ISSN 0974 - 9373

Vol. 14 No.3 (2010) **Journal of International Academy of Physical Sciences** pp. 305-322

Analysis of an SIR Model with Saturating Contact Rate and Carrier- Dependent Infectious Diseases under the Effect of Environmental Discharge*

Manju Agarwal and Vinay Verma
Department of Mathematics & Astronomy
University of Lucknow, Lucknow-226007, India
Email: manjuak@yahoo.com ; vinay.verma09@rediffmail.com

(Received February 20, 2010)

Abstract: This paper deals with an *SIR* epidemic model that incorporates constant recruitment rate and disease caused death. We consider the saturating contact rate of individuals contact given by Heesterbeek and Metz[1]. The model for carrier-dependent infectious diseases, like cholera, diarrhea etc. caused by direct contact of susceptibles with infectives as well as by carriers is proposed and analyzed assuming the logistic growth of carrier population. Stability analysis of this model is carried out using usual theory of nonlinear differential equation. The criteria for asymptotic and global stability of an interior equilibrium are obtained. By computer simulation it is shown that if the growth rate of recovery and intrinsic growth rate of carrier population increase, the infective human population decreases and increases respectively. It is concluded from the analysis that if the death rate of carrier population increases, both of the infective and carrier population decreases.

Keywords: Susceptible, Infective, Recovery, Saturating Contact Rate, Carrier-Dependent, Stability Analysis, Numerical Simulation.
Mathematical Subject Classification: 92Bxx

1. Introduction

There are many carrier dependent infectious diseases which afflict human population around the world. However, the third world countries are most affected by such diseases due to lack of sanitation wide occurrence of carriers such as flies, ticks, mites, etc. generally present in the environment[2]. For example, air-borne carriers spread diseases such as tuberculosis and measles, while water-borne carriers are responsible for the spread of dysentery, gastroenteritis, diarrhea, etc[3,4]. These carriers transport infectious agents of diseases from infectives to susceptibles and thus spread such diseases in human population. In this paper, we have used the term carriers

*Presented at CONIAPS XI, University of Allahabad, Feb. 20-22, 2010.

as a mode of transmission only, which transmit infectious agents of diseases from infectives to susceptibles, without having clinical symptoms.

Thieme and Castillo-Chavez[5] suggest that the general from of a population size dependent incidence as $\beta C(N)\frac{X_1}{N}X_2$. Here X_1 and X_2 are the numbers of susceptibles and infectives at time t respectively, β is the probability per unit time of transmitting the infection between two individuals to take part in a contact, and $C(N)$ is the 'unknown' probability for an individual to take part in contact. $C(N)$ is usually called the contact rate, and $\beta C(N)$ which the average number of adequate contacts of an individual per unit time is adequate contact rate. It is a contact which is sufficient for transmission of the infection from an infective to a susceptible. In most of the research study, the adequate contact rate $\beta C(N)$ frequently takes two forms. One is linearly proportional forms $\beta N\frac{X_1}{N}X_2 = \beta X_1 X_2$, and the other a constant φ, the corresponding incidence $\varphi\frac{X_1}{N}X_2$ is called standard form. When the total population size N is not too large, since the number of contacts made by an individual per unit time should increase as the total population size N increases, the linear adequate contact rate βN would be suitable. But when the total size is large, since the number of contacts made by an infective per unit time should be limited, or should grow less rapidly as the total population size N increases, the linear adequate contact rate βN is not suitable and the constant adequate contact rate φ may be more realistic. Hence the two adequate contact rates discussed above are actually two extreme cases for the total population size N being very small and very large respectively. More generally, it may be assumed that the adequate contact rate is a function $C(N)$ of the total population size N and some demands on $C(N)$ are that it should be a non-decreasing function of N and that $\frac{C(N)}{N}$ should be a non-increasing function of N. Furthermore, $C(N)$ should behave linearly in N for small N, and it should be independent of N, for large N. Heesterbeek and Metz[1] derived the expression for the saturating contact rate of individual contacts in a population that mixes randomly

$$C(N) = \frac{bN}{1 + bN + \sqrt{1+2bN}}, \tag{1.1}$$

then we see that, for N small, $C(N) \sim bN$, whereas for N large, $C(N) \sim 1$. Furthermore, $C(N)$ is non decreasing and $\frac{C(N)}{N}$ is non-increasing.

In this paper, we consider an SIR model with saturating contact rate $C(N)$ defined by (1.1). Let A be constant recruitment rate into the population. Let natural deaths occurs at a rate proportional to the population size, so the death rate term is μN where μ is the death rate constant. Thus in the absence of disease, the differential equation for the population size N is $\frac{dN}{dt} = A - \mu N$. For this structure the total population size $N(t)$ approaches $\frac{A}{\mu}$ for any non-zero initial population size. Then the constant $\frac{A}{\mu}$ is the carrying capacity. Although N varies in the finite interval $(0, \frac{A}{\mu})$, it can still be for greater (for example, A is very large and μ is very small) than the value at which $C(N)$ reaches its saturating state. Thus the saturation effect of the saturating contact rate $C(N)$ can still take place that is it is reasonable that the saturating contact rate $C(N)$ is used in the SIR model with recruitment.

In previous research studies, it is assumed that the disease incubation period is negligible so that once infected, each susceptible individual (in the class S) instantaneously become infectious (in the class I) and later recovers (in the class R) with a permanent or temporary acquired immunity. A compartmental model based on these assumptions is customarily called an SIR model. The SIR type models are widely studied [6-11]. In particular, Greenhalgh[12] has studied an infectious disease model with population – dependent death rate. Zhou and Hethcote[13] have studied a few models for infectious disease using various kinds of demographics. Hethcote[14] has discussed an epidemic model in which the carrier population is assumed to be constant. But in general the size of carrier population varies and depends on the natural conditions of the environment as well as on various discharges in to it by the human population. In particular, Shukla et al[16] studied the spread of carrier-dependent infectious diseases with environmental effect using variable carrier population. We have modified this model by considering saturating contact rate and environmental discharge dependent carrying capacity of carrier population with an extra recovered class of human population.

2. The Mathematical Model

In this paper we consider an *SIR* model governing by the set of the following system of non linear ordinary differential equations;

$$
\begin{aligned}
\frac{dX_1}{dt} &= A - \mu X_1 - \frac{\beta C(N) X_1 X_2}{N} - \lambda X_1 K,\\
\frac{dX_2}{dt} &= \frac{\beta C(N) X_1 X_2}{N} + \lambda X_1 K - X_2(\mu+\alpha+\gamma_0),\\
\frac{dR}{dt} &= \gamma_0 X_2 - \mu R,\\
\frac{dK}{dt} &= sK\left(1-\frac{K}{L(E)}\right) - s_1 K,\\
\frac{dE}{dt} &= Q(N) - \delta_0 E,
\end{aligned}
\tag{2.1}
$$

with initial condition: $X_1(0) \geq 0, X_2(0) \geq 0, R(0) \geq 0, K(0) \geq 0, E(0) \geq 0.$ Where $N = X_1 + X_2 + R$, $Q(N) = Q_0 + lN$, and $s > s_1$. Also,

$$L(E) = L_0 + L_1 E . \tag{2.2}$$

Since $C(N) = \dfrac{bN}{1 + bN + \sqrt{1+2bN}} = \dfrac{bN}{h(N)}$. So we can write, $\dfrac{\beta C(N) X_1 X_2}{N} = \dfrac{\beta b X_1 X_2}{h(N)} = \dfrac{a_0 X_1 X_2}{h(N)}$. Where $a_0 = \beta b$ and

$$h(N) = 1 + bN + \sqrt{1+2bN} . \tag{2.3}$$

We obtain the following system analogous to (2.1)

$$
\begin{aligned}
\frac{dX_1}{dt} &= A - \mu X_1 - \frac{a_0 X_1 X_2}{h(N)} - \lambda X_1 K,\\
\frac{dX_2}{dt} &= \frac{a_0 X_1 X_2}{h(N)} + \lambda X_1 K - X_2(\alpha+\mu+\gamma_0),\\
\frac{dR}{dt} &= \gamma_0 X_2 - \mu R,\\
\frac{dK}{dt} &= sK\left(1-\frac{K}{L(E)}\right) - s_1 K,\\
\frac{dE}{dt} &= Q(N) - \delta_0 \mathrm{E}.
\end{aligned}
\tag{2.4}
$$

By eliminating $X_1 (= N - X_2 - R)$ from (2.4) it is further reduced to the following subsystem (2.5),

$$\frac{dX_2}{dt} = \frac{a_0(N - X_2 - R)X_2}{h(N)} + \lambda(N - X_2 - R)K - X_2(\mu + \alpha + \gamma_0),$$

$$\frac{dR}{dt} = \gamma_0 X_2 - \mu R,$$

$$\frac{dN}{dt} = A - \mu N - \alpha X_2, \tag{2.5}$$

$$\frac{dK}{dt} = sK\left(1 - \frac{K}{L(E)}\right) - s_1 K,$$

$$\frac{dE}{dt} = Q(N) - \delta_0 E.$$

In above model (2.5), $N(t)$ is the total human population at time t with immigration of susceptibles at constant rate A. Here the total population is divided into three subclasses: the susceptible $X_1(t)$, the infectives $X_2(t)$, and the recovered individuals $R(t)$. We assume that recovered population at time $t = 0$ is zero. In the modeling process, it is assumed that the susceptibles become infective by the direct interaction with infectives $X_2(t)$ and also by carrier population of density $K(t)$, which is governed by a generalized logistic model. $E(t)$ is the cumulative density of environmental discharges conducive to the growth of carrier population. μ is the natural death rate; β and λ are the probabilities per unit time of infection transmission coefficients due to the infectives and the carrier population respectively; α is the disease related death rate constant and γ_0 is the recovery rate constant. s is intrinsic growth rate of carrier population and the constant s_1 is the death rate coefficient of carriers due to natural factors as well as by control measures. $Q(N)$ is the cumulative rate of environmental discharges and it is taken as to be increasing function of human population density and δ_0 is the natural depletion rate coefficient of the environmental discharges. $L(E)$ is the carrying capacity of the carrier population and it is taken to be increasing function of cumulative density of environmental discharges. It is assumed that carrier population attains value $L(E)\left(1 - \frac{s_1}{s}\right)$ as compared to usual logistic model. We also assume that the modified carrying capacity increases with cumulative density of environmental discharges, so that $L(0) = L_0 > 0$ and $L'(E) \geq 0$. where L_0 is the value of $L(E)$ when $E = 0$. We see that even if cumulative density of environmental discharges related factors are absent, carrier population density increases in its natural environment and it tends to $L_0\left(1-\frac{s_1}{s}\right)$ which may become zero if $s \to s_1$. In the model (2.5), all the dependent variables and parameter are assumed to be non negative. $C(N)=\frac{bN}{1+bN+\sqrt{1+2bN}} \cong \frac{bN}{h(N)}$ $(b>0)$, derived in [5], is the saturating contact rate of individual contacts in a population that mixes randomly.

3. Region of attraction

Theorem1: *The region of attraction for the system (2.5) is given by,*

$$\Omega = \left\{(X_2, R, N, K, E):\ 0 \le X_2 \le N \le \frac{A}{\mu}\ ,\right.$$

$$\left. 0 \le R \le \frac{\gamma_0 A}{\mu^2}\ , 0 \le K \le K_m, 0 \le E \le \frac{Q(A/\mu)}{\delta_0}\right\}$$

which attracts all solutions initiating in the positive orthant, where $K_m = L(E)\left(1-\frac{s_1}{s}\right)$.

Proof: From third equation of model (2.5) we get,

$$\frac{dN}{dt} \le A - \mu N.$$

This implies that, $0 \le N \le \frac{A}{\mu}$ or $0 \le X_1 + X_2 + R \le \frac{A}{\mu}$.

From second equation of model (2.5) we get,

$$\frac{dR}{dt} + \mu R \le \frac{\gamma_0 A}{\mu}.$$

On solving above differential equation we get,

$$R \le \left(\frac{\gamma_0 A}{\mu^2}\right)\left(1-\frac{1}{e^{\mu t}}\right).$$

When $t \to \infty$ we have,

$$0 \le R \le \frac{\gamma_0 A}{\mu^2}.$$

From fifth equation of model (2.5) we get,

$$\frac{dE}{dt} + \delta_0 E \le Q(A/\mu).$$

On solving above differential equation we get,

$$E \le \frac{Q(A/\mu)}{\delta_0}\left(1 - \frac{1}{e\delta_0 t}\right).$$

When $t \to \infty$ we have,

$$0 \le E \le \frac{Q(A/\mu)}{\delta_0}.$$

From fourth equation of model (2.5) we get,

$$\frac{dK}{dt} \le sK\left(1 - \frac{K}{L(E)}\right) - s_1 K.$$

This implies that, $0 \le K \le L(E)\left(1 - \frac{s_1}{s}\right)$ or $0 \le K \le K_m$.

4. Equilibrium Analysis

The system (2.5) has non-negative equilibria $E_1(0,0,\overline{N},0,\overline{E})$ and $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$.

Existence of $E_1(0,0,\overline{N},0,\overline{E})$: Here $\overline{N}$ and $\overline{E}$ are given by the solution of the following equations;

$$A - \mu\overline{N} = 0 \text{ and } Q(\overline{N}) - \delta_0\overline{E} = 0.$$

Clearly, $\overline{N} = \dfrac{A}{\mu} > 0$ and $\overline{E} = \dfrac{Q(\overline{N})}{\delta_0} = \dfrac{Q(A/\mu)}{\delta_0} > 0.$

So the equilibrium point $E_1(0,0,\overline{N},0,\overline{E})$ exists.

Existence of $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$: The non trivial interior equilibrium $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ is the positive solution of the following algebraic equation;

(4.1) $$a_0(N - X_2 - R)X_2 + \lambda K(N - X_2 - R)h(N) - X_2(\alpha + \mu + \gamma_0)h(N) = 0.$$

(4.2) $$\gamma_0 X_2 - \mu R = 0.$$

(4.3) $$A - \mu N - \alpha X_2 = 0.$$

(4.4) $$sK\left(1 - \frac{K}{L(E)}\right) - s_1 K = 0.$$

(4.5) $$Q(N) - \delta_0 E = 0.$$

Now from equation (4.3) and (4.2) we get,

$$X_2 = \frac{A-\mu N}{\alpha} \text{ and } R = \frac{\gamma_0 (A-\mu N)}{\mu\alpha}.$$

Also from equation (4.4) and (4.5) we get,

$$K = L(E)\left(1-\frac{s_1}{s}\right) and \ \mathrm{E} = \frac{Q_0 + lN}{\delta_0}.$$

Now putting value of X_2, R and K in equation (4.1) then whole equation reduces to N. So we can write,

$$F(N) = a_0\left[N - \frac{(A-\mu N)}{\alpha} - \frac{\gamma_0 (A-\mu N)}{\alpha\mu}\right]\left[\frac{(A - \mu N)}{\alpha}\right] \tag{4.6}$$

$$+\lambda K_m\left[N - \frac{(A-\mu N)}{\alpha} - \frac{\gamma_0 (A-\mu N)}{\alpha\mu}\right]h(N) - \left[\frac{(A-\mu N)(\alpha+\mu+\gamma_0)h(N)}{\alpha}\right].$$

It is clear from equation (4.6) that,

$$F(0) = -\left(\frac{A}{\alpha}\right)\left[\left(1+\frac{\gamma_0}{\mu}\right)\left(\frac{a_0 A}{\alpha} + 2\lambda K_m\right) + 2(\alpha+\mu+\gamma_0)\right] < 0.$$

And $F(A/\mu) = \lambda\left(\frac{A}{\mu}\right)K_m h\left(\frac{A}{\mu}\right) > 0.$ This implies that there exists a root N of $F(N) = 0$ in $0 < N < (A/\mu)$. Also,

$$F'(N) = \frac{(\alpha+\mu+\gamma_0)(A-\mu N)}{\alpha}\left[\left(\frac{a_0}{\alpha} - h'(N)\right)\right] + \frac{(\alpha+\mu+\gamma_0)h(N)}{\alpha}[\lambda K_m + \mu]$$

$$+\left[\frac{N\mu(\alpha+\mu+\gamma_0) - A(\mu+\gamma_0)}{\alpha\mu}\right]\left[h'(N)\lambda K_m - \frac{\mu a_0}{\alpha}\right] > 0,$$

provided $\frac{a_0}{\alpha} > h'(N), N\mu(\alpha+\mu+\gamma_0) > A(\mu+\gamma_0)$ and $h'(N)\lambda K_m > \frac{\mu a_0}{\alpha}$.

Hence, there exists a unique root $\hat{N}$ give by $F(N) = 0$ in $0 < N < A/\mu$. So the equilibrium point $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ exist.

5. Stability Analysis

Now we present the stability analysis of these equilibria. The local stability results are stated in the following theorem

Theorem1: *The equilibria* $E_1(0,0,\overline{N},0,\overline{E})$ *is unstable and* $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ *is locally asymptotically stable provided*

(5.1) $$(\gamma_0+\alpha)<\left[\frac{a_0\hat{X}_2}{h(\hat{N})}+\lambda\hat{K}+\frac{\lambda(\hat{N}-\hat{R}-\hat{X}_2)\hat{K}}{\hat{X}_2}\right],$$

(5.2) $$\left[\frac{a_0\hat{X}_2}{h(\hat{N})}+\lambda\hat{K}\right]<\mu,$$

(5.3) $$\left[\frac{a_0\hat{X}_2}{h(\hat{N})}+\lambda\hat{K}-\frac{a_0(\hat{N}-\hat{R}-\hat{X}_2)\hat{X}_2h'(N)}{\left[h(\hat{N})\right]^2}+l\right]<\mu,$$

(5.4) $$\lambda(\hat{N}-\hat{X}_2-\hat{R})<(s-s_1),$$

(5.5) $$\frac{s\hat{K}^2L_1}{\left[L(\hat{E})\right]^2}<\delta_0.$$

Proof: The variational matrix M_1 at $E_1(0,0,\overline{N},0,\overline{E})$ corresponding to the system of equation (2.5) is given by

$$M_1=\begin{pmatrix} 0 & 0 & 0 & \lambda N & 0 \\ \gamma_0 & -\mu & 0 & 0 & 0 \\ -\alpha & 0 & -\mu & 0 & 0 \\ 0 & 0 & 0 & (s-s_1) & 0 \\ 0 & 0 & l & 0 & -\delta_0 \end{pmatrix}$$

Since one eigen value $(s-s_1)$ of matrix M_1 is positive because $s>s_1$. So E_1 is unstable. The variational matrix M_2 at $E_2(\hat{X}_2,\hat{R},\hat{N},\hat{K},\hat{E})$ corresponding to the system of equation (2.5) is given by

$$M_2=\begin{pmatrix} B_1 & \dfrac{-a_0\hat{X}_2}{h(\hat{N})}-\lambda\hat{K} & B_2 & \lambda(\hat{N}-\hat{X}_2-\hat{R}) & 0 \\ \gamma_0 & -\mu & 0 & 0 & 0 \\ -\alpha & 0 & -\mu & 0 & 0 \\ 0 & 0 & 0 & -(s-s_1) & \dfrac{s\hat{K}^2L_1}{\left[L(\hat{E})\right]^2} \\ 0 & 0 & l & 0 & -\delta_0 \end{pmatrix},$$

where $$B_1=\frac{-a_0\hat{X}_2}{h(\hat{N})}-\lambda\hat{K}-\frac{\lambda(\hat{N}-\hat{X}_2-\hat{R})\hat{K}}{\hat{X}_2},$$

$$B_2 = \frac{a_0 \hat{X}_2}{h(\hat{N})} - \frac{a_0(\hat{N} - \hat{X}_2 - \hat{R})\hat{X}_2 h'(\hat{N})}{\left[h(\hat{N})\right]^2} + \lambda \hat{K}.$$

By using Gerschgorin's theorem[15] all eigen values of M_2 have negative real parts if the following inequalities holds;

$$(\gamma_0 + \alpha) < \left[\frac{a_0 \hat{X}_2}{h(\hat{N})} + \lambda \hat{K} + \frac{\lambda(\hat{N} - \hat{X}_2 - \hat{R})\hat{K}}{\hat{X}_2}\right],$$

$$\left[\frac{a_0 \hat{X}_2}{h(\hat{N})} + \lambda \hat{K}\right] < \mu,$$

$$\left[\frac{a_0 \hat{X}_2}{h(\hat{N})} + \lambda \hat{K} - \frac{a_0(\hat{N} - \hat{X}_2 - \hat{R})\hat{X}_2 h'(\hat{N})}{\left[h(\hat{N})\right]^2} + l\right] < \mu,$$

$$\lambda(\hat{N} - \hat{X}_2 - \hat{R}) < (s - s_1),$$

$$\frac{s\hat{K}^2 L_1}{\left[L(\hat{E})\right]^2} < \delta_0.$$

Hence equilibrium point $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ is locally asymptotically stable.

6. Global stability

Theorem 2: *In addition to the assumption (2.2) and (2.3), let* $L(E)$ *and* $h(N)$ *satisfy in the region* Ω, *(6.1)* $L_m \le L(E) \le L_0$ *and* $0 \le -L'(E) \le p$, *also*

(6.2) $$h_m \le h(N) \le h_0 \text{ and } 0 \le -h'(N) \le q,$$

for some positive constants L_m, h_m *and* p, q. *Let the following inequalities are satisfied*

$$\left[\frac{a_0 X_2}{h(N)} + \lambda K - \gamma_0\right]^2 < \frac{4}{3} B_3 \mu,$$

$$\left[sK\xi(E)\right]^2 < \frac{\mu s}{L(\hat{E})},$$

$$\left[\frac{a_0 X_2}{h(N)} + a_0 X_2 \eta(N)(\hat{N} - \hat{X}_2 - \hat{R}) + \lambda \hat{K} - \alpha\right]^2 < \frac{2}{3} B_3 \mu,$$

$$\left[\lambda(\hat{N} - \hat{X}_2 - \hat{R})\right]^2 < \frac{2}{3} B_3 \frac{s}{L(\hat{E})},$$

$$[l]^2 < \mu\delta_0,$$

where $B_3 = \left[\dfrac{a_0\hat{X}_2}{h(\hat{N})} + \dfrac{a_0 X_2}{h(N)} + (\alpha+\mu+\gamma_0) + \lambda\hat{K} - \dfrac{a_0(\hat{N}-\hat{R})}{h(\hat{N})}\right],$

then $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ *is globally asymptotically stable with respect to the all solution initiating in the positive orthant.*

Proof: Let us consider the following positive definite function about $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$.

$$V(X_2, R, N, K, E) = \frac{1}{2}(X_2 - \hat{X}_2)^2 + \frac{1}{2}(R-\hat{R})^2 + \frac{1}{2}(N-\hat{N})^2$$
$$+ (K - \hat{K} - \hat{K}\log\frac{K}{\hat{K}}) + \frac{1}{2}(E-\hat{E})^2.$$

Now differentiating above equation with respect to t we get,

$$\frac{dV}{dt} = (X_2 - \hat{X}_2)\frac{d\hat{X}_2}{dt} + (R-\hat{R})\frac{d\hat{R}}{dt} + (N-\hat{N})\frac{d\hat{N}}{dt} + \frac{(K-\hat{K})}{K}\frac{d\hat{K}}{dt}$$
$$+ (E-\hat{E})\frac{dE}{dt}.$$

After some algebraic manipulations and considering functions;

$$\xi(E) = \begin{cases} \dfrac{\left(\dfrac{1}{L(E)} - \dfrac{1}{L(\hat{E})}\right)}{(E-\hat{E})}, & E \neq \hat{E}, \\ -\dfrac{L'(E)}{L^2(\hat{E})}, & E = \hat{E}. \end{cases} \qquad \text{and}$$

$$\eta(N) = \begin{cases} \dfrac{\left(\dfrac{1}{h(N)} - \dfrac{1}{h(\hat{N})}\right)}{(N-\hat{N})}, & N \neq \hat{N} \\ -\dfrac{h'(N)}{h^2(\hat{N})}, & N = \hat{N}. \end{cases}$$

Then by using the assumptions of the theorem and the mean value theorem, we have,

$$|\xi(E)| < \frac{p}{L^2_m} \text{ and } |\eta(N)| < \frac{q}{h^2_m},$$

derivative of V i.e. $\dot{V}$ can be written as the sum of the quadratics,

$$
\begin{aligned}
\dot{V} = & -\frac{1}{3}a_{22}(X_2-\hat{X}_2)^2 + a_{23}(X_2-\hat{X}_2)(R-\hat{R}) - a_{33}(R-\hat{R})^2 \\
& -\frac{1}{3}a_{22}(X_2-\hat{X}_2)^2 + a_{24}(X_2-\hat{X}_2)(N-\hat{N}) - \frac{1}{2}a_{44}(N-\hat{N})^2 \\
& -\frac{1}{3}a_{22}(X-\hat{X}_2)^2 + a_{25}(X_2-X_2)(K-\hat{K}) - \frac{1}{2}a_{55}(K-\hat{K})^2 \\
& -\frac{1}{2}a_{44}(E-\hat{E})^2 + a_{45}(E-\hat{E})(K-\hat{K}) - a_{55}\frac{1}{2}(K-\hat{K})^2 \\
& -\frac{1}{2}a_{44}(N-\hat{N})^2 + a_{46}(N-\hat{N})(E-\hat{E}) - \frac{1}{2}a_{66}(E-\hat{E})^2,
\end{aligned}
$$

where

$$
\begin{aligned}
a_{22} &= -\left[\frac{a_0(\hat{N}-\hat{R})}{h(\hat{N})} - \lambda\hat{K} - (\mu+\alpha+\gamma_0) - \frac{a_0X_2}{h(N)} - \frac{a_0\hat{X}_2}{h(\hat{N})}\right], \quad a_{44} = \mu, \\
a_{23} &= -\left[\frac{a_0X_2}{h(N)} + \lambda\hat{K} - \gamma_0\right], \quad a_{45} = -sK\xi(E), \\
a_{24} &= \left[\frac{a_0X_2}{h(N)} + a_0X_2\eta(N)(\hat{N}-\hat{X}_2-\hat{R}) + \lambda K - \alpha\right], \quad a_{46} = l, \\
a_{25} &= \lambda(\hat{N}-\hat{X}_2-\hat{R}), \quad a_{55} = \frac{s}{L(\hat{E})}, \quad a_{33} = \mu, \quad a_{66} = \delta_0.
\end{aligned}
$$

Then sufficient condition for $\frac{dV}{dt}$ to be negative definite are,

(6.3) $a_{23}^2 < \frac{4}{3}a_{22}a_{33}$, $a_{24}^2 < \frac{2}{3}a_{22}a_{44}$, $a_{25}^2 < \frac{2}{3}a_{22}a_{55}$, $a_{45}^2 < a_{44}a_{55}$,
$a_{46}^2 < a_{44}a_{66}$.

So the interior equilibrium point $E_2(\hat{X}_2, \hat{R}, \hat{N}, \hat{K}, \hat{E})$ is globally asymptotically stable with respect to all the solution initiating in the region Ω.

7. Numerical simulation

In this section, we present numerical simulation to explain the applicability of the result discussed above. We choose the following values of the parameters in model (2.5) $A = 5$, $\alpha = 0.04$, $L_0 = 100$, $a_0 = 0.028$, $\gamma_0 = 0.7$, $L_1 = 0.05$, $\mu = 2$, $s = 1$, $s_1 = 0.02$, $\delta = 5$, $\lambda = 0.018$, $l = 0.02$, $Q_0 = 20$, $b = 1$.

With these values of parameters, it can be checked that the interior equilibrium E_2 exists and is given by

$\hat{X}_2 = 0.76554,$ $\hat{R} = 0.26793,$ $\hat{N} = 2.48468,$ $\hat{K} = 80.09938,$ $\hat{E} = 4.00993.$

Again with the set of parameters given above it can be verified that the conditions (5.1-5.5) in Theorem1 are satisfied. This shows that E_2 is locally asymptotically stable.

By choosing $L_m = 80, h_m = 0.2$ and $p = q = 0.01$ in Theorem 2 it can be checked that the conditions given in (6.3) are satisfied which shows that E_2 is globally asymptotically stable.

The results of numerical simulation are displayed graphically in figures 1-3. The effects of various parameters, i.e. L_0, λ *and* s on the infective population have been shown. It is noted from these figures that the infective population increases as these parameter value increase.

Figures 4-5 show that if the death rate of carrier population i.e. s_1 and recovery rate constant γ_0 increase, the infective population decreases. Also figure 6 shows that if the death rate of carrier population i.e. s_1 increases, the carrier population decreases. To display global stability of the system simulation is performed for different initial positions in figure 7. From this figure, it is clear that this equilibrium is globally stable provided that we start away from the other equilibria.

8. Conclusion

In this paper, a non linear *SIR* model with saturating contact rate is proposed and analyzed to study the spread of carrier-dependent infectious diseases, like cholera, diarrhea, gastroenteritis, etc. caused by direct contact of susceptibles with infectives and by carrier population density present in the environment. It is assumed that the density of carrier population is growing logistically. By stability analysis of ordinary differential equation, the criteria for asymptotic stability and global stability of an interior equilibrium is obtained. It is concluded from the analysis that if the intrinsic growth rate and carrying capacity of carrier population increases, the endemic level of infected human population increases. Also, when the growth rate of recovery and transmission coefficients of carrier population increases, then the infective human population decreases and increases respectively. It is also noted that as the death rate of carrier population increases, the endemic level of infective human population and carrier population decreases.

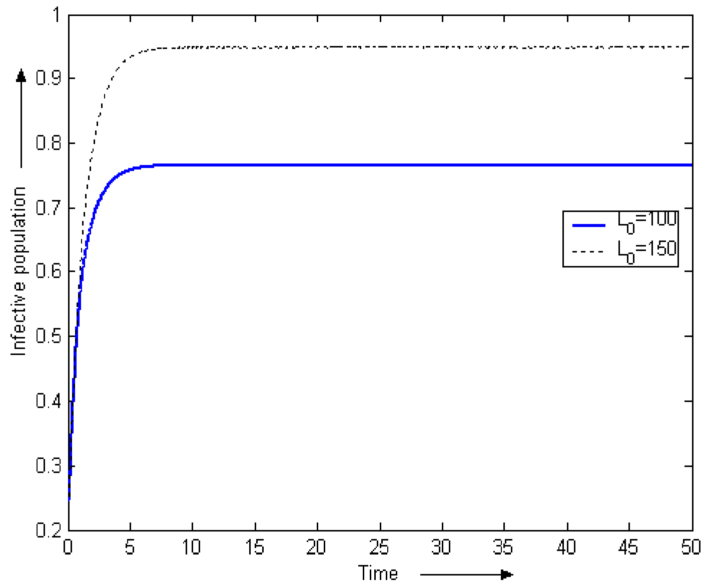

Fig.1. Variation of infective population with time for different carrying capacities of carrier population.

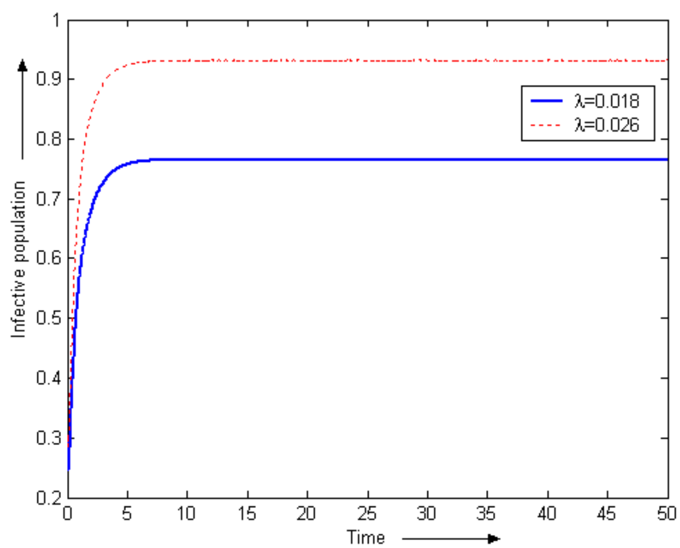

Fig.2. Variation of infective population with time for different transmitting coefficient of carrier population.

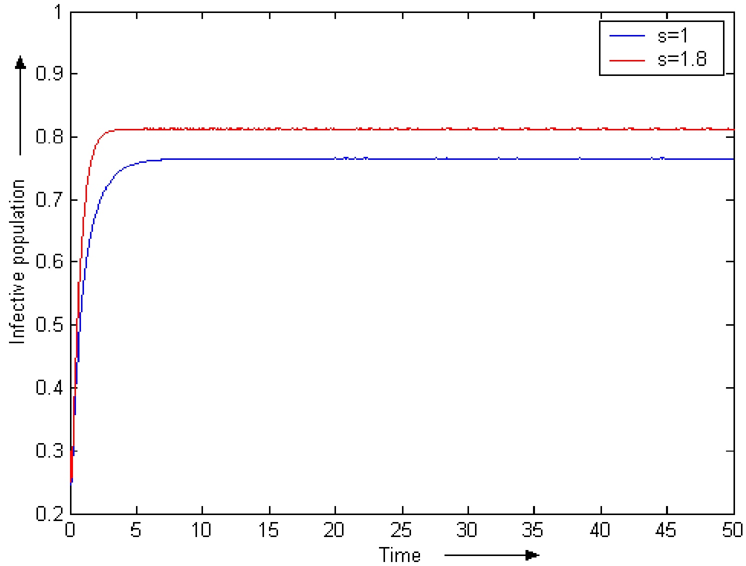

Fig.3. Variation of infective population with time for different intrinsic growth rates of carrier population

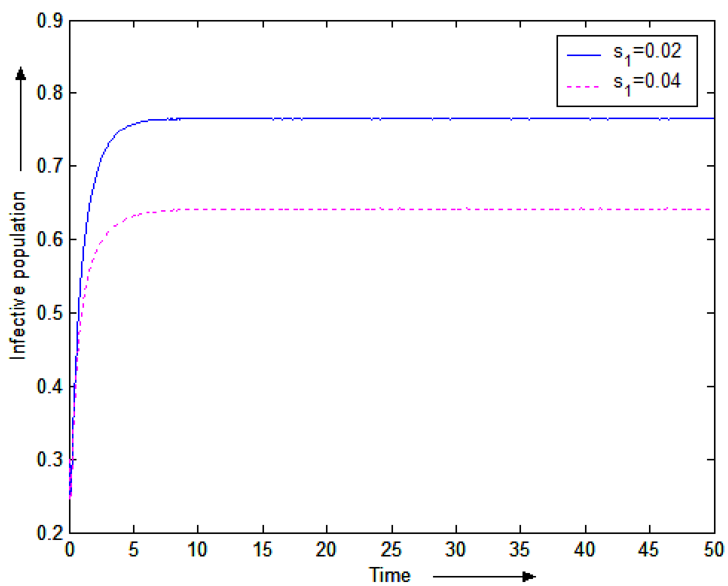

Fig.4.Variation of infective population with time for different death rate coefficients of carrier population due to the cumulative environmental discharges

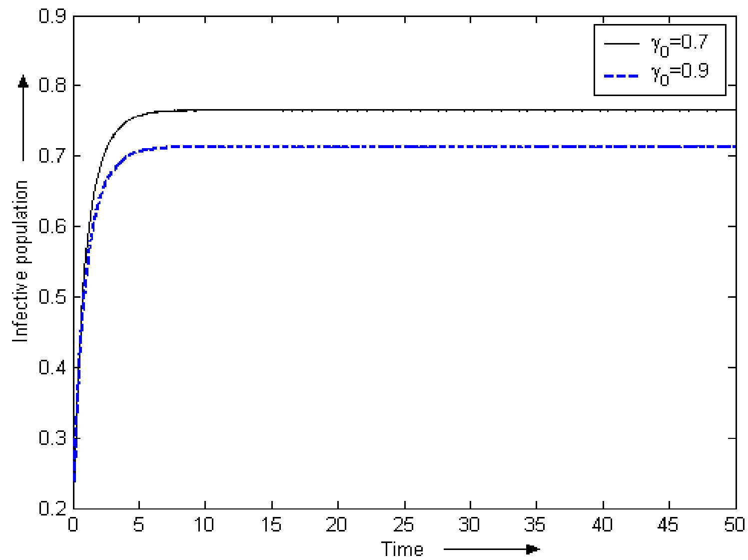

Fig.5. Variation of infective population with time for different recovery rate constant

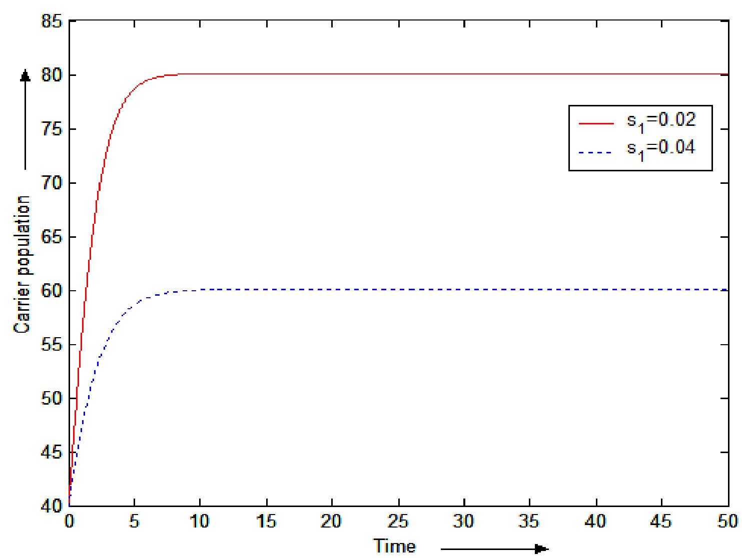

Fig.6. Variation of carrier population with time for different death rate coefficients of carrier population due to the cumulative environmental discharges

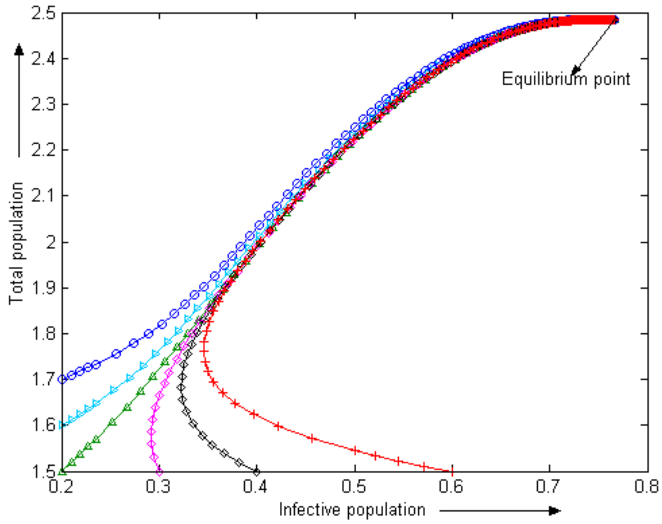

Fig.7. Variation of Total population with Infective population

References

1. J. A. P. Heesterbeek and J. A. J. Metz, The saturating contact rate in marriage and epidemic models, *J. Math. Biol.,* **31** (1993) 529.
2. D. P. Harry and S. W. Johan, *Flea of health importance and control*, US Department of Health, Education and welfare, Communicable Disease Center, Atlanta, Georgia, 1962.
3. S. Cairncross and R. G. Feachem, *Environmental Health Engineering in the Tropics*, Johan Wiley, New York, 1983.
4. I. Taylor and J. Knowelden, *Principles of Epidemiology*, Little, Brown and Co. Boston. MA, 1964.
5. H. R. Thieme and C. Castillo-Chavez, *On the role variable infectivity in the dynamics of the human immunodeficiency virus epidemic*, in C. Castillo-Chavez (Ed.), Mathematical and Statistical Approaches to AIDS Epidemiology, Lecture Notes in *Biomathematics*, **83**, Springer, Berlin, 1989, 157.
6. S. Busenberg and P. van den Driessche, Analysis of a disease transmission model with varying population size*, J. Math. Biol*., **29** (1990) 257.
7. W. R. Derrick and P. van. den Driessche, A disease transmission model in a non-constant population, *J. Math. Biol.,* **31** (1993) 495.
8. L. Q. Gao and H. W. Hethcote, Disease transmission model with density-dependent demographics, *J. Math. Biol.,* **30** (1992) 717.
9. J. Mena-Lorca and H. W. Hethcote, Dynamics models of infectious diseases as regulators of population sizes*, J. Math. Biol.,* **30** (1992) 693.
10. F. Brauer and P. van den Driessche, Models for transmission of disease with immigration of infectives, *Math. Biosci.,* **171** (2001)143.
11. N. T. J. Bailey, spatial models in the epidemiology of infectious diseases, *Lecture Notes in Biomath.,* **38** (1980) 233-261.
12. D. Greenhalgh, Some results for an SEIR epidemic models with density dependence in the death rate, *IMA J. Math. Appl. Med. Biol*., **9** (1992) 67-106.

13. J. Zhou, H. W. Hethcote, Population size dependent incidence in models for diseases without immunity, *J. Math. Biol.,* **32**(1994) 809-834.
14. H. W. Hethcote, Qualitative analysis of communicable disease models*, Math. Biosci*., **28** (1976) 335-356.
15. P. Lancaster and M. Tismenetsky, The Theory of matrices, second edition, Academic Press New York (1985) 387.
16. M. Ghosh, J. B. Shukla, P. Chandra and P. Sinha, An epidemiological model for carrier-dependent infectious diseases with environmental effect*, Int. J. Appl. Sc. Comp.,* **7**(2000)188-204.