain the foremost threat to human health. Historically, the bubonic plague claimed over 20% of Europe's population over seven years in the 14th century, while the Great Plague of London (1664–1666) resulted in the deaths of more than 75,000 of the city's 460,000 inhabitants. The 1918–1919 influenza pandemic caused an estimated 25 million deaths across Europe.

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Although medical progress in the 20th and 21st centuries has improved disease control, it has not provided a universal solution, as diseases like HIV continue to devastate populations worldwide. Since its first identification in 1981, HIV has claimed over 25 million lives, with more than 30 million individuals currently living with the virus

Disease incidence refers to the rate at which susceptible individuals become infected through contact with infectious persons [1], representing the number of new cases per unit time—essentially, how quickly new waves of infection emerge. The choice of incidence function in a mathematical model is crucial since it directly influences the epidemic's dynamics by determining how new infections are generated. The three common incidence functions used in deterministic models are the saturated incidence, the standard incidence, and the mass action incidence.

The mass action incidence is expressed as $\beta \frac{SI}{N}$, where $SI\ and$ denote the number of susceptible and infectious individuals, respectively, and N represents the total population. Here, β is the transmission coefficient, and the term $\frac{SI}{N}$ reflects the number of adequate contacts necessary for disease transmission. This incidence function is most appropriate when the total population size N is not excessively large [2] as it assumes that the contact rate depends on the population size, implying that the frequency of encounters increases with community density. It is a density-dependent model, with the contact rate per infectious individual proportional to the host density. Diseases such as measles, mumps, rubella, chickenpox, polio, and influenza are often modeled using the mass action incidence. Although the choice of incidence function primarily depends on the disease's transmission characteristics, analytical simplicity sometimes favors the use of mass action formulations. Notably, this approach has also been applied in modeling HIV transmission [3],[4], [5].

In [6], researchers examined how the choice of incidence function influences epidemic behavior, particularly in the context of vaccine-induced backward bifurcation in HIV models. Several examples demonstrate that backward bifurcations tend to occur with standard incidence but are absent when using equivalent models based on the mass action incidence.

2 MODEL FORMULATION

In this section, we introduce a mass action epidemic model incorporating vital dynamics within the Susceptible-Infected-Recovered (SIR) framework. We will demonstrate that the solutions of the model remain positive over time and establish their local stability through linearization techniques

2.1 Model Variables

The variables used in the model are defined as follows

$S(t)$ - The number of susceptible individuals at time t

$I(t)$ - The number of infected individuals at time t

$R(t)$ - The number of recovered individuals at time t

2.2. Parameters of the model

The model parameters are defined below

Λ_0 - Birth rate

β - Contact rate

μ - Natural death rate
δ - Disease induced death rate
Γ - Recovery rate per unit time

2.3. Assumptions of the Model

(i) All newborns are susceptible
(ii) Recovered individuals acquire lifelong immunity
(iii) Infected individuals transmit the disease to susceptible and remain infectious for a certain period before transitioning to the recovered class

2.4. The Model Equation

The governing equations for the model are outlined below

$$\frac{ds}{dt} = \Lambda_0 - \beta SI - \mu S$$

$$\frac{dI}{dt} = \beta SI - (\delta + \Gamma + \mu)I \qquad 2.0$$

$$\frac{dR}{dt} = \Gamma I - \mu R$$

2.5. Feasible Solution Region

The biologically meaningful region for the model is defined by the systems parameters and initial conditions.

$$\phi = \left\{(S, I, R) \in \mathbb{R}^{3+} : S + I + R = N \leq \frac{\Lambda_0}{\mu}\right\}$$

It will be demonstrated that this region remains positively invariant under the dynamics of the model.

The total population considering the interactions among susceptible, infected, and recovered individuals is expressed as follows:

$$N = S + I + R$$

That is

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

Therefore, when we add the differential equation (2.0), we have

$$\frac{dN}{dt} = \Lambda_0 - \mu N$$

Integrating

$$\frac{dN}{dt} = \Lambda_0 - \mu N$$

has an integrating factor $e^{\mu t}$.

This gives,

$$\frac{dN}{dt} e^{\mu t} + \mu N e^{\mu t} = \Lambda_0 e^{\mu t}$$

Such that,

$$(Ne^{\mu t})^r \leq \Lambda_0 e^{\mu t}$$

$$\int (Ne^{\mu t})^r \leq \int \Lambda_0 e^{\mu t}$$

$$Ne^{\mu t} \leq \frac{\Lambda_0}{\mu} e^{\mu t} + c$$

Now, when $t = 0$

$$N(0) \leq \frac{\Lambda_0}{\mu} + C$$

$$C \geq N(0) - \frac{\Lambda_0}{\mu}$$

Hence,

$$Ne^{\mu t} \leq \frac{\Lambda_0}{\mu} e^{\mu t} + \left(N(0) - \frac{\Lambda_0}{\mu}\right)$$

$$N \leq \frac{\Lambda_0}{\mu} + \left(N(0) - \frac{\Lambda_0}{\mu}\right) e^{-\mu t}$$

$$N \leq N(0)e^{-\mu t} + \frac{\Lambda_0}{\mu}(1 - e^{-\mu t})$$

As $t \to \infty, N(t) \leq \frac{\Lambda_0}{\mu}$. This implies that any solution starting within the specified region remains there for all future time. i.e for all $t > 0$. Consequently, our model, ϕ is biologically realistic, mathematically well-defined, and maintains its invariance within the region.

3. POSITIVITY OF SOLUTION

We will now demonstrate that all variables in the model described in (2.0) remain non-negative. i.e. $(S, I, R) \geq 0 \ \in \phi$

Lemma 1

Suppose the initial conditions are given; then, the solutions $(S, I, R)(t)$ to model (2.0) stay positive for all time i.e. $t > 0$

Proof

From the first equation of (2.0) assuming

$$\frac{dS}{dt} = \Lambda_0 - \beta SI - \mu S \geq -(\beta I + \mu)S$$

Then,

$$\frac{dS}{dt} \geq -(\beta I + \mu)S$$

Or

$$\frac{dS}{dt} \geq -(\beta I + \mu)dt$$

When we integrate both sides of the inequalities, we have

$$\int \frac{dS}{S} \geq \int -(\beta I + \mu)dt$$

$$lnS(t) \geq -(\beta I + \mu)dt + C$$

$$S(t) \geq ce^{-(\beta I+\mu)dt}$$

Now,

When $t = 0$, we have

$$S(t) \geq S(0)e^{-(\beta I+\mu)dt} \geq 0$$

From the second equation of (2.0), we have

$$\frac{dI}{dt} = \beta SI - (\delta + \Gamma + \mu)I \ \geq \ -(\delta + \Gamma + \mu)I$$

We have

$$\frac{dI}{dt} \geq -(\delta + \Gamma + \mu)I$$

Or

$$\frac{dI}{dt} \geq -(\delta + \Gamma + \mu)dt$$

When both sides of the equation are integrated, it gives

$$\int \frac{dI}{I} \geq -\int (\delta + \Gamma + \mu)dt$$

$$\ln I(t) \geq -(\delta + \Gamma + \mu)t$$

When $t = 0, we\ have$

$$I(t) \geq I(0)e^{-(\delta+\Gamma+\mu)t} \geq 0$$

Since, $(\delta + \Gamma + \mu) > 0$

Next, from the third equation of our model

$$\frac{dR}{dt} = \Gamma I - \mu R$$

This has an integrating factor $e^{\mu t}$, so

$$\frac{dR}{dt}e^{\mu t} + \mu R e^{\mu t} = \Gamma I e^{\mu t}$$

$$(Re^{\mu t})^r = \ \Gamma I e^{\mu t}$$

$$(Re^{\mu t})^r = \frac{\Gamma I}{\mu}e^{\mu t} + c$$

When $t = 0$

$$R(0) = \frac{\Gamma I}{\mu} + c$$

$$R(0) - \frac{\Gamma I}{\mu} = c$$

$$Re^{\mu t} = \frac{\Gamma I}{\mu}e^{\mu t} + \left(R(0) - \frac{\Gamma I}{\mu}\right)$$

$$R(t) = \frac{\Gamma I}{\mu} + \left(R(0) - \frac{\Gamma I}{\mu}\right) e^{-\mu t}$$

$$R(t) = R(0)e^{-\mu t} + \frac{\Gamma I}{\mu}(1 - e^{\mu t} > 0 \; since \; \mu > 0$$

Therefore, all variables are positive for $t > 0$

4. EXISTENCE AND STABILITY OF THE DISEASE FREE EQUILIBRIUM (DFE)

The equilibrium points of the system (SIR) was obtained by equating the rate of change to zero ie

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

From our model, we have

$$\Lambda_0 - \beta SI - \mu S = 0$$

$$\beta SI - (\delta + \Gamma + \mu)I = 0$$

$$\Gamma I - \mu R = 0$$

All the disease state variables are all zeros (ie. The disease dies out or goes into extinction). From the above model only the first equation remains as there is no disease and is reduced to

$$\Lambda_0 - \mu S = 0$$

$$S_0 = \frac{\Lambda_0}{\mu}$$

Therefore, $E_0 = (S_0, 0, 0) = (\frac{\Lambda_0}{\mu}, 0, 0)$

4.1. The Basic Reproduction Number, R_0

In the field of mathematical epidemiology, the concept of the basic reproduction number, denoted by R_0, is fundamental. It quantifies the potential of an infectious disease to spread within a population or to die out after introduction. Specifically, R_0 is defined as the expected number of secondary cases generated by a single infectious individual in a completely susceptible population [7]. This threshold parameter determines the disease dynamics; if the disease-free equilibrium is locally asymptotically stable, then the disease cannot establish itself in the population (i.e. $R_0 < 1$) whereas if the number of infected individuals increases over time, the disease has the capacity to invade and spread i.e. $R_0 > 1$. [8],[9]

The calculation of the basic reproduction number for the system described by equation (2.0) can be derived through the following process

$$F = (\beta S_0)I$$

Where $S_0 = \frac{\Lambda_0}{\mu}$

$$V = (\mu + \delta + \Gamma)$$

$$V^{-1} = \frac{1}{(\mu + \delta + \Gamma)}$$

$R_0 =$ Spectral radius of FV^{-1}

$$R_0 = \frac{\beta S_0}{\mu + \delta + \Gamma}$$

$$R_0 = \frac{\beta \Lambda_0}{\mu(\mu + \delta + \Gamma)}$$

4.2. Local Asymptotic Stability of the Disease Free Equilibrium

We will study the local stability of the disease free equilibrium in this section. This will be determined by Jacobian matrix $J(S, I, R)$ of the equation (2.0).

$$J(S, I, R) = \begin{bmatrix} -\beta I - \mu & \beta S & 0 \\ \beta I & \beta S - (\mu + \delta + \Gamma) & 0 \\ 0 & \Gamma & -\mu \end{bmatrix}$$

$$J\left(\frac{\Lambda_0}{\mu}, 0,0\right) = \begin{bmatrix} -\mu & \beta \frac{\Lambda_0}{\mu} & 0 \\ 0 & \beta \frac{\Lambda_0}{\mu}(\mu + \delta + \Gamma) & 0 \\ 0 & \Gamma & -\mu \end{bmatrix}$$

The given eigen values are

$$\lambda_1 = \lambda_3 = -\mu \; and \; \lambda_2 = \beta \frac{\Lambda_0}{\mu}(\mu + \delta + \Gamma)$$

$$\lambda_1, \lambda_3 < 0, \lambda_2 < 0 \; if \; \beta \frac{\Lambda_0}{\mu} - (\mu + \delta + \Gamma) < 0$$

Alternatively $\beta \frac{\Lambda_0}{\mu} < (\mu + \delta + \Gamma)$ or $\frac{\beta \Lambda_0}{\mu(\mu+\delta+\Gamma)} < 0$ or $R_0 < 1$

Thus, the following theorem has been proved.

Theorem 1: Local Asymptotic Stability of E_0
The disease free equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$

GLOBAL ASYMPTOTIC STABILITY OF THE DISEASE FREE EQUILIBRIUM

Let's consider the Lyapunov function L=I

$$L = I = \beta SI - (\delta + \Gamma + \mu)I$$

$$(\beta S - (\delta + \Gamma + \mu)I \leq \quad \beta S_0 - (\delta + \Gamma + \mu)I$$

$$= \beta \frac{\Lambda_0}{\mu} - (\delta + \Gamma + \mu)I$$

$$= (\delta + \Gamma + \mu)(R_0 - 1)I \leq 0 \quad \text{for} \quad R_0 < 1$$

Given that all model parameters are positive, it follows that for; the lyapunov function is valid only if the condition holds. Consequently, this function serves as a lyapunov function as the domain D. Applying La Salle's invariance principle; we conclude that all solutions to the system described by equation (2.0), with initial conditions within D, tend to the equilibrium point as $t \to \infty$. Hence, we have established the following theorem.

Theorem 2: Global Asymptotic Stability of E_0

If $R_0 < 1$, the disease free equilibrium of system (2.0)is globally asymptotically stable.

5. EXISTENCE OF ENDEMIC EQUILIBRIUM

The persistence of the disease in the population will be investigated in this section.

All the model equations equal zero at equilibrium, that is

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

Or

$$\begin{aligned} \Lambda_0 - \beta SI - \mu S &= 0 \\ \beta SI - (\delta + \Gamma + \mu)I &= 0 \qquad 2.1 \\ \Gamma I - \mu R &= 0 \end{aligned}$$

Taking the second equation of (2.1) gives

$$\beta SI = (\delta + \Gamma + \mu)I$$

Alternatively, $S^* = \frac{(\delta+\Gamma+\mu)}{\beta}$

From the first equation of (2.1), we have

$$\Lambda_0 - \beta SI - \mu S = 0$$

Or

$$\Lambda_0 - (\delta + \Gamma + \mu)I - \mu\frac{(\delta + \Gamma + \mu)}{\beta} = 0$$

$$I^* = \frac{\beta\Lambda_0 - \mu(\delta + \Gamma + \mu)}{\beta(\delta + \Gamma + \mu)}$$

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

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

From the third equation of (2.1)

$$\Gamma I = \mu R$$

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

Thus, the lemma below has been proven.

***Lemma 2*.**

The Endemic equilibrium state of the model given as

$E^* = \left(\frac{\delta+\Gamma+\mu}{\beta}, (R_0 - 1)\frac{\mu}{\beta}, (R_0 - 1)\frac{\Gamma}{\beta}\right)$ exist iff $R_0 > 1$

5.1. GLOBAL STABILITY OF THE ENDEMIC EQUILIBRIUM

The local stability of the endemic equilibrium will be studied in this section.

Theorem 3. (Routh-Hurwitz conditions)

Let $J = \begin{bmatrix} f_x(x_*, y_*) & f_y(x_*, y_*) \\ g_x(x_*, y_*) & g_y(x_*, y_*) \end{bmatrix}$

Be the Jacobian matrix of the non-linear system

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

$$\frac{dy}{dt} = g(x, y)$$

Evaluate at the critical point (x_*, y_*).

Then the critical point(x_*, y_*) is

(i) Asymptotically stable if $Trace(J) < 0 \; and \; Det(J) > 0$
(ii)Stable but not asymptotically stable if $Trace(J) = 0 \; and \; Det(J) > 0$
(iii) Unstable if $Trace(J) < 0 \; and \; Det(J) < 0$

Next, we will study the stability of the endemic equilibrium by applying the Routh-Hurwitz. The Jacobian matrix associated with the system (2.0) is

$$J(S^*, I^*, R^*) = \begin{bmatrix} -\beta I^* - \mu & \beta S^* & 0 \\ \beta I^* & \beta S^* - (\mu + \delta + \Gamma) & 0 \\ 0 & \Gamma & -\mu \end{bmatrix}$$

Clearly, $-\mu$ is an eigen value, the other two eigen values are negative if the Routh-Hurwitz condition hold.

i.e. $Trace \; of \; G < 0$

Determinant of $G < 0$

Where $G = \begin{bmatrix} -\beta I^* - \mu & \beta S^* \\ \beta I^* & \beta S^* - (\mu + \delta + \Gamma) \end{bmatrix}$

The trace of G is given as

$$Trace \; G = -\beta I^* - \mu + \beta S^* - (\mu + \delta + \Gamma)$$

$$= -\beta(R_0 - 1)\frac{\mu}{\beta} - \mu + \beta\frac{(\mu + \delta + \Gamma)}{\beta} - (\mu + \delta + \Gamma)$$

$$= -\beta(R_0 - 1)\frac{\mu}{\beta} - \mu < 0 \; for \; R_0 > 1$$

The determinant of G is given by

$$Det\ G = -(\beta I^* + \mu)[\beta S^* - (\mu + \delta + \Gamma)] - \beta^2 S^* I^*$$

$$= \left(-\beta(R_0 - 1)\frac{\mu}{\beta} + \mu\right)\left[\frac{\beta(\mu + \delta + \Gamma)}{\beta} - (\mu + \delta + \Gamma)\right]$$

$$-\left(\frac{\beta^2(\mu + \delta + \Gamma)}{\beta}(R_0 - 1)\frac{\mu}{\beta}\right)$$

$$= -\left(\beta(R_0 - 1)\frac{\mu}{\beta} + \mu\right)[0] - ((\mu + \delta + \Gamma)(R_0 - 1)\mu)$$

$= -\big((\mu + \delta + \Gamma)(R_0 - 1)\mu\big) < 0\ for\ R_0 > 1.$

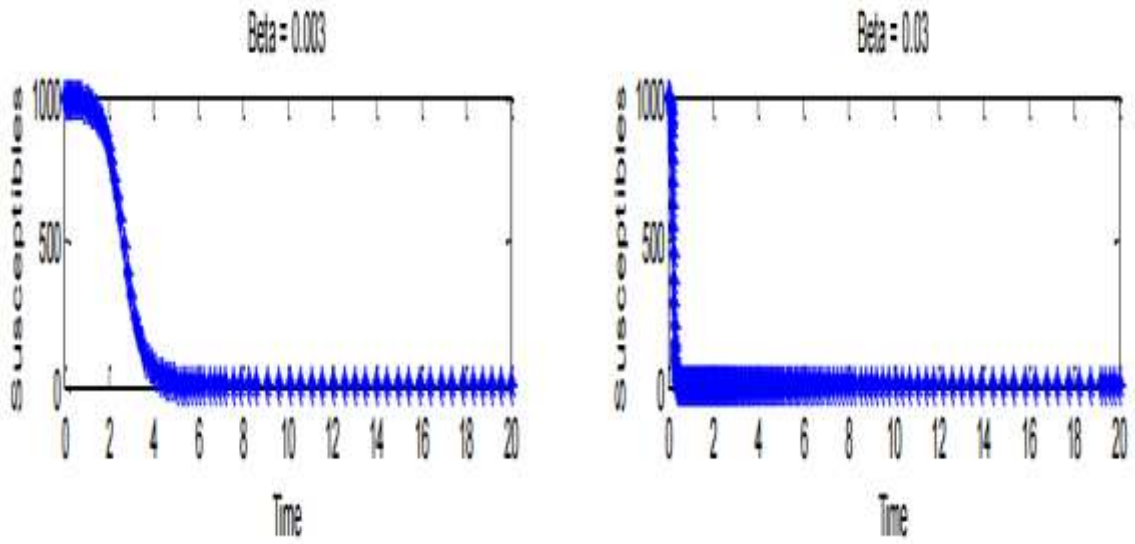
One of the conditions given above for negativity of the eigen values is violated. This implies that the endemic equilibrium of the model system (2.0) is unstable for $R_0 > 1$.

6. MODEL SIMULATION

A numerical simulation of the model is performed using the parameter values as listed below

Parameter	Value
Λ_0	3
β	[0.003,0.03]
Γ	0.4
δ	0.0023
μ	0.02

Table: Parameter values used in simulation



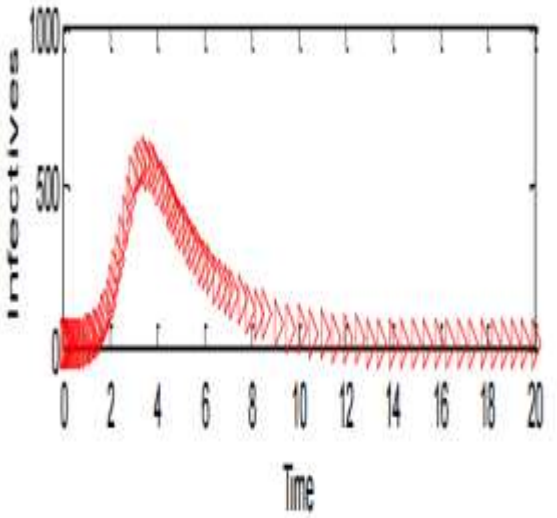


Fig. 1. Simulation of the mass action epidemic model with $\beta = 0.003$

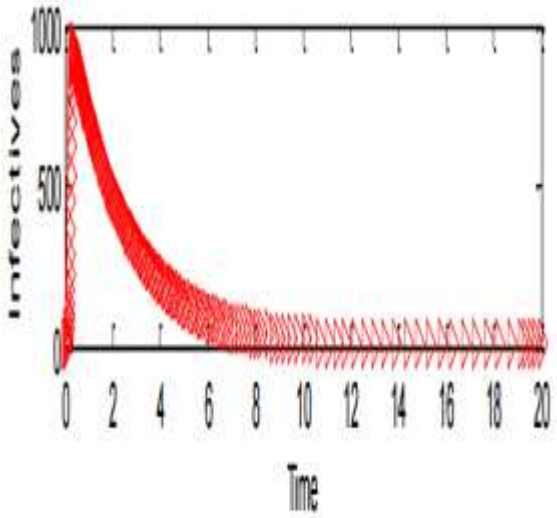
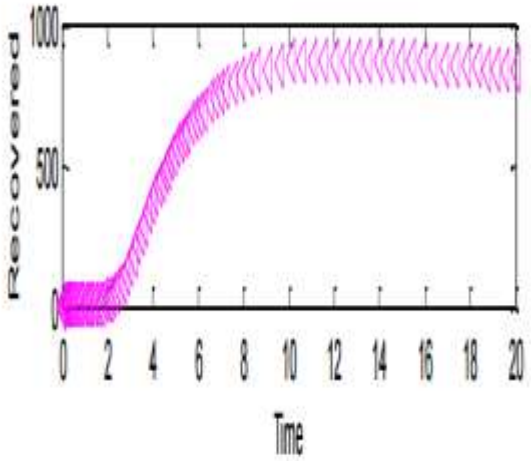
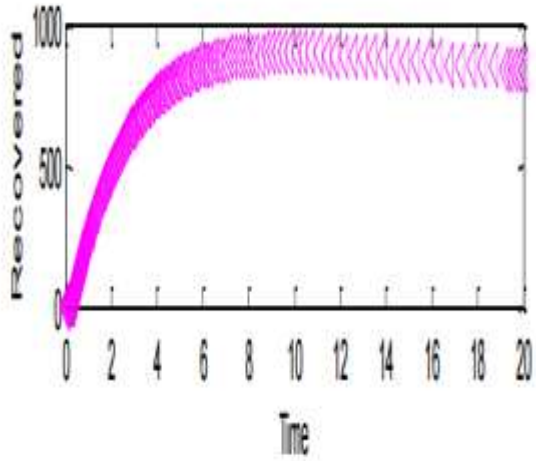


Fig.2. Simulation of the mass action epidemic model with $\beta = 0.03$





Figures 1 and 2 show that an increase in the contact rate has an impact on the transmission dynamics of the disease which is detrimental. When the contact rate is higher, the number of infectious individuals rise significantly leading to a rapid spread of the disease and has the potential to cause the infection to dominate the entire population quickly.

CONCLUSION

In this study, we examined a mass action epidemic model incorporating vital dynamics. Our findings primarily hinge on the behavior of the basic reproduction number, R_0. Specifically, if when $R_0 < 1$, the disease free equilibrium is both locally and globally asymptotically stable, indicating disease elimination.

Conversely, the endemic equilibrium exists only when $R_0 > 1$, suggesting that effective control strategies should aim to reduce R_0 below unity to eradicate the disease from the population. One practical approach to achieve this is by lowering the contact rate, which is a parameter that can be readily adjusted through intervention measures.

As demonstrated in Figures 1 and 2 increasing the contact rate has a detrimental impact on disease transmission dynamics. Higher contact rates lead to a significant rise in the number of infectious individuals, facilitating rapid disease spread and potentially causing the infection to dominate the entire population quickly.

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