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STABILITY ANALYSIS OF SIR MODEL WITH NON-LINEAR HARVESTING AND VACCINATION

Omer Saad Ali
AL-Bahaa Primary school, Baghdad, Iraq
Corresponding author email: pipe.math@gmail.com

ABSTRACT
In this paper, a mathematical model SIR with non-linear harvesting and vaccination, in addition to the disease spread by contact between susceptible and infected individuals. The type of functional response for describing the harvesting as well as linear incidence for describing transition of diseases are used and vaccine . The existence, uniqueness, boundedness of the solution and the stability analysis of all possible equilibrium points are studied. The Lyapunov function is used to study the global dynamics of the model. The effect of the vaccination and harvest on the dynamical of the system is discussed by using numerical simulation.

KEYWORDS: SIR model, functional response, non-linear harvesting, vaccination.

INTRODUCTION

A mathematical model is a description of system using mathematical concepts and language. Typically math-matical formula is used to describe the interactions of the various components of the system by depending on two important different fields; these are the ecology and epidemiology. The ecology is the branch of biology that deals with the relations and interactions between organisms and their environment, while the epidemiology is the branch of medicine that deals with the incidence and prevalence of disease in large populations and with detection of the source and cause of epidemics of infectious disease[1]. The mathematical models not only help us to understand the system, but also are instrumental to yield insight into the complex processes involved in biological systems by extracting the essential meaning of the hypotheses (Wimsatt, 1987; Bedau, 1999; Schank, 2008) and allows to study the effects of changes in its components and/or environmental conditions on the system's behavior; that is, they allow the control and optimization of the system[2]. Anderson and May[3,4] were the first whose merged the above two fields and formulated Lotka-Volterra prey-predator model with infection disease spread among prey by contact between them and no reproduction in infected prey.

Disease outbreaks can take the epidemic proportions depending on the intensity of the pathogen, its mode of transmission, herd immunity, and prevalence and incidence of the illness and disease in the community. Endemic occurs when a disease is constantly present in a community. Disease outbreaks take the forms of pandemics when a large population, over a broad geographical area becomes susceptible to the disease agents, as a result of the simultaneous outbreaks of the diseases (Halsey 1986). In between these two extremes of disease outbreaks, when there is a temporary increase of cases in the disease prevalence in the population, then the outbreak is called epidemic. It is defined as an outbreak of disease [5]

In fact, the study of effect of infectious disease in ecological system is now becoming an important factor for regulating animal and human population size. So in the last years; mathematical models have become extremely important tools in analyzing and understanding the spread and control of infectious disease through the study of the different types from disease for example SI, SISE and SIR, where some of infectious disease in the ecology system is transmitted by contact in same of species have proposed and studied from some of researchers, Ali and Majeed[1] studied a prey-predator model with SI infectious disease in prey and predator with harvesting . Moreover, there are some of infectious diseases are transmitted in the species not only through contact, but also directly from environment. Majeed and Shawka[6] studied prey-predator model with SI and SIS infectious disease in prey population and the disease transmitted within the same species by contact and external source. Recently, Sudipa Chauhan *et al.* [7] had proposed and studied SIR with vaccination. The harvest rate has a strong influence on the dynamic development of the population, perhaps one of the most important hunting the fish or eradication on the disease. Majeed and Ismaeel[8] studied the dynamical behavior of stage structured prey- predator model in the harvesting and toxin presence, and he assumed that the harvest can eradication the disease, also Anjana Das *et al* [9] studied prey-predator model with harvest in both, and he assumed that the harvest can remove a parasite. In general, there are three kinds of harvesting function [10,11] have been studied in the literature Constant harvesting, linear Proportionate harvesting and Non-linear harvesting.

In this paper, SIR model with non-linear harvesting and vaccination in susceptible population has been proposed

and analyzed. Further, in this model, Holling type-II functional response for the harvesting of susceptible population and linear functional response for the vaccination of population as well as linear incidence rate for describing the transition of disease are used. Our aim is to study the effect of harvesting and vaccination on the dynamics of the system.

2. Mathematical model:

The SIR Model is used in epidemiology to compute the amount of susceptible, infected, recovered people in a population. In this model we added a non-linear harvest using, Holling type-II functional response and constant vaccination.

This model is an appropriate one to use under the following assumptions:

We assumed that the population size was constant and that the death rate and births were equal and suppose that the disease is transmitted through direct contact with the patient, and here the person moves from the group of people exposed to the infection to infected people and when the person recovers he acquires immunity, as defined earlier: The model starts with some basic notation: • S (t) is the number of susceptible individuals at time t. • I (t) is the number of infected individuals at time t • R (t) is the number of recovered individuals at time t • N is the total population size The assumptions lead us to a set of differential equations.

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dT} = \beta SI - \gamma I \qquad (1.1)$$
$$\frac{dR}{dT} = \gamma \mathrm{I}$$

where S (t) is the number of susceptible at time t, I (t) is the number of infected people at time t, R (t) is the number of recovered at time t, β is the transmission of disease from an infected person in a time period γ is the recovery rate and the population size is constant, so that

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

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

By considering the total population density, we have

$S(t) + I(t) + R(t) = 1$ we get $R(t) = 1 - S(t) - I$

Now, we present our second model in which we have also consisting vaccination and non-linear harvesting and the suggested model is as follows

$$\frac{dS}{dt} = (1-p)\mu - \beta SI - \mu S - \frac{aS}{b+S}$$
$$\frac{dI}{dt} = \beta SI - \mu I - \gamma I \qquad (1.2)$$
$$\frac{dR}{dt} = \gamma \mathrm{I} - \mu \mathrm{R}$$
$$\frac{dH}{dt} = \frac{aS}{b+s} + \mu p - \mu H$$

With $S(0) \geq 0$,$I(0) \geq 0$, $R(0) \geq 0$ and $H(0) \geq 0$.

where S (t) is the number of susceptible at time t, I (t) is the number of infected people at time t, R (t) is the number of recovered at time t ,H(t) is the number of harvesting at time t, β is the transmission of disease from an infected person in a time period, μ is the is the death and birth rate which are assumed to be equal, γ is the recovery rate ,p is the number of individual taken dosage or vaccine , a is the effort applied to harvesting individual , b is suitable positive constant and the population size is constant, so that

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

and

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} + \frac{dH}{dt} = 0$$

By considering the total population density, we have

$$S(t) + I(t) + R(t) + H(t) = 1$$

We get

$$R(t) = 1 - S(t) - I(t) - H(t)$$

Therefore it is enough consider the model

$$\begin{aligned}\frac{dS}{dt} &= (1-p)\mu - \beta SI - \mu S - \frac{aS}{b+S} = f_1(S,I,H)\\ \frac{dI}{dt} &= \beta SI - \mu I - \gamma I = f_2(S,I,H)\\ \frac{dH}{dt} &= \frac{aS}{b+s} + \mu p - \mu H = f_3(S,I,H)\end{aligned} \qquad (1.3)$$

It is easy to verify that all the interaction functions f_1, f_2 *and* on the right hand side of system (1.3) are continuous and have continuous partial derivatives on R_+^3 with respect to dependent variables S , I and H. Accordingly they are Lipschitzian functions and hence system (1.3) has a unique solution for each non-negative initial condition. Furthermore the boundedness of the system is shown in the following theorem.

Theorem (1.1): All the solutions of system (1.3) which initiate in R_+^3 are uniformly bounded.

Proof

Let $(S(t), I(t), H(t))$ be any solution of the system (1.3) with non-negative initial condition $(S(0), I(0), H(0))$. According to the first equation of system (1.3) we have. Define the function $M(t) = S(t) + I(t) + H(t)$. Therefore,

$$\frac{dM}{dt} < \mu - \mu M$$

Now, by using the comparison theorem [12] on the above differential inequality, we get that:

$$M(t) \leq 1 + (M(0) - 1)\, e^{-\mu t} \quad .$$

Thus $0 \leq M(t) \leq 1$ as $t \to \infty$. Hence all the solutions of system (1.3) are uniformly bounded and the proof is complete

3. Existence of equilibrium points

In this section, the existence of all possible equilibrium points of the system (1.3) is discussed. it is observed that , system (1.3) has at most two equilibrium points.

1) The disease-free equilibrium point $E_1 = (\bar{S}, 0, \bar{H})$ where ;

$$(1-p)\mu - \mu S - \frac{aS}{b+S} = 0 \qquad (2.3a)$$

$$\frac{aS}{b+S} + p\mu - \mu H = 0 \qquad (2.3b)$$

From equation $(2.3a)$ we have,

$$M_1 S^2 + M_2 S + M_3 = 0 \qquad (2.3c)$$

Where

$$\begin{aligned} M_1 &= \mu \\ M_2 &= -\big((1-p)\mu - \mu b - a\big) \\ M_3 &= -(1-p)\mu b \end{aligned}$$

Straightforward computation shows that equation (2.3c) has a unique positive root namely $\bar{S}$

Note that the equation $(2.3b)$ has a positive root:

$$\bar{H} = \frac{1}{\mu}\left(a\,\frac{\bar{S}}{b+\bar{S}} + p\mu\right) \qquad (2.3d)$$

2) The endemic equilibrium point $E_2 = (S^*, I^*, H^*)$ exists if and only if there is a positive solution to the following set of equations:

$$(1-p)\mu - \beta SI - \mu S - \frac{aS}{b+S} = 0 \qquad (2.4a)$$

$$\beta S - \mu - \gamma = 0 \qquad (2.4b)$$

$$\frac{aS}{b+s} + \mu p - \mu H = 0 \qquad (2.4c)$$

From equation (2.4b) we have,

$$S^* = \frac{\mu+\gamma}{\beta} \qquad (2.4d)$$

Now, by substituting equation (2.4b) in equation (2.4c) we get:

$$H^* = \frac{1}{\mu}\left(\frac{a(\gamma+\mu)}{\beta b+\gamma+\mu} + p\mu\right) \qquad (2.4e)$$

Then by substituting equation (2.4d) and (2.4e) in (2.4a) we get:

$$I^* = \frac{(1-p)\mu}{\gamma+\mu} - \frac{\mu}{\beta} - \frac{a}{\beta b+\gamma+\mu} \qquad (2.4f)$$

The equation (2.4f) is positive Provided that :

1) $\beta > \gamma + \mu$
2) $\mu(\beta b+\gamma+\mu)(\beta-(\gamma+\mu) > \mu\beta p(\beta b+\gamma\mu)+a\beta(\gamma+\mu)$ (2.4

4. Local Stability Analysis.

In this section, we analyzed the local stability of the model (1.3) around only free disease equilibrium point and endemic equilibrium point and discussed through computing the Jacobian matrix $J(S, I, H)$ and determined the eigenvalues of system (1.3) at each of them the Jacobian matrix $J(S, I, H)$ of the system (1.3) at each of them can be written:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial H} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial H} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial H} \end{bmatrix} = \begin{bmatrix} -\beta I-\mu-\frac{ab}{(b+S)^2} & -\beta S & 0 \\ \beta I & \beta S-\gamma-\mu & 0 \\ \frac{ab}{(b+S)^2} & 0 & -\mu \end{bmatrix} . \qquad (1.3a)$$

Stability of the equilibrium point $E_1 = (\bar{S}, 0, \bar{H})$

The Jacobian matrix of system (1.3) at E_1 can be written as,

$$J_1 = J(E_1) = \begin{bmatrix} -\mu-\frac{ab}{(b+\bar{S})^2} & -\beta\bar{S} & 0 \\ 0 & \beta\bar{S}-\gamma-\mu & 0 \\ \frac{ab}{(b+\bar{S})^2} & 0 & -\mu \end{bmatrix} \qquad (1.3b)$$

Then the characteristic equation of $J(E_1)$ is given by:

$$\left(-\mu-\tfrac{ab}{(b+\bar{S})^2}-\lambda\right)(\beta\bar{S}-\gamma-\mu-\lambda)(-\mu-\lambda) = 0,$$

So, the eigenvalues of J_1 are $\lambda_S = -\mu-\frac{ab}{(b+\bar{S})^2}$, $\lambda_I = \beta\bar{S}-\gamma-\mu, \lambda_H = -\mu$

Thus, the equilibrium point E_1 is locally asymptotically stable in the. R_+^3, provided that,

$\mu+\gamma > \beta\bar{S}$ (1.3c)

- **Stability of the equilibrium point $E_2 = (S^*, I^*, H^*)$**

The Jacobian matrix of system (1.3) at E_2 can be written as,

$$J_2 = J(E_2) = \begin{bmatrix} -\mu-\frac{ab}{(b+S^*)^2} & -\beta S^* & 0 \\ \beta I^* & \beta S^*-\gamma-\mu & 0 \\ \frac{ab}{(b+S^*)^2} & 0 & -\mu \end{bmatrix} \qquad (1.3d)$$

Then the characteristic equation of $J(E_2)$ is given by:

$$(-\mu-\lambda)[\lambda^2+B_1\lambda+B_2) = 0 \qquad (1.3e)$$

Then the characteristic equation of $J(E_2)$ is given by:

$$[\lambda^2+B_1\lambda+B_2](-\mu-\lambda) = 0,$$

where:

$$B_1 = (\mu + \frac{ab}{(b+S^*)^2}) > 0$$
$$B_2 = \beta^2 S^* I^* > 0$$
So, either
$$(-\mu - \lambda) = 0, \ \lambda_H = -\mu \qquad (1.3Fa)$$
Or

$$\lambda^2 + B_1 \lambda + B_2 = 0$$
which gives that other two eigenvalues of J_2 with negative real parts which are (by using Routh Hurwitz criteria)

$$\lambda_{21} = \frac{1}{2}\left(-B_1 + \sqrt{B_1^2 - 4B_2}\right),$$
$$\lambda_{22} = \frac{1}{2}\left(-B_1 - \sqrt{B_1^2 - 4B_2}\right).$$
So, equilibrium point E_2 is locally asymptotically stable in the . R_+^3 . However, it is unstable otherwise.

5. Global stability analysis:
In this section the global stability analysis for the equilibrium points, which are locally asymptotically stable of system (1.3) is studied analytically by using a suitable Lyapunov function as shown in the following theorems.
Theorem (2.4a):
Assume that the predator free equilibrium point $E_1 = (\bar{S}, 0, \bar{H})$ of system (1.3) is locally asymptotically stable in the R_+^3. Then E_1 is globally asymptotically stable on the sub region $\omega_1 \subseteq R_+^3$ provided that the following conditions hold:

$$\bar{\beta}_1 > \bar{\beta}_2 \qquad (2.4aA)$$
where
$$\bar{\beta}_1 = \mu(1 + \bar{S} + \bar{H}) + \frac{aH\bar{S}}{S}$$
and
$$\bar{\beta}_2 = \mu\left(S + \frac{\bar{S}}{S}(1-P) + H + \frac{P\bar{H}}{H}\right)$$
Proof: Consider the following function
$$G_1(S,I,H) = \left(S - \bar{S} - \bar{S}\, ln\frac{S}{\bar{S}}\right) + (H - \bar{H} - \bar{H} ln\frac{H}{\bar{H}}) + I$$
It is easy to see that $G_1(S,I,H) \in C^1(R_+^3, R)$, and $G_1(E_1) = 0$, and $G_1(S,I,H) > 0; \forall (x,y,z,w) \neq E_1$. Now by differentiating G_1 with respect to time t and going some algebraic handling, given that:

$$\frac{dG_1}{dt} = \frac{(S-\bar{S})}{S}\left((1-p)\mu - \beta SI - \mu S - \frac{aS}{b+S}\right) + \beta SI - \mu I - \gamma I + \frac{(H-\bar{H})}{H}(\frac{aS}{b+S} + P\mu - \mu H)$$
$$\frac{dG_1}{dt} < \mu(1 + \bar{S} + \bar{H}) + \frac{aH\bar{S}}{S} - \mu\left(S + \frac{\bar{S}}{S}(1-P) + H + \frac{P\bar{H}}{H}\right) = \bar{\beta}_1 - \bar{\beta}_2$$
Thus, $\frac{dG_1}{dt}$ is negative definite and hence G_1 is Lyapunov function under the condition $(1.3C)\ and\ (2.4aA)$. So E_1 is a globally asymptotically stable on the sub region $\omega_1 \subseteq R_+^3$ and then the proof is complete

Theorem (2.4b):
Assume that the endemic equilibrium point $E_2 = (S^*, I^*, H^*)$ of system (1.3) is locally asymptotically stable. Then E_2 is globally asymptotically stable on in the sub region $\omega_2 \subseteq R_+^3$ provided that the following conditions hold:

$$S > S^*, I > I^*, H > H^* \qquad (2.4ba)$$
$$\beta_1^* > \beta_2^* \qquad (2.4bb)$$

Where
$$\beta_1^* = \mu + \beta(S^*I - SI^*),$$
$$\beta_2^* = \mu(S - S^*) + (\mu + \gamma)(I - I^*) + \mu(H - H^*) + \frac{S^*}{S}((1-P)\mu - a),$$

Proof: Consider the following function
$$G_4(S,I,H) = \left(S - S^* - S^* ln\frac{S}{S^*}\right) + \left(I - I^* - I^* ln\frac{I}{I^*}\right) + \left(H - H^* - H^* ln\frac{H}{H^*}\right)$$

It is easy to see that, $G_2(S,I,H)\epsilon\, C^1(R_+^3,R)$, and $G_2(E_2)=0$, and $G_2(S,I,H)>0\ ;\forall(S,I,H)\neq E_2$. Now by differentiating G_2 with respect to time t and going some algebraic handling, given that:

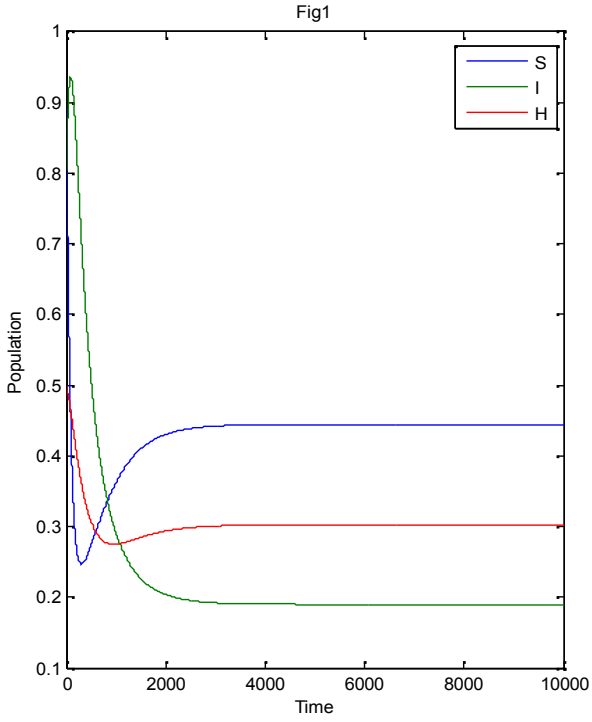
$$\frac{dG_2}{dt}<\mu+\beta(S^*I-SI^*)-\mu(S-S^*)-(\mu+\gamma)(I-I^*)-\mu(H-H^*)-\frac{S^*}{S}\big((1-P)\mu-a\big)=\beta_1^*-\beta_2^*$$

Thus, $\frac{dG_2}{dt}$ is negative definite and hence G_2 is Lyapunov function under the conditions $(2.4aa)-(2.2bb)$. So E_2 is a globally asymptotically stable on the sub region $\omega_2\subseteq R_+^3$ and then the proof is complete■

6. Numerical simulation

In this section, we confirmed our obtained results in the previous sections numerically by using Runge Kutta method along with predictor corrector method. Note that, we use turbo C++ in programming and matlab in plotting and then discuss our obtained results. The system (1.3) is studied numerically for different sets of parameters and different sets of initial points. The objectives of this study are: first investigate the effect of varying the value of each parameter on the dynamical behavior of system (1.3) and second confirm our obtained analytical results. It is observed that, for the following set of hypothetical parameters:

$$\boldsymbol{p=0.1,\mu=0.3,\beta=0.9,a=0.17,b=0.8,\gamma=0.1}\quad\boldsymbol{(2.5a)}$$



Clearly, figure (1) shows that system (1.3) approaches asymptotically to the endemic equilibrium point E_1 = (0.444, 0.0.189, 0.302) starting from initial point and this is confirming our obtained analytical results

Now, in order to discuss the effect of the parameters values of system (1.3) on the dynamical behavior of the system, the system is solved numerically for the data given in (2.5a) with varying one parameter at each time and sometime two parameters the obtained results are given below.

The dosage rate in the range $0.4 < p < 0.9$, keeping other parameters as data given in (2.5a), causes extinction in the infected and the system will approach to the infected free equilibrium point. However for $0.1 < p < 0.3$, it is observed that system (1.3) still approach asymptotically to the endemic equilibrium point. On the other hand the death and birth rate in the range $0.7 < \mu < 0.9$, keeping other parameters as data given in (2.5a) ,causes extinction in the infected and the system will approach to the infected free equilibrium point. However for $0.1 < \mu < 0.6$, it is observed that system (1.3) still approach asymptotically to the endemic equilibrium point.

The transmission of disease rate in the range $0.1 < \beta < 0.6$, keeping other parameters as data given in (2.5a), causes extinction in the infected and the system will approach to the infected free equilibrium point. However for $0.7 < \beta < 0.99$, it is observed that system (1.3) still approach asymptotically to the endemic equilibrium point.

The harvesting rate in the range $0.7 < a < 0.9$, keeping other parameters as data given in (2.5a), causes extinction in the infected and the system will approach to the infected free equilibrium point. However for $0.1 < a < 0.6$, it is

observed that system (1.3) still approach asymptotically to the endemic equilibrium point. Further for $0.1 < \alpha < 0.6$ and p=0 the solution of the system (1.3) approaches to the endemic equilibrium point; additional for $0.7 < a \leq 0.9$ and p=0 causes extinction in the infected population and the system will approach the infected free equilibrium point.

The recovery rate in the range $0.2 < \gamma < 0.9$,keeping other parameters as data given in (2.5a) ,causes extinction in the infected and the system will approach to the infected free equilibrium point as shown in the following figure. However for $0.1 < \gamma < 0.2$, it is observed that system (1.3) still approach asymptotically to the endemic equilibrium point.

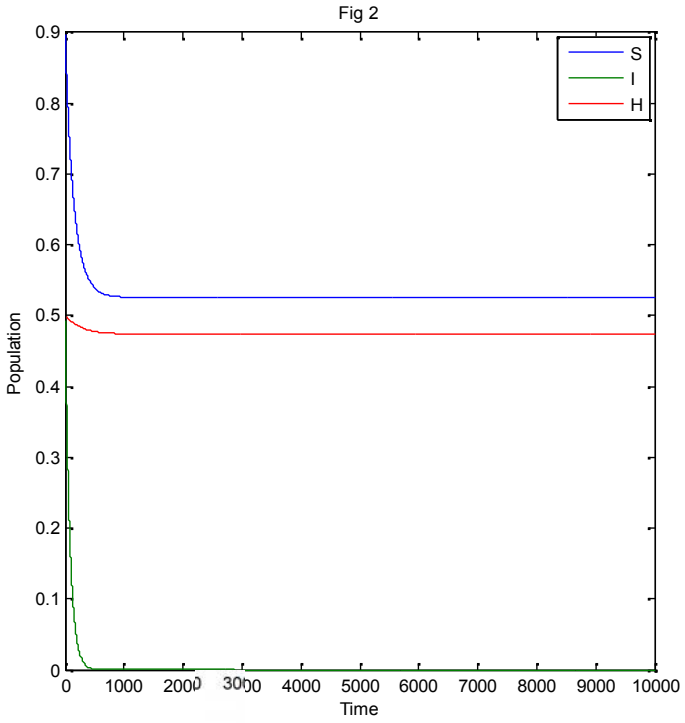


Fig. 2 Time series of the solution of system (1.3) approaches asymptotically to the infected free equilibrium point E_1 = (0.44, 0 , 0.53) for the data given in (2.5a) with $\gamma = 0.4$.

CONCLUSION

In this paper, model have been discussed SIR with vaccination and non-linear harvesting on only susceptible population are discussed. The model it is found that the dynamics of the infection were dependent on the value for the basic eigenvalues, it is observed that disease-free equilibrium state, exist only when the reproduction number eigenvaluves are negative and all the trajectories will be approaching towards the (DFE) E_1. The local and global stability of the disease free equilibrium point is also discussed.

There is the endemic (positive) equilibrium point E_2 of system (1.3) exist provided that the condition (2.4d) is hold. The local and global stability of the endemic equilibrium point is also discussed by using Lyapunov function and also study cases of all parameters. Further, the effect of infection rate is also seen on the susceptible and infected population. The susceptible population gradually decreases and infected population increases as the infection rate β increases. But as we induce vaccination and harvesting in the susceptible population, the infected population suddenly decreases to a very lower level. It was noticed that the absence of the vaccine and the presence of the harvest works to stabilize the system, and this gives great importance to the harvest. This lead to stop the disease. Thus, we can conclude that the infection rate and harvesting rate plays a very important role for an epidemic to occur and this epidemic can be controlled by vaccination and harvesting

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