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Analysis of a model for carrier dependent infectious diseases with sanitation as a control strategy

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Abstract

The sanitation plays a very important role to control the spread of infectious diseases and is much effective public health intervention. Inadequate sanitation is a major cause of spread of carrier dependent infectious diseases such as typhoid, dysentery and cholera. In this paper, a nonlinear mathematical model is proposed to study the effect of sanitation on the spread of such diseases in a homogeneously mixed human population. In modeling the process, it is assumed that the disease spreads directly from the infectives to susceptibles as well as indirectly by the carriers present in the environment. The density of carrier population is assumed to grow logistically but it declines due to sanitation effort applied whereas the sanitation effort also follows a logistic model with its desired increase taken directly proportional to the density of carrier population. The proposed model is also extended to an optimal control problem and is analyzed using Pontryagin Maximum Principle. The model analysis reveals that the carrier population density decreases with increase in the sanitation effort applied resulting to decrease the infective population and hence decline in the disease prevalence. Thus, the spread of carrier dependent infectious diseases can be controlled significantly if suitable sanitation effort is applied to curb the carrier population in the environment. Numerical simulations performed also support the analytical findings.

Keywords mathematical model; infectious diseases; carrier population; sanitation effort; stability; numerical simulation.

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1 Introduction

Sanitation refers to public health conditions such as the one related to clean drinking water, proper management of solid and animal waste, environmental hygiene, household cleanliness etc. These sanitation facilities aim to protect the health of people by providing a clean environment that helps reducing the

transmission of infectious diseases since the improper sanitation has always been a major cause of infectious diseases and illness. The spread of diseases due to inadequate sanitation are particularly related with poverty and infancy and it alone accounts for about 10% of the global burden of disease (Mara et al., 2010). The cost of improper sanitation may lead to economic loss, loss of education and the loss in other economic activities (such as tourism) due to polluted environment. As per (WHO) estimate in 2014, 842,000 deaths per year were attributable to unsafe sanitation, drinking water and hygiene which included 361,000 deaths of children under age five, mostly in developing countries. Despite the several efforts made for proper management of sewage disposal, use of toilets etc., 2.3 billion people lacked even basic sanitation facilities, 600 million people used limited sanitation services and 892 million people worldwide practiced open defecation (WHO/UNICEF). Improved sanitation facilities, good hygienic conditions and clean drinking water are fundamental to good health and socioeconomic development, particularly in developing countries. Since sanitation facilities are much effective public health interventions, improvement in such facilities not only reduces the morbidity and severity of various infectious diseases but also improve the lives of people in developing countries (WHO). In view of this, India has taken various steps for providing proper sanitation and has launched vigorous health campaigns like School Water and Sanitation towards Health and Hygiene (SWASTHH) and Swachh Bharat Abhiyan (Clean India Campaign) which are noted as the world's largest sanitation programs. This provided opportunities to millions of people getting access to toilets and brought about a behavioral change towards their usage (Siddiqui, 2016). These public efforts have contributed significantly to make our environment clean and free from bacteria, carriers, vectors etc. leading to decline in the spread of infectious diseases.

There are many infectious diseases which are transmitted from infectives to susceptibles through direct human-to-human contacts and also indirectly through carriers such as flies, ticks, mites, snails etc. present in the environment. The bacteria of infectious diseases such as measles, typhoid fever, leprosy, cholera, gastroenteritis, dysentery, tuberculosis, diarrhea etc. are transported by these carriers from the environment to susceptibles leading to faster spread of such diseases in human population (Gonzalez-Guzman, 1989). Some of these diseases like dysentery, gastroenteritis, cholera, typhoid fever, etc., called water borne diseases, spread by flies carrying the bacteria of these diseases into the food and water of susceptible population. The diseases such as tuberculosis and measles are spread by air borne carriers in the environment. The transmission of these infectious diseases is further aggravated due to lack of sanitation leading to unhygienic environmental conditions in a habitat which provides a very conducive environment to these carriers to flourish. In the last few decades, various modeling studies have been made to understand and analyze the spread of carrier-dependent infectious diseases like typhoid, cholera, diarrhea, etc. These studies have been made by considering the direct transmission of diseases without taking into account the role of carrier population (Agarwal and Verma, 2010; Anderson and May, 1979; Hethcote, 2000; Hsu and Zee, 2004; Keeling and Danon, 2009; Nadjafikhah and Shagholi, 2017; Nyerere et al., 2018; Zhang et al., 2020). However, to capture the realistic dynamics of the spread of infectious diseases where carriers play an important role in spreading such diseases, the effect of carrier population present in the environment must be incorporated in the models (Balamuralitharan and Radha, 2017; Das et al., 2005; Ghosh et al., 2004; Kalajdzievska and Michael, 2011; Kumar and Singh, 2013; Misra et al., 2013; Naresh and Pandey, 2009; Shukla et al., 2011; Singh, 2017; Singh et al., 2003; Singh et al., 2005; Singh et al., 2009; Tiwari et al., 2014). In particular, Singh et al. (2003) have proposed SIS and SIRS models for carrier dependent infectious diseases with environmental effects and other human population related factors. Their study reveals that the spread of infectious diseases increases as carrier population density increases. Ghosh et al., (2004) proposed an SIS model for carrier dependent infectious diseases by taking into account the direct contact of susceptibles with infectives and the indirect contact of susceptibles with carriers assuming the logistic growth for both human population density and the carrier

population density. They concluded that the spread of carrier dependent infectious disease increases with increase in the growth of carrier population density caused by conducive household discharges. Models have also been proposed to analyze the role of vaccination in controlling the spread of such infectious diseases (Manyombe et al., 2016; Naresh et al., 2008; Zhou and Cui, 2011). In particular, Naresh et al. (2008) studied a nonlinear mathematical model for the spread of carrier dependent infectious diseases incorporating the role of vaccination and found that with the high efficacy of vaccine the spread of diseases can be controlled. Some epidemiological models have also been proposed to minimize the infected individuals in the course of an epidemic using treatment and vaccination as control measures (Kumar and Srivastava, 2017; Srivastav and Ghosh, 2016; Yusuf and Benyah, 2012). Kumar and Srivastava (2017) proposed a compartmental model to analyze the effect of people's behavioral response due to information on the prevalence of the disease. Optimal control strategy has also been used to minimize possible expenditure and disease mortality (Hota et al., 2017; Lenhart and Workman, 2007; Mishra et al., 2015). Since vaccination against diseases may not always provide permanent immunity, the vaccinated people may again become infected. Thus, if people present a positive attitude towards the control of infectious diseases, the resulting behavioral change in population may play a vital role in reducing the size of the epidemic. In view of this, various studies incorporated media coverage and awareness campaigns as control measures to stop spreading of infectious diseases (Cui et al., 2007; Dubey et al., 2015; Greenhalgh et al., 2015; Liu, 2013; Misra et al., 2018; Samanta et al., 2013; Sharma and Misra, 2014; Tripathi and Naresh, 2019). For example, Samanta et al. (2013) proposed a mathematical model to study the effect of awareness programs through media on the prevalence of infectious diseases and found that the execution of awareness programs has a significant effect on curtailing the spread of diseases.

In some of the above studies, though the carrier population is explicitly modeled but the effect of sanitation has not been taken into account. The sanitation provides an important public health intervention to curb the spread of carrier dependent infectious diseases (Rai et al., 2019). Keeping this aspect in view, the objective of this paper is to model and analyze the effect of sanitation effort to control the spread of such diseases. The carrier population is assumed to grow logistically and is suppressed due to application of sanitation effort applied. Also due to limited availability of resources, the sanitation effort applied to reduce the carrier population is modeled using logistic growth and is also taken to be directly proportional to the increase in carrier population density.

2 Mathematical Formulation and Description

Assuming the homogeneously mixed population in the region under consideration, where disease spreads through direct contacts between suseptibles and infectives and indirectly through carriers present in the environment. We consider the total human population $N(t)$ divided into two subclasses: the susceptibles $X(t)$ and the infectives $Y(t)$ at any time t. The density of carrier population is denoted by $C(t)$ at any time t in the environment. As pointed out in the introduction, the unhygienic environmental conditions due to poor and inadequate sanitation provide favorable and conducive environment to the carrier population to flourish. To curb the spread of infectious diseases, a suitable sanitation effort denoted by $F_s(t)$ is applied.

It is assumed that the interaction of susceptibles with infected individuals and carrier population follows simple law of mass action so that susceptible population looses individuals on becoming infected by direct contacts with infectives with a transmission rate coefficient β and indirectly by carrier population present in the environment with a transmission rate coefficient λ. The susceptible population, however, increases due to constant immigration with the rate A. The susceptible population is further increased due to recovery of infected individuals who again become susceptible with a rate coefficient ν. The dynamics of susceptible population is, therefore, governed by the following equation,

$$\frac{dX}{dt} = A - (\beta Y + \lambda C)X - dX + \nu Y \quad .$$

(1)

As discussed above, the infective population increases due to direct interaction of susceptibles with infectives and indirectly by the carrier population present in the environment. Thus, the equation governing the dynamics of infective population is given by,

$$\frac{dY}{dt} = (\beta Y + \lambda C)X - (d + \alpha + \nu)Y.$$

(2)

where α is the disease-induced death rate. In both the classes, d represents the natural death rate.

The third equation of the model system governs the density of carrier population assumed to grow logistically. The parameter s is the growth rate of carrier population density and s_1 is the rate of decrease of carrier population due to natural factors such that $(s - s_1)$ is the intrinsic growth rate of carrier population and $L\left(\frac{(s-s_1)}{s_0}\right)$ is its carrying capacity. Since the sanitation effort is applied to curb the carrier population, it is assumed that the decrease in the carrier population density is in direct proportion to the sanitation effort applied $(i.e. s_2 C F_s)$, where s_2 denotes the depletion rate coefficient of carrier population density due to sanitation effort applied.

$$\frac{dC}{dt} = sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_s \quad .$$

(3)

The last equation of the model system governs the logistic growth of sanitation effort with intrinsic growth rate ϕ_s and carrying capacity $\frac{\phi_s}{\phi_0}$. It is also assumed that the increase in sanitation effort applied to curb the carrier population is directly proportional to the density of carrier population in the environment $(i.e. \phi C F_s)$, where ϕ is the growth rate coefficient of sanitation effort due to increase in carrier population. The decrease in sanitation effort due to its consumption in fighting against the carrier population is considered in direct proportion to the carrier population density (i.e. $\phi_1 C F_s$), where $\phi_1$is its depletion rate coefficient. Further, ψ_1 is the rate of sanitation effort applied and ψ_2 is the rate of decrease of sanitation effort due to some other factors.

$$\frac{dF_s}{dt} = \phi_s F_s - \phi_0 F_s^2 - \phi_1 C F_s + \phi C F_s.$$

(4)

with initial conditions $X(0) > 0, Y(0) \geq 0, C(0) \geq 0,\ F_s(0) > 0$ and $\phi_s = (\psi_1 - \psi_2) > 0$.

Since $N = X + Y$ we have,

$$\frac{dY}{dt} = \beta(N - Y)Y + \lambda C(N - Y) - (d + \alpha + \nu)Y;$$

$$\frac{dN}{dt} = A - dN - \alpha Y \quad ;$$

(5)

$$\frac{dC}{dt} = sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_s;$$

$$\frac{dF_s}{dt} = \phi_s F_s - \phi_0 F_s^2 - \phi_1 C F_s + \phi C F_s.$$

The continuity of right hand side of model system (5) and its derivative imply that the model is well posed for $N > 0$. All dependent variables and parameters of the model are assumed to be non-negative.

To show that the model system (5) is epidemiologically feasible, we show that all variables of the model system (5) are non-negative for all time t > 0. The following lemma is stated for this:

Lemma 2.1.1 The solution *Y(t), N(t), C(t)* and $F_s(t)$ of model system (5) with initial conditions $Y(0) \geq 0$, $N(0) > 0, C(0) \geq 0$ and $F_s(0) > 0$ are positive for all $t > 0$.

The region of attraction giving the bounds of dependent variables is stated in the form of a lemma as follows,

Lemma 2.1.2 The region of attraction Ω for the model system (5) is given by,

$$\Omega = \left\{(Y(t), N(t), C(t), F_s(t)) \in R_+^4 : 0 \leq Y \leq \frac{A}{\alpha+d}, 0 < N \leq \frac{A}{d}, 0 \leq C \leq C_m, 0 < F_s \leq F_{sm}\right\},$$

which attracts all solutions starting in the positive octant, where

$C_m = \frac{L(s-s_1)}{s_0}$ and $F_{sm} = \frac{(\phi-\phi_1)C_m+\phi_s}{\phi_0}$.

3 Equilibrium Analysis

In this section, we find the equilibria of the model system (5) to discuss the qualitative behavior around the equilibrium using stability theory of ordinary differential equations and to get the insight regarding disease dynamics and sanitation control strategy. The existence of equilibria is carried out by equating the right hand side of model system (5) to zero. We obtain the following five feasible non-negative equilibria:

1. $\boldsymbol{E_0\left(0, \frac{A}{d}, 0, 0\right)}$. This is disease-free equilibrium and its existence is obvious. It implies that in the absence of infection in the population, both directly through susceptible-infective interaction and indirectly through carrier population, no increased sanitation effort is required to be applied. In such a case, the human population will always remain at its equilibrium A/d.

2. $\boldsymbol{E_1(\bar{Y}, \bar{N}, 0, 0)}$. This is carrier-free equilibrium without sanitation effort. It implies that in the absence of carrier population in the system, no sanitation effort is required. However, the disease still persists in the population due to direct interaction of susceptibles with infectives and remains at its equilibrium $\bar{Y}$ with human population maintained at its reduced equilibrium $\bar{N}$.

3. $\boldsymbol{E_2\left(0, \bar{\bar{N}}, 0, \bar{\bar{F}_s}\right)}$. This is also disease-free equilibrium with no carriers, the existence of which is obvious where, $\bar{\bar{N}} = \frac{A}{d}$ and $\bar{\bar{F}_s} = \frac{\phi_s}{\phi_0}$. It implies that since no carrier population is present in the system and disease also does not persist, the human population will remain at its equilibrium $\bar{\bar{N}}$. Moreover, in the absence of carrier population, the sanitation effort is neither consumed in fight against carrier population nor it increases due to growth of carrier population and hence it remains at its natural level $\bar{\bar{F}}_s$.

4. $\boldsymbol{E_3\left(\ddot{Y}, \ddot{N}, \ddot{C}, 0\right)}$. This is sanitation-free equilibrium. It implies that in the absence of sanitation effort, the persistence of disease is higher and hence the human population remains at its reduced equilibrium $\ddot{N}$ with carrier population at its carrying capacity $\ddot{C}$.

5. $\boldsymbol{E^*(Y^*, N^*, C^*, F_s^*)}$. This is endemic equilibrium.

The existence of equilibria E_0 and E_2 is obvious, as stated above. In the following, we show the existence of equilibria E_1, E_3 and E^*.

3.1 Existence of equilibrium $\boldsymbol{E_1(\bar{Y}, \bar{N}, 0, 0)}$

This equilibrium can be easily obtained as,

$$\bar{Y} = \frac{\beta A - d(d+\alpha+\nu)}{\beta(\alpha+d)}, \qquad \bar{N} = \frac{A-\alpha Y}{d}.$$

which exists if $R_0 > 1$, where, the basic reproduction number $R_0 = \frac{\beta A}{d(d+\alpha+\nu)}$.

3.2 Existence of equilibrium $E_3(\ddot{Y}, \ddot{N}, \ddot{C}, \mathbf{0})$

From the model system (5) we have

$$\beta(N-Y)Y + \lambda C(N-Y) - (d+\alpha+\nu)Y = 0,$$

(6)

$$A - dN - \alpha Y = 0,$$

(7)

$$sC - \frac{s_0 C^2}{L} - s_1 C = 0 \quad ,$$

(8)

From Eq. (8) we have, $(C \neq 0)$

$$\ddot{C} = \frac{L(s-s_1)}{s_0}.$$

(9)

From Eq. (7) we have,

$$\ddot{N} = \frac{A-\alpha Y}{d}.$$

(10)

putting the values of $\ddot{C}$ and $\ddot{N}$ from Eq. (9) and (10) respectively in Eq. (6), we get a quadratic equation in $\ddot{Y}$,

$$a\ddot{Y}^2 - b\ddot{Y} - c = 0,$$

(11)

where,

$$a = \beta(\alpha + d) > 0,$$

$$b = A\beta - \lambda(\alpha + d)\frac{L(s-s_1)}{s_0} - d(d+\alpha+\nu) \text{ and}$$

$$c = \frac{A\lambda L(s-s_1)}{s_0} > 0 \text{ since } (s > s_1).$$

Since a and c are always positive, from Eq. (11) $\ddot{Y}$ has atleast one positive root by Discarte's rule of sign. Using the value of $\ddot{Y}$ so obtained, the value of $\ddot{N}$ can be found using Eq. (10).

3.3 Existence of equilibrium $E^*(Y^*, N^*, C^*, F_s^*)$

We prove the existence of endemic equilibrium E^* by solving the following system of algebraic equations obtained by setting right hand side of equations in model system (5) to zero,

$$\beta(N-Y)Y + \lambda C(N-Y) - (d+\alpha+\nu)Y = 0,$$

(12)

$$A - dN - \alpha Y = 0,$$

(13)

$$sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_s = 0,$$

(14)

$$\phi C F_s - \phi_0 F_s^2 - \phi_1 C F_s + \phi_s F_s = 0.$$

(15)

From Eq. (15) we have, $(F_s \neq 0)$

$$F_s = \frac{1}{\phi_0}[(\phi - \phi_1)C + \phi_s].$$

(16)

Using Eq. (16) in Eq. (14), we get, $(C \neq 0)$

$$C^* = \frac{L(\phi_0(s-s_1)-s_2\phi_s)}{s_0\phi_0+Ls_2(\phi-\phi_1)}.$$

(17)

provided $\phi_0(s-s_1) > s_2\phi_s$.

Using the value of C^* from Eq. (17) we can find F_s^* from Eq. (16).

Now on solving Eq. (12) and (13) and using the value of C^*, we get a quadratic equation in Y^* as follows,

$$p_1Y^{*2} - p_2Y^* - p_3 = 0,$$

(18)

where,

$$p_1 = \beta(\alpha+d) > 0,$$

$$p_2 = \beta A - (\alpha+d)\lambda\frac{L(\phi_0(s-s_1)-s_2\phi_s)}{s_0\phi_0+Ls_2(\phi-\phi_1)} - d(d+\alpha+\nu) \text{ and}$$

$$p_3 = A\lambda\frac{L(\phi_0(s-s_1)-s_2\phi_s)}{s_0\phi_0+Ls_2(\phi-\phi_1)} > 0.$$

Now using Descarte's rule of sign, the quadratic Eq. (18) has a unique positive real root of Y^* if either $p_2 > 0$ or $p_2 < 0$. After finding the value of Y^*, we can find the value of N^* showing the existence of unique endemic equilibrium E^*.

From the eq.(18), the following remark can be made**,**

Remark 1. Effect of s_2 on Y^*

To show the effect of the parameter s_2 on Y^*, differentiating Eqn. (18) with respect to the parameter s_2 and using it again, we obtain,

$$\frac{dY^*}{ds_2} = -\frac{\lambda L\phi_0Y^*\left(s_0\phi_s+L(s-s_1)(\phi-\phi_1)\right)\left(A-Y^*(\alpha+d)\right)}{\left(s_0\phi_0+Ls_2(\phi-\phi_1)\right)^2(p_1Y^{*2}+p_3)} < 0. \quad (19)$$

This indicates that the equilibrium number of infective population decreases with increase in s_2, the control parameter representing the depletion rate coefficient of carrier population density due to sanitation effort. In a similar way, we can show that $\frac{dY^*}{d\beta} > 0$ and $\frac{dY^*}{d\lambda} > 0$, $\frac{dY^*}{dA} > 0$. Thus, we conclude that the equilibrium number of infective population decreases with increase in control parameters whereas it increases if the direct and indirect transmission rate coefficient increases. The disease, however, becomes more endemic due to immigration of susceptibles in the population.

4 Sensitivity Analysis for R_0

Sensitivity indices measure how the basic reproduction number R_0 changes in response to the small shifts in the value of a parameter. The initial disease transmission is directly related to the basic reproduction number R_0 which is defined as the average number of secondary infections produced by one infective over the duration of the infectious period into a completely susceptible population. By using next generation matrix method Van den Driessche and Watmough (2002), the basic reproduction number (R_0) associated to the differential system (5) is given by:

$$R_0 = \frac{\beta A}{d(d+\alpha+\nu)} .$$

(20)

If $R_0 < 1$, then the disease cannot pervade the population and the infection will die out over a period of time. If $R_0 > 1$, then the infection triggers an epidemic in the population.

The normalized forward sensitivity index of R_0, that depends differentially on a parameter ρ is defined by

$$\gamma_\rho^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0} \quad . \tag{21}$$

Proceeding in a similar manner, we determine and measure the sensitivity indices of R_0 using the parameter values as given in Section 7. Table 1 shows the sensitivity indices of R_0 for the parameters. The parameters are ordered from most sensitive to least. In the sensitivity indices of R_0, since $\gamma_A^{R_0} = 1$ that means, increasing(or decreasing) the recruitment rate of population (A) by 10%, increases(or decreases) the reproduction number R_0 by 10%. Similarly increasing (or decreasing) the transmission coefficient rate of disease (β) by 10%, increases (or decreases) the reproduction number R_0 by 10%. Further, increasing (or decreasing) the natural death rate d by 10 % decreases (or increases) R_0 by 13.947 % and in the same manner increasing (or decreasing) the recovered rate (ν) and the disease induced death rate (α) by 10%, decreases (or increases) R_0 by 5.263% and 0.789% respectively.

Table 1 Sensitivity indices of R_0 evaluated at the parameter values given in Section 7.

Parameter	Sensitivity index
d	-1.3947
A	+1
β	+1
ν	-0.5263
α	-0.0789

Now the effect of various parameters is shown on Reproduction number R_0 in Figs. 1 and 2. In Fig. 1, the effect of variation in β and A is depicted on R_0. It is noted that as the value of β and A increases, the value of R_0 also increases. The effect of other parameters d and α can be seen in Fig. 2.

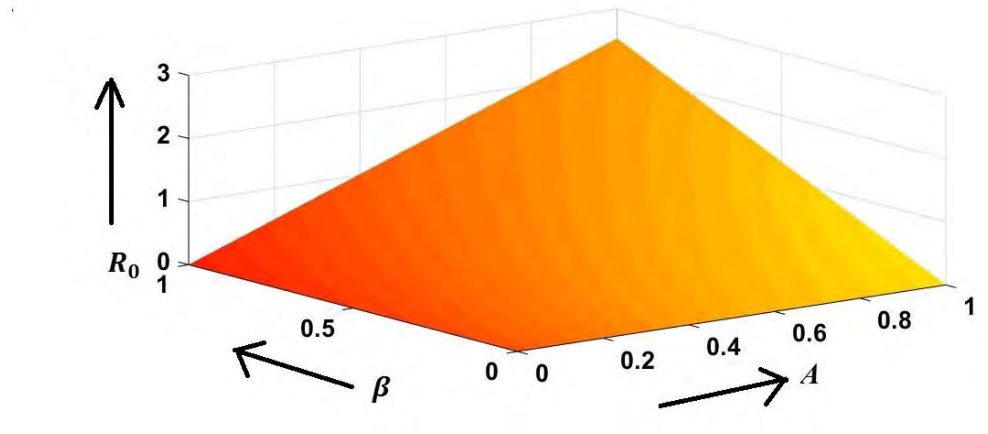

Fig. 1 Surface plot showing the effect of β and A on R_0.

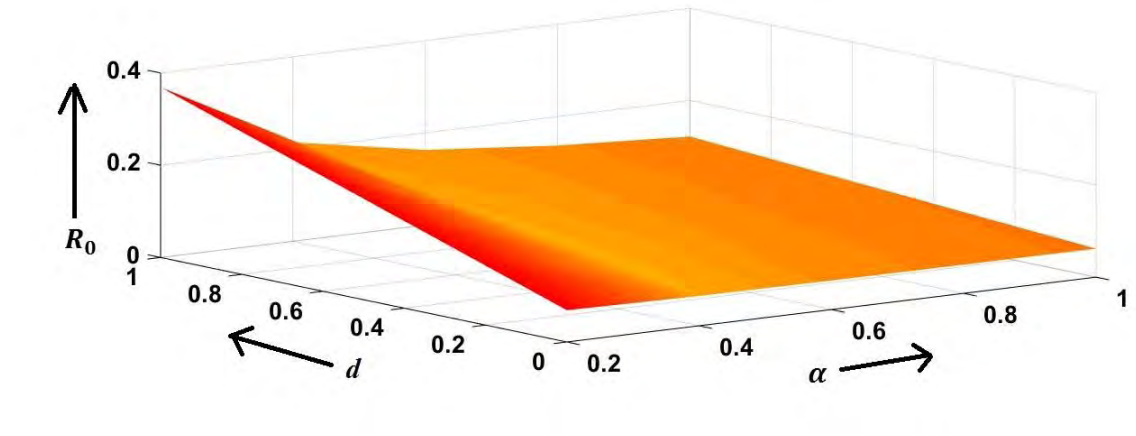

Fig. 2 Surface plot showing the effect of d and α on R_0.

5 Stability Analysis

In the following, we present the results of local and nonlinear stability analysis of equilibria $E_0, E_1, E_2,\ E_3$ and E^* using the method of Jacobian matrix corresponding to the model system (5) and Liapunov method. Thus, we get the following results regarding local and nonlinear stability of different equilibria.

Theorem 5.1 The equilibria E_0, E_1, E_2 and E_3 are unstable and the endemic equilibrium E^* is locally asymptotically stable provided the following condition is satisfied,

$$\lambda^2(N^* - Y^*)^2 < \left(\beta Y^* + \frac{\lambda C^* N^*}{Y^*}\right)\frac{s_0 C^*}{L} \quad . \tag{22}$$

Proof : By computing the Jacobian matrix for the model system (5) at the equilibrium $E_i(i = 0,1,2,3)$, we can

easily find that these equilibria are unstable. Further, to establish the local stability of endemic equilibrium E^*, we consider the following positive definite function

$$U_1 = \frac{1}{2}(m_0 y^2 + m_1 n^2 + m_2 c^2 + m_3 f_s^2),$$

where y, n, c, f_s are small perturbations about E^*, defined as $Y = y + Y^*$, $N = n + N^*$, $C = c + C^*$ and $F_s = f_s + F_s^*$. The positive constants $m_i (i = 0, 1, 2, 3)$ are obtained as

$$m_0 = 1, m_1 = \frac{\beta Y^* + \lambda C^*}{\alpha},\ m_2 = 1 \text{ and } m_3 = \frac{s_2 C^*}{(\phi - \phi_1) F_s^*}.$$

We get $\frac{dU_1}{dt}$ to be negative definite showing that U_1 is a Liapunov function and hence E^* is locally asymptotically stable provided the condition (22) is satisfied.

Theorem 5.2 The endemic equilibrium E^* is nonlinearly asymptotically stable in the region Ω provided the following conditions are satisfied:

$$2\alpha\left(\beta + \frac{\lambda C_m}{Y^*}\right) < \beta d \quad , \tag{23}$$

$$m = \beta\left(\frac{s_0}{L}\right) - \lambda^2\left(\frac{N^* - Y^*}{Y^*}\right)^2 > 0 \quad . \tag{24}$$

Proof : We consider the following positive definite function to establish the nonlinear stability of endemic equilibrium E^*,

$$U_2 = k_0\left(Y - Y^* - Y^* \ln\frac{Y}{Y^*}\right) + \frac{k_1}{2}(N - N^*)^2 + k_2\left(C - C^* - C^* \ln\frac{C}{C^*}\right) + k_3\left(F_s - F_s^* - F_s^* \ln\frac{F_s}{F_s^*}\right),$$

where, $k_i (i = 0,1,2,3)$ are positive constants obtained as

$$k_0 = 1, k_1 = \frac{1}{\alpha}\left(\beta + \frac{\lambda C_m}{Y^*}\right),\ k_2 = 1 \text{ and } k_3 = \frac{s_2}{(\phi - \phi_1)}.$$

We get $\frac{dU_2}{dt}$ to be negative definite showing that U_2 is a Liapunov function and hence E^* is nonlinearly asymptotically stable provided the condition (23) and (24) are satisfied.

Remark 2. If the transmission coefficient λ of disease through indirect contact of susceptibles with carrier population tends to zero, then condition (24) will be satisfied automatically showing that λ has a destabilizing effect on the model system (5).

6 The Optimal Control Model

In this section, the model system (5) is extended for the formulation of optimal control problem,

$$\frac{dY}{dt} = \beta(N - Y)Y + \lambda C(N - Y) - (d + \alpha + \nu);$$

$$\frac{dN}{dt} = A - dN - \alpha Y;$$

(25)

$$\frac{dC}{dt} = sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_s;$$

$\frac{dF_s}{dt} = \phi_s F_s - \phi_0 F_s^2 - \phi_1 C F_s + u(t) C F_s.$

satisfying the initial conditions $X(0) > 0, Y(0) \geq 0, C(0) \geq 0, F_s(0) > 0$. The control parameter $u(t)$ (say) represents the control of carrier population density by applying sanitation effort. Our main aim is to maximize the rate of sanitation effort with minimal cost of control to minimize the infective population.

The control parameter $u(t)$ is a Lebesgue measurable function on a finite interval $[0, T_f]$. The objective functional for the above problem is given by,

$J(u) = \int_0^{T_f} (PY(t) + Qu^2(t))\, dt,$

where the parameter $P > 0$ and $Q > 0$ represents the weight constant which help to balance the units of integrand. For simplicity, we denote $u(t) = u$.

Our objective in this section is to find the optimal control $u^*(t)$ such that

$J(u^*) = \min_{u \in U} J(u)$, (26)

where U, the control set is defined as

$U = \{u(t)$: Lebesgue measurable and $0 \leq u(t) \leq 1$ for $t \in [0, T_f]\}$.

6.1 Existence of the control

Theorem 6.1.1 There exist an optimal control parameter $u^* \in U$ for the system (25) and Eq. (26).

Proof : Here the state and control variables are positive. From the prior bounds, the solutions of the system (25) are bounded and right hand side functions of system (25) satisfy the Lipschitz condition with respect to state variables. Then by applying Picard's Lindelof theorem, we see that the set of solution of system (25) with control variable in U is non-empty. The control set U is closed and convex by definition and the system (25) can be written as a linear function in control variable u with coefficient depending on state variable and time. Furthermore, the integrand of the functional $(PY + Qu^2)$ is convex due to quadratic nature of control variable in the control. Also set U and the state variables are bounded. Thus, we conclude that there is a control pair u^* such that $J(u^*) = \min(J(u))$.

6.2 Characterization of optimal control functions

Theorem 6.2.1 Let u^* be the optimal control parameter and Y^*, N^*, C^* and F_s^* are corresponding optimal state variables of the control system (25) and Eq. (26). Then there exists adjoint variable $\lambda = (\lambda_1, \lambda_2, \lambda_3, \lambda_4) \in R^4$ that satisfies the following equations:

$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial Y}, \frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial N}, \frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial C}$ and $\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial F_s}$,

with transversality conditions

$\lambda_1(T_f) = 0, \lambda_2(T_f) = 0, \lambda_3(T_f) = 0$ and $\lambda_4(T_f) = 0$. (27)

The optimal control u^* is given as,

$u^* = \min\left\{max\left\{0, -\frac{\lambda_4 C F_s}{2Q}\right\}, 1\right\}.$

Proof: By Pontryagin Maximum principle, there exists adjoint variables $\lambda_1, \lambda_2, \lambda_3$ and λ_4 which satisfies the following equations:

$\frac{d\lambda_1}{dt} = -\frac{\partial H}{\partial Y} = -P - \lambda_1(\beta N - 2\beta Y - \lambda C - (d + \alpha + \nu)) + \lambda_2 \alpha;$

$$\frac{d\lambda_2}{dt} = -\frac{\partial H}{\partial N} = -\lambda_1(\beta Y + \lambda C) + \lambda_2 d;$$

$$\frac{d\lambda_3}{dt} = -\frac{\partial H}{\partial C} = -\lambda_1\big(\lambda(N-Y)\big) + \lambda_3\left(s - \frac{2s_0C}{L} - s_1 - s_2F_s\right) - \lambda_4(uF_s - \phi_1F_s);$$

$$\frac{d\lambda_4}{dt} = -\frac{\partial H}{\partial F_s} = \lambda_3 s_2 C - \lambda_4(uC - 2\phi_0F_s - \phi_1C + \phi_s).$$

with transversality conditions given in (27) where Hamiltonian H is defined as,

$$H = PY + Qu^2 + \lambda_1\dot{Y} + \lambda_2\dot{N} + \lambda_3\dot{C} + \lambda_4\dot{F_s},$$

$$= PY + Qu^2 + \lambda_1(\beta Y(N-Y) + \lambda C(N-Y) - (d+\alpha+\nu)Y) + \lambda_2(A - dN - \alpha Y)$$

$$+\lambda_3\left(sC - \frac{s_0C^2}{L} - s_1C - s_2CF_s\right) + \lambda_4(uCF_s - \phi_0F_s^2 - \phi_1CF_s + \phi_sF_s).$$

Now from optimality condition, we have

$\frac{\partial H}{\partial u} = 0$ at $u = u^*$,

Thus we get,

$$u^* = \min\left\{max\left\{0, -\frac{\lambda_4CF_s}{2Q}\right\}, 1\right\}.$$

7 Numerical Simulation and Discussion

We give here the numerical solution of the model system (5) to show the existence of equilibrium values and to check the feasibility of stability conditions. For this, we integrate the systems by fourth order Runge-Kutta method using MATLAB with the following set of parameter values,

$A = 120, \beta = 0.0005, \lambda = 0.00002, \nu = 0.2, d = 0.15, \alpha = 0.03, s = 0.4, s_0 = 0.9,$

$s_1 = 0.2, s_2 = 0.0004, \phi = 0.5, \phi_0 = 0.26, \phi_1 = 0.04, \psi_1 = 0.3, \psi_2 = 0.003, L = 500.$

The optimality system is solved numerically by using forward-backward sweep method (Lenhart and Workman, 2007), with initial condition $Y(0) = 10, N(0) = 650, C(0) = 100, F_s(0) = 150$ and choosing the weight constant $P = 10$ and $Q = 10$ & $U_{max} = 1$.

The equilibrium values of endemic equilibrium $E^*$is computed as,

$Y^* = 63.544, N^* = 787.291, C^* = 79.572, F_s^* = 141.924$.

The eigenvalues corresponding to Jacobian matrix of endemic equilibrium $E^*$are: -0.0630, -0.1385, -0.1439, -36.8996. Since all the eigenvalues are found to be negative, therefore, the endemic equilibrium E^* is locally asymptotically stable for the above set of parameter values. The results of the numerical simulation are displayed graphically in Figs. 3-13. In Fig.3, we have shown the solution trajectories for initial starts, as given below, of the total human population(N), infective population (Y) and carrier population density (C) which approach towards equilibrium point showing that endemic equilibrium E^* is nonlinearly asymptotically stable. The initial starts of all trajectories are taken as follows,

(1) $Y(0) = 100,\ N(0) = 450, C(0) = 200, F_s(0) = 150.$
(2) $Y(0) = 150,\ N(0) = 500,\ C(0) = 250, F_s(0) = 150.$
(3) $Y(0) = 200, N(0) = 550,\ C(0) = 300, F_s(0) = 150.$
(4) $Y(0) = 250,\ N(0) = 600,\ C(0) = 350, F_s(0) = 150.$

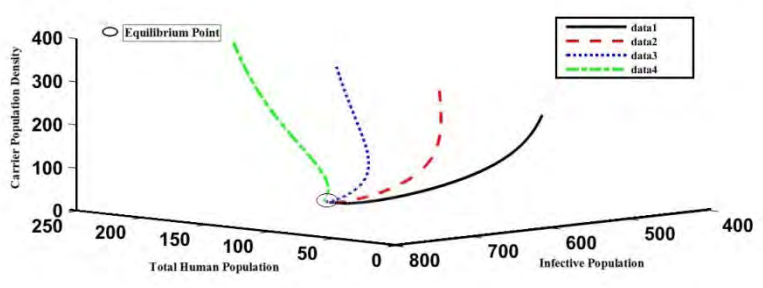

Fig. 3 Variation of total human population with infective population and carrier population density.

Fig. 4 and 5 show the variation of carrier population density and that of infective population respectively with time *t* for distinct values of parameter s, the growth rate of carrier population density. It is seen that with increase in the growth rate of carrier population, the carrier population density increases (Fig. 4) which ultimately increases the infective population (Fig. 5). This implies that the increased carrier population density which leads to increase the infective population needs to be controlled by using suitable sanitation effort to curb the carrier population density. In Fig. 6 and 7, the variation of carrier population density and that of infective population, respectively, is displayed with time *t* for distinct values of rate of decrease of carrier population due to sanitation effort i.e. s_2. It is found that as the rate of decrease of carrier population due to sanitation effort increases, the carrier population declines (Fig. 6) and consequently infective population also decreases (Fig. 7). Thus, if proper sanitation effort is applied to eradicate the carrier population density present in the environment, the spread of carrier dependent infectious diseases can be controlled. The effect of ϕ, the growth rate coefficient of sanitation effort due to increased carrier population density is shown on carrier population density and infective population with time *t* in Figs. 8 & 9 respectively. From these figures, it is observed that with increase in the rate of sanitation effort applied due to increased carrier population, the carrier population density present in the environment declines (Fig. 8). This decrease in the carrier population density due to increased sanitation effort ultimately reduces the infective population (Fig. 9). This implies the increased sanitation effort, applied in proportion to increased carrier population, not only helps diminish the carrier population density but subsequently reduce the spread of disease in the population. Thus, the proper implementation of sanitation strategy applied for curbing the carrier population density present in the environment can be of immense help to control the spread of carrier dependent infectious diseases in the population. In Figs. 10 and 11, the effect of optimal control is shown on carrier population density and infective population respectively. It is seen from these figures that the carrier population density (Fig. 10) and infective population (Fig. 11) have reduced significantly when optimal control is applied than without optimal control. The control profile $u(t)$ with time *t* can be seen as given in Fig. 12. In Fig. 13, the nonlinear stability condition m (condition 24) with respect to the critical parameter λ is plotted to study the effect of the parameters on stability condition. It can be seen from Fig. 13 that m remains positive for λ < 0.0000825 and becomes negative for λ > 0.0000825. This implies that stability condition (24) is satisfied for 0 < λ < 8.25E-5. Hence, λ has destabilizing effect on the model system (5).

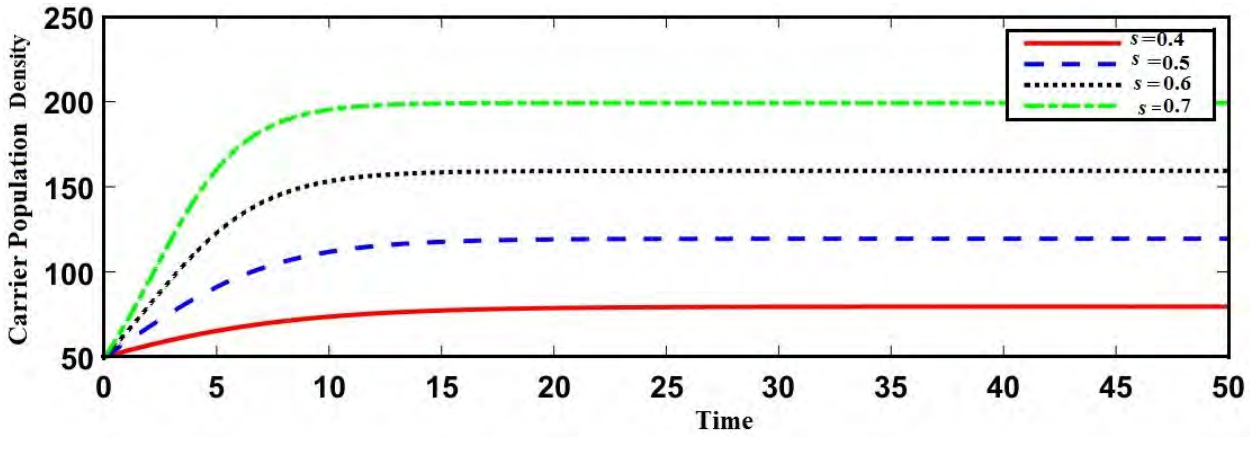

Fig. 4 Variation of carrier population density with time for distinct values of s.

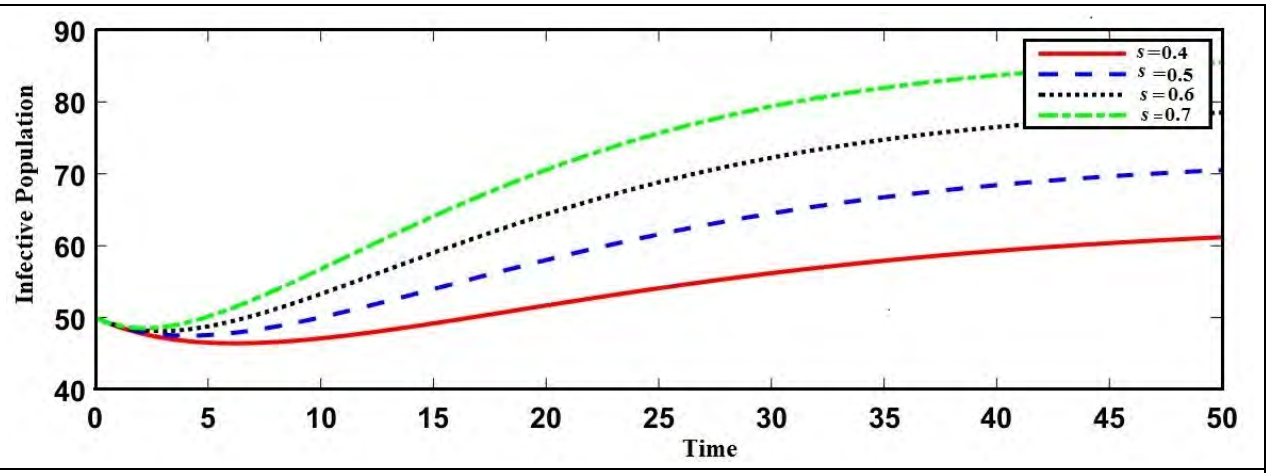

Fig. 5 Variation of infective population with time for distinct values of s.

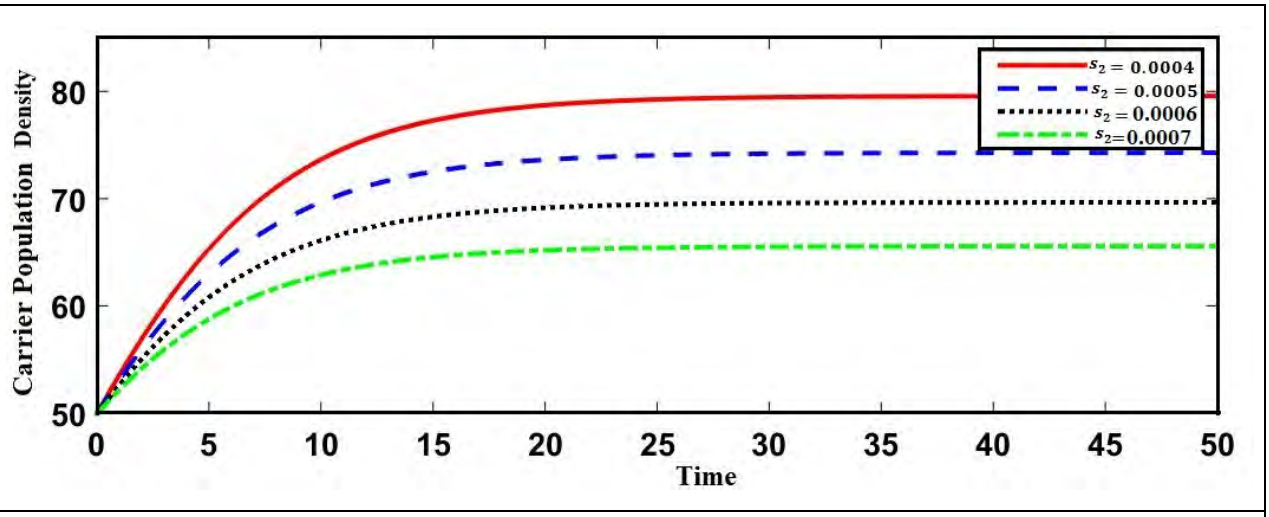

Fig. 6 Variation of carrier population density with time for distinct values of s_2.

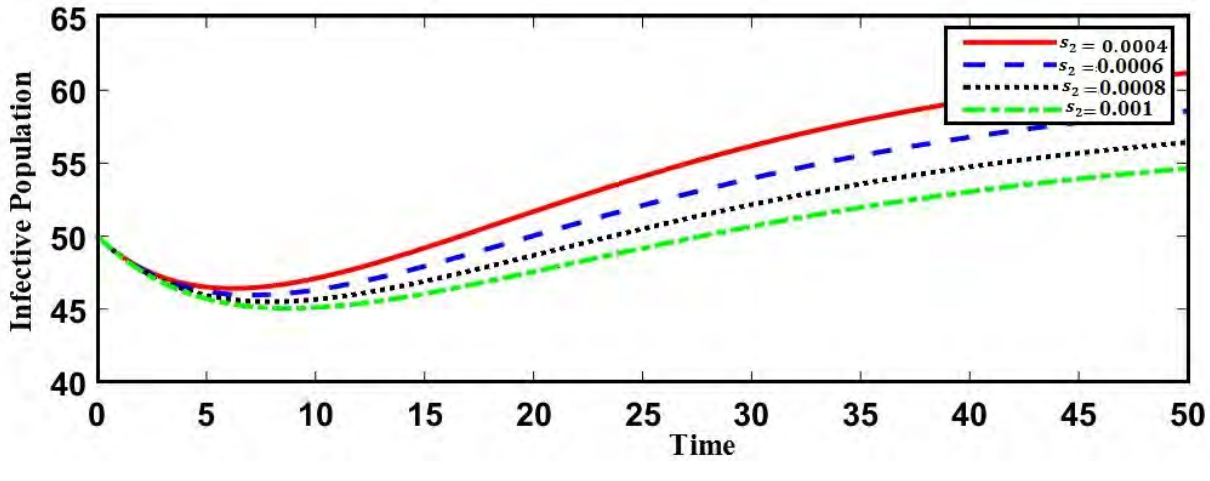

Fig. 7 Variation of infective population with time for distinct values of s_2.

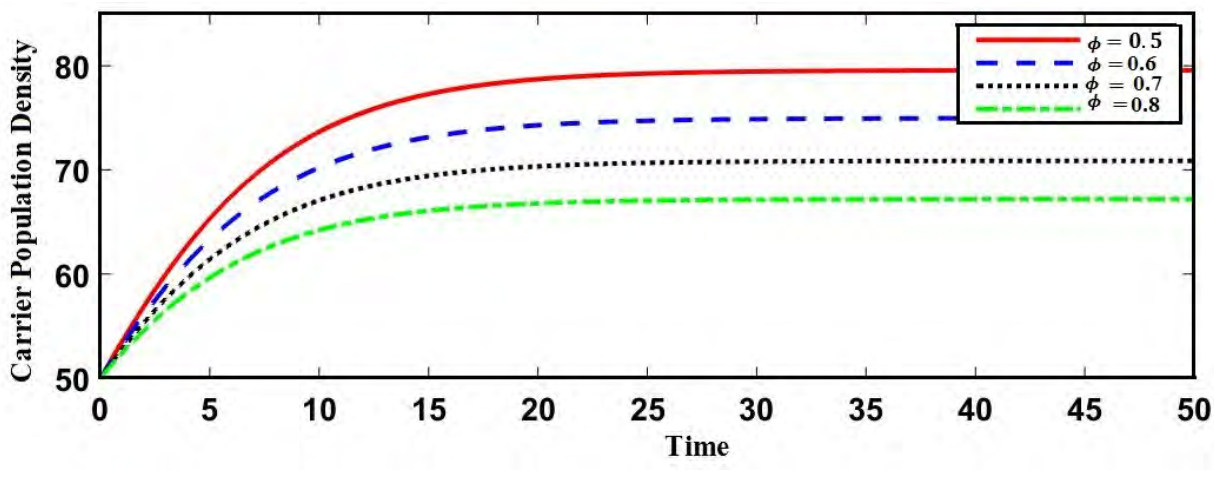

Fig. 8 Variation of carrier population density with time for distinct values of ϕ.

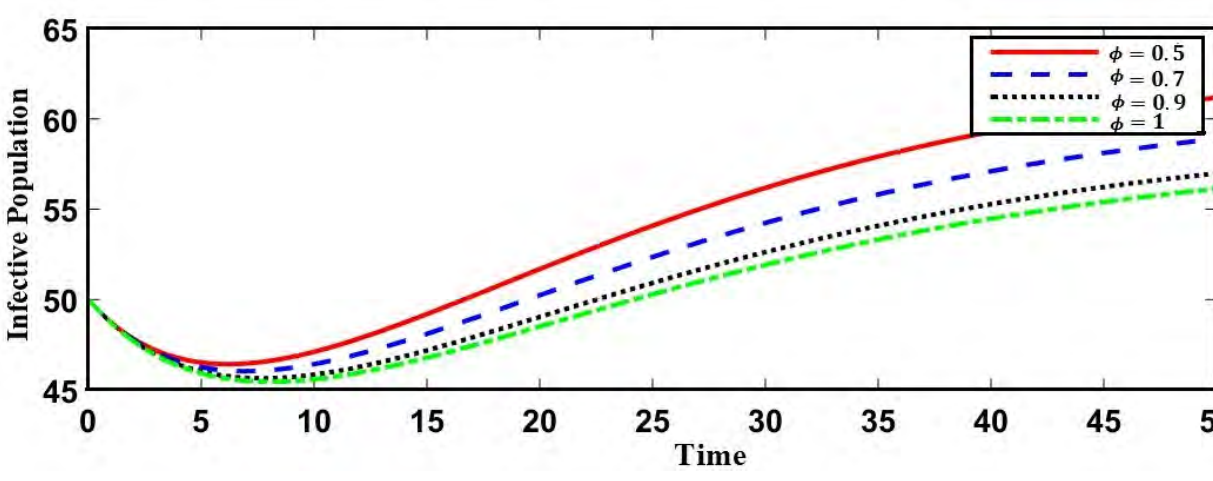

Fig. 9 Variation of infective population with time for distinct values of ϕ.

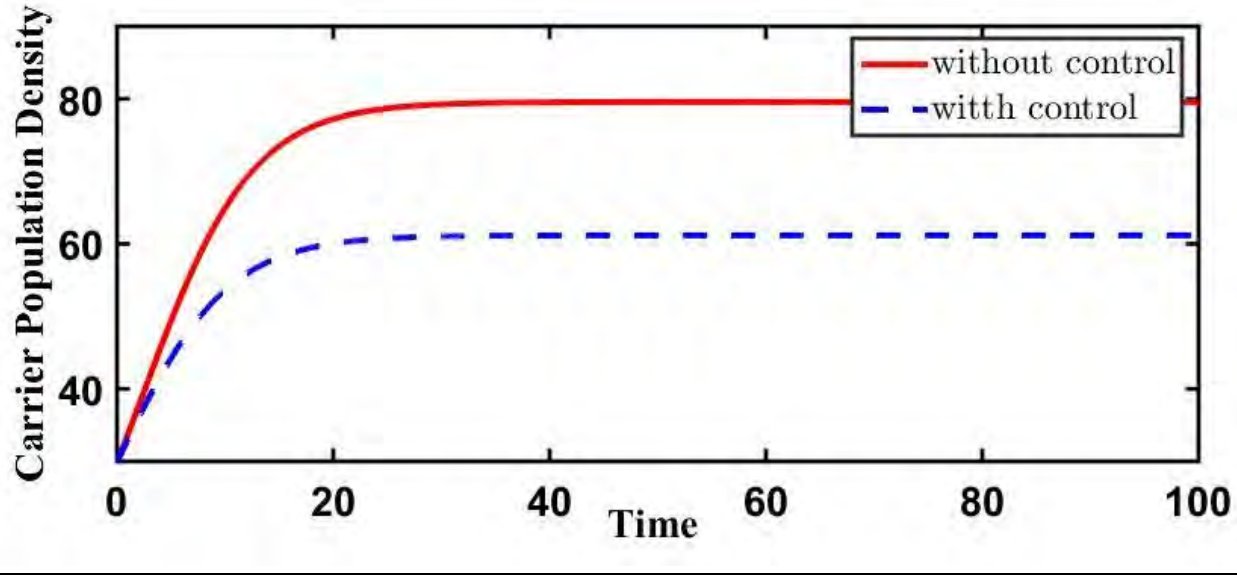

Fig. 10 Variation of carrier population density with and without optimal control.

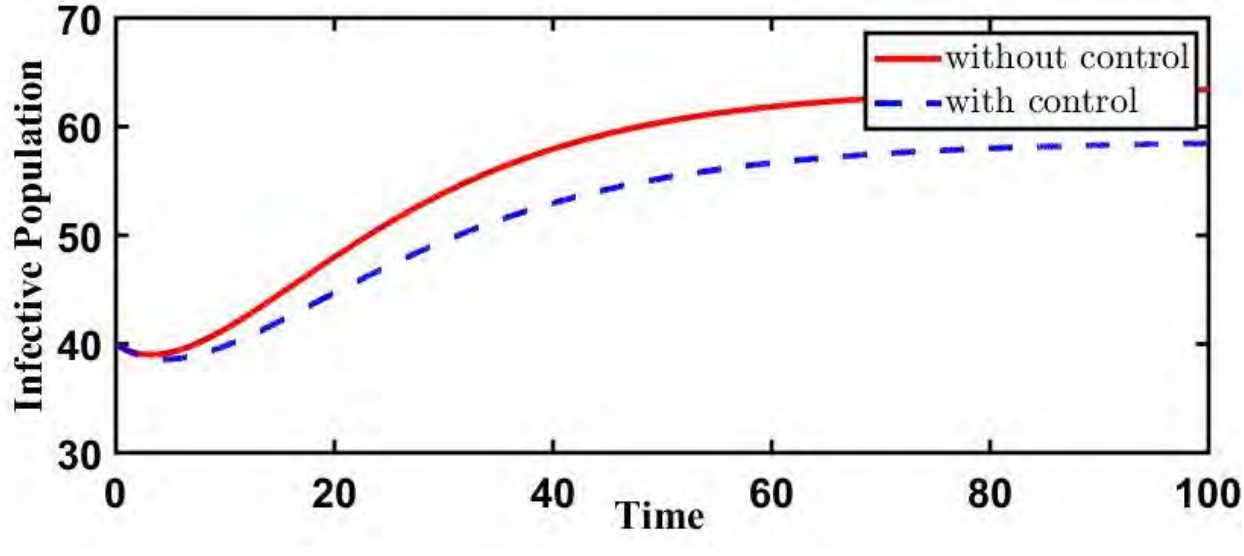

Fig. 11 Variation of infective population with and without optimal control.

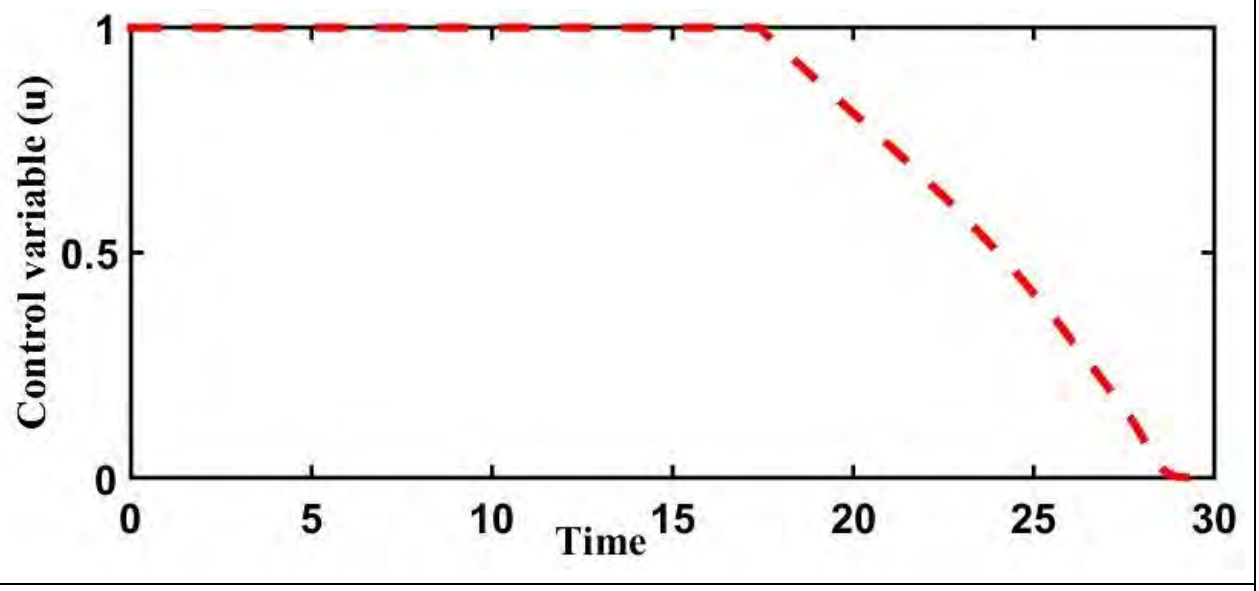

Fig. 12 Control profile of u with time.

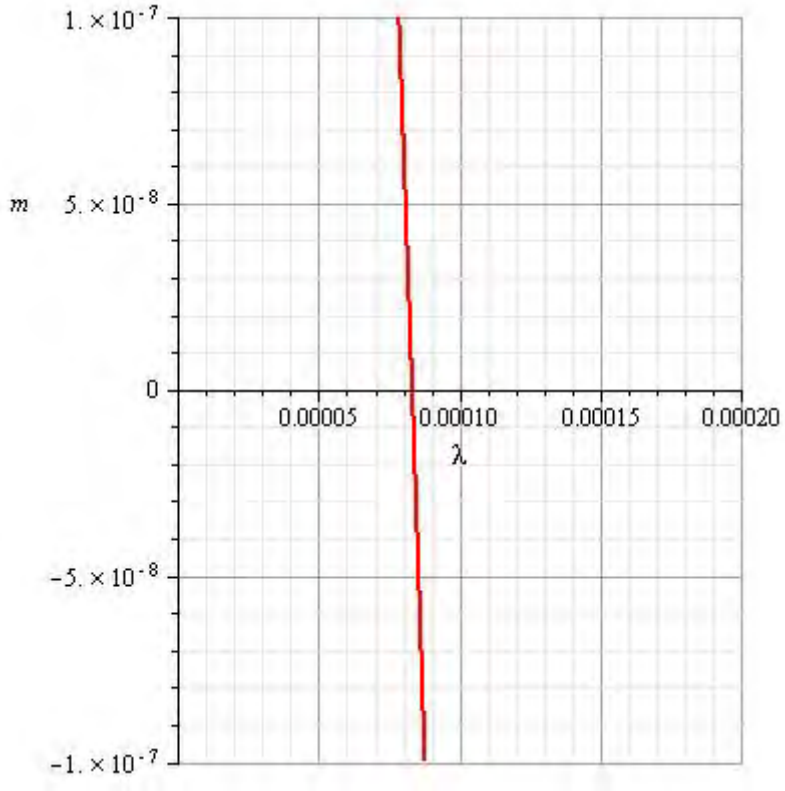

Fig. 13 Variation of stability condition m with λ.

8 Conclusions

In this paper, a nonlinear mathematical model is proposed to study the effect of sanitation effort on the spread of carrier dependent infectious diseases in a homogeneously mixed human population. It is assumed that the infectious diseases spread through direct human-to-human contacts between susceptibles and infectives and indirectly through carrier population present in the environment. In the modeling process, it is assumed that the

density of carrier population grows logistically with its intrinsic growth rates. Since the sanitation effort is applied to curb the carrier population density, it is assumed that the decrease in the carrier population density is in direct proportion to the sanitation effort applied. The sanitation effort applied is also assumed to follow logistic model with its intrinsic growth rate and carrying capacity. Further, the increase in sanitation effort applied to curb the carrier population is taken to be directly proportional to the density of carrier population in the environment. However, the sanitation effort applied decreases due to its consumption in fighting against the carrier population and is considered in direct proportion to the carrier population density. The proposed model has five non-negative equilibria. The model has been analyzed using the stability theory of differential equations and numerical simulation and certain inferences have been drawn by establishing the local and nonlinear stability results. The effect of various parameters and optimal control on carrier population density and infective population are shown graphically for different parameter values. The analysis of the model reveals that the increase in sanitation effort applied helps to eradicate the carrier population density present in the environment which ultimately reduces the infective population and hence the spread of disease in the population is controlled. Thus, the spread of carrier dependent infectious diseases can be controlled significantly if suitable sanitation effort is applied to curb the carrier population in the environment.

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