Applications and Applied Mathematics: An International Journal (AAM)

Volume 18 | Issue 2 Article 6

6-2023

(R2032) Modeling the Effect of Sanitation Effort on the Spread of Carrier-dependent Infectious Diseases due to Environmental Degradation

Ram Naresh
Harcourt Butler Technical University

Sandhya Rani Verma
Harcourt Butler Technical University

J. B. Shukla
Innovative Internet University for Research (A Think Tank)

Manju Agarwal
University of Lucknow

Available at
http://pvamu.edu/aam
Appl. Appl. Math.
ISSN: 1932-9466

Applications and Applied Mathematics:
An International Journal
(AAM)

Vol. 18, Issue 2 (December 2023), Article 6, 24 pages

Modeling the Effect of Sanitation Efforts On the Spread of Carrier-dependent Infectious Diseases Due to Environmental Degradation

[1]Ram Naresh, [1*]Sandhya Rani Verma, [2]J.B. Shukla and [3]Manju Agarwal

[1]Department of Mathematics
School of Basic & Applied Sciences
Harcourt Butler Technical University
Kanpur- 208002, India
ramntripathi@yahoo.com; verma.sandhya.15@gmail.com

[2]Innovative Internet University
for Research (A Think Tank)
Kanpur- 208017, India
jbs@iitk.ac.in

[3]Department of Mathematics & Astronomy
University of Lucknow
Lucknow-226007, India
manjuak@yahoo.com

*Corresponding Author

Received: October 11, 2022; Accepted: August 16, 2023

Abstract

In the present study, an SIS model is proposed and analyzed to study the effect of sanitation effort in controlling the spread of carrier-dependent infectious diseases in a human habitat due to environmental degradation. The dynamics of the model consist of six dependent variables, the susceptible population density, infective population density, carrier population density, cumulative density of environmental degradation and the density of sanitation effort applied on carrier population and degraded environment. In the modeling process, the carrier population density and that of sanitation effort are modeled logistically and the degradation of the environment is assumed to be directly proportional to the population in the habitat. The analysis of the model is performed by using the stability theory of differential equations and numerical simulations. The study of the model shows that as the degradation of environment increases, the density of the carrier population increases which ultimately increases the infective population. Further, the result shows that by applying suitable sanitation effort on the carrier population density and on the cumulative density of environmental degradation, the carrier population density decreases and hence the infective population. Thus, it is very important to keep our environment clean by applying proper sanitation to prevent the spread of carrier-dependent infectious diseases.

Keywords: Mathematical model; Carrier population; Environmental degradation; Sanitation SIS model; Logistic growth; Numerical simulation; Stability; Local aymptomatic stable; Nonlinear aymptomatic stable

MSC 2010 No.: 34D20, 92D30

1. Introduction

The spread of infectious diseases has caused tremendous harm to the human population. Several efforts have been applied in controlling the spread of infectious diseases for decades. Success is achieved in controlling some diseases, but several harmful diseases still persist in the human population. Various mathematical models have been proposed to identify the key parameters which are responsible for the spread of infectious diseases (Harry and Kent (1961), Hsu and Zee (2004), Keeling and Rohani (2008), Brauer and Castillo-Chavez (2012) and Dubey et al. (2013)). In particular, Hsu and Zee (2004) developed simple models for the global spread of infectious diseases; they emphasize on human mobility via air travel and the variation of public health infrastructure from region to region. They also derived formulas relating the total and peak number of infections in two countries to the rate of travel between them and their respective epidemiological parameters. In spite of several measures taken to control the spread of infectious diseases, it is found that the diseases persist. It is also found that there must be some factors that significantly contribute to the spread of diseases such as degraded environment, temperature and humidity, human-related activities like lack of sanitation (McMichael et al. (2003)).

In developing countries, the poor environmental conditions have larger impact on the spread of carrier-dependent infectious diseases. The environmental degradation (i.e., the deterioration of the environment due to depletion of natural resources such as air, water and soil) is one of the largest threats that are being looked at in the world today. It is caused due to increasing population rate, economic growth and depletion of natural resources. One of the major components of environmental degradation is unsanitary living conditions in developing countries which give rise to certain infectious diseases. Kumari and Sharma (2018) proposed a new type of epidemic model to study the impact of environmental pollution on the spread of infectious diseases.

There are many infectious diseases in which infection is transmitted by direct human-to-human contact, while there are some diseases which transmit indirectly through carriers such as flies, ticks, mites, etc., present in the environment. The bacterial diseases such as typhoid fever, leprosy, cholera, gastroenteritis, measles, dysentery, tuberculosis, diarrhea, etc., are transferred by these carriers from the environment to susceptibles leading to faster spread of such diseases in human population (Misra et al. (2012)). Some of these diseases like dysentery, gastroenteritis, cholera, typhoid fever, etc., called water borne diseases, are 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 con-

ditions in a habitat which provides a very conducive environment to these carriers to flourish. The environment conducive to the growth of carrier population further helps in the spread of infectious diseases (Ghosh et al. (2005), Ghosh et al. (2006), Naresh et al. (2008) and Naresh and Pandey (2009)).

In particular, Naresh and Pandey (2009) modeled and analyzed the cumulative effect of ecological factors in the habitat on the spread of tuberculosis (TB) in human population. They assumed that the TB not only spread by direct contacts with infectives in the population but also indirectly by bacteria which are emitted by infectives in the habitat. It is assumed further that bacteria survive due to conducive ecological factors such as flower pots, plants, grasses, human clothes, etc., in the habitat. The cumulative density of ecological factors in the habitat is assumed to be governed by a population density dependent logistic model. Their analysis shows that as parameters governing the conducive ecological factors in the habitat increase, the spread of TB increases. Also, the migration of population from environmentally degraded region to a cleaner region plays a vital role in the spread of infectious diseases as infected persons act as carrier of infection (Ghosh et al. (2005)).

Many mathematical models are proposed by assuming that the diseases are transmitted from susceptible to infected individuals via direct person to person contacts (Ma and Li (2009)). For example, Nthiiri et al. (2016) formulated a mathematical model to study the dynamics of typhoid fever disease incorporating protection against infection. Their study shows that an increase in protection leads to low disease prevalence in a population. Later on, it was discovered that that the diseases also spread indirectly due to the carriers (e.g., flies, insects or other entities) present in the environment (Toomey et al. (1947), Harry and Kent (1961), Gonzalez-Guzman (1989), Graczyk et al. (2001), Shukla et al. (2011), Lanzas and Gröhn (2011) Misra et al. (2011) and Misra et al. (2012)). In particular, Shukla et al. (2011) proposed a model with immigration for the spread of an infectious disease with bacteria and carriers in the environment. They assumed that susceptibles get infected directly by infectives as well as by their contacts with bacteria discharged by infectives in the environment. They concluded that the spread of the infectious disease increases due to growth of bacteria and carriers in the environment and disease becomes more endemic due to immigration.

A mathematical model was proposed assuming that the whole population is potentially aware of the risk but only a certain proportion chooses to respond appropriately by trying to limit their probability of becoming infectious or seeking treatment early (Kiss et al. (2010)). They conclude from their model that the infection can be eradicated if the dissemination of information is fast. The transmission of these infectious diseases further become worse due to lack of sanitation that leads to unhygienic environmental conditions in a habitat which provides a very conducive environment to these carriers to flourish. Inadequate sanitation is the primary cause of the spread of infectious diseases. It produces more feces in the habitat which travel via food, flies, etc., to us and spread various infectious diseases. About 79% of world's population lack access to safe drinking water, sanitary, etc., which cause infectious diseases. The use of unimproved sanitation, on-site sanitation and disposal of human excreta are the main cause of the spread of diarrheal diseases in India.

To control the spread of infectious diseases, several measures such as vaccination, treatment and awareness program among people are taken, but sanitation plays an important role in curtailing

the infectious disease. Thus, in our study, we have applied two sanitation efforts: one in direct proportion to carrier population, and the one in direct proportion to the environmental degradation.

2. Mathematical Model

In this section, a nonlinear mathematical model dealing with the spread of carrier dependent infectious diseases due to environmental degradation is proposed. 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 $E_m(t)$ and represents the density of environmental degradation at any time t. To control the spread of infectious diseases, a suitable sanitation effort is applied to decrease carrier population and to clean the degraded environment denoted by $F_{s1}(t)$ and $F_{s2}(t)$, respectively. In all the above variables, time t is taken in months.

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 parameter α and d represents the disease-induced death rate and the natural death rate, respectively. The density of carrier population is 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 $\frac{L(s-s_1)}{s_0}$ 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_2CF_s), where s_2 denotes the depletion rate coefficient of carrier population density due to sanitation effort applied. As the environmental degradation increases the carrier population also increases, therefore, it is assumed that the carrier population density is in direct proportion to the environmental degradation (i.e., s_3CE_m), where s_3 denotes the increase in carrier population density due to environmental degradation. The cumulative environmental degradation conducive to the growth of the carrier population is assumed to be at constant rate Q_0. The parameter θ_1 is the rate of increment of degradation of the environment due to human population-related factors, θ_2 and θ_0 is the rate coefficient of depletion of environmental degradation due to sanitation effort applied and some other factors, respectively.

The parameter ϕ is the rate of sanitation effort applied in direct proportion to the carrier population density, $\frac{\phi_s}{\phi_0}$ is the carrying capacity in the habitat, ϕ_1 is the rate of decrease of sanitation effort in proportion to carrier population density, the rate of sanitation effort applied and the rate of decrease of sanitation effort due to some other factors are ξ_1 and ξ_2, respectively. The last equation of the model system governs the logistic growth of sanitation effort with intrinsic growth rate ψ_s and carrying capacity $\frac{\psi_s}{\psi_0}$. It is also assumed that the increase in sanitation effort applied to clean the degraded environment is directly proportional to the environmental degradation (i.e.,

$\psi E_m F_s$), where ψ is the growth rate coefficient of sanitation effort due to increase in environment degradation. The decrease in sanitation effort due to its consumption in fighting against the cleaning the environment is considered in direct proportion to the environmental degradation (i.e., $\psi_1 E_m F_s$), where ψ_1 is its depletion rate coefficient. Further, ζ_1 is the rate of sanitation effort applied and ζ_2 is the rate of decrease of sanitation effort due to some other factors.

For convenience, we use $X(t) = X, Y(t) = Y, C(t) = C, E_m(t) = E_m, F_{s1}(t) = F_{s1}$ and $F_{s2}(t) = F_{s2}$. The model is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dX}{dt} &= A - \beta XY - \lambda CX - dX + \nu Y,\\
\frac{dY}{dt} &= \beta XY + \lambda CX - dY - \alpha Y - \nu Y,\\
\frac{dC}{dt} &= sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_{s1} + s_3 C E_m,\\
\frac{dE_m}{dt} &= Q_0 - \theta_0 E_m + \theta_1 (A - dN) - \theta_2 E_m F_{s2},\\
\frac{dF_{s1}}{dt} &= \phi C F_{s1} - \phi_0 F_{s1}^2 - \phi_1 C F_{s1} + \phi_s F_{s1},\\
\frac{dF_{s2}}{dt} &= \psi E_m F_{s2} - \psi_0 F_{s2}^2 - \psi_1 E_m F_{s2} + \psi_s F_{s2}.
\end{aligned} \tag{1}$$

where, $\phi_s = (\xi_1 - \xi_2) > 0$ and $\psi_s = (\zeta_1 - \zeta_2) > 0$ and $X(0) > 0$, $Y(0) \geq 0$, $C(0) > 0$, $E_m(0) > 0$, $F_{s1}(0) > 0$ and $F_{s2}(0) > 0$.

Since $N = X + Y$, the above model system (1) can be rewritten as follows,

$$\begin{aligned}
\frac{dY}{dt} &= \beta Y(N - Y) + \lambda C(N - Y) - (d + \alpha + \nu) Y,\\
\frac{dN}{dt} &= A - dN - \alpha Y,\\
\frac{dC}{dt} &= sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_{s1} + s_3 C E_m,\\
\frac{dE_m}{dt} &= Q_0 - \theta_0 E_m + \theta_1 (A - dN) - \theta_2 E_m F_{s2},\\
\frac{dF_{s1}}{dt} &= \phi C F_{s1} - \phi_0 F_{s1}^2 - \phi_1 C F_{s1} + \phi_s F_{s1},\\
\frac{dF_{s2}}{dt} &= \psi E_m F_{s2} - \psi_0 F_{s2}^2 - \psi_1 E_m F_{s2} + \psi_s F_{s2}.
\end{aligned} \tag{2}$$

2.1. Region of attraction

The region of attraction Ω for the system (2) is given by,

$\Omega = \{(Y, N, C, E_m, F_{s1}, F_{s2}) \in R_+^6, 0 \leq Y \leq \frac{A}{\alpha + d}, 0 < N \leq \frac{A}{d}, 0 \leq C \leq C_m,$
$0 \leq E_m \leq \frac{Q_0 + \theta_1 A}{\theta_0}, 0 < F_{s1} \leq F_{s1m}, 0 < F_{s2} \leq F_{s2m}\},$

which attracts all solutions starting in the positive octant, where,

C_m= $\frac{L(s-s_1)}{s_0}$, F_{s1m}= $\frac{(\phi-\phi_1)C_m+\phi_s}{\phi_0}$ and F_{s2m}= $\frac{(\psi-\psi_1)(Q_0+\theta_1 A)+\psi_s\theta_0}{\psi_0\theta_0}$.

Proof:

From system (2), we have,

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

$$\Longrightarrow \frac{dN}{dt} \leq A - dN,$$

$$\Longrightarrow \lim_{t\to\infty} \sup(N) \leq \frac{A}{d}.$$

Similarly, it can be obtained that

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

$$\Longrightarrow \frac{dN}{dt} \leq A - dN - \alpha N, \qquad (\text{since } \ Y < N)$$

$$\Longrightarrow \lim_{t\to\infty} \sup(N) \leq \frac{A}{\alpha + d}.$$

Further, it is noted that $Y < N$, and therefore, $0 < Y \leq \frac{A}{\alpha+d}$.

From the third equation of model system (2), we have,

$$\frac{dC}{dt} \leq sC - \frac{s_0 C^2}{L} - s_1 C.$$

From the theory of differential inequality (Lakshmikantham and Leela (1969)), we obtain

$$\lim_{t\to\infty} sup(C) \leq \frac{L(s-s_1)}{s_0}$$
$$= C_m \text{ (say)}.$$

This implies that $0 \leq C \leq C_m$ for large $t > 0$.

Further, from the fourth equation of model system (2), we obtain

$$\frac{dE_m}{dt} \leq Q_0 - \theta_0 E_m + \theta_1 A,$$

which implies

$$\lim_{t\to\infty} \sup(E_m) \leq \frac{Q_0 + \theta_1 A}{\theta_0}.$$

From the fifth equation of model system (2), we have

$$\frac{dF_{s1}}{dt} \leq (\phi - \phi_1) C_m F_{s1} - \phi_0 F_{s1}^2 + \phi_s F_{s1},$$

from which we get,

$$\lim_{t\to\infty} \sup(F_{s1}) \leq \frac{(\phi - \phi_1) C_m + \phi_s}{\phi_0} = (F_{s1m}) \text{ (say)}.$$

Similarly, from the sixth equation of model system (2), we have

$$\frac{dF_{s2}}{dt} \leq (\psi - \psi_1) \frac{Q_0 + \theta_1 A}{\theta_0} F_{s2} - \psi_0 F_{s2}^2 + \psi_s F_{s2},$$

which gives,

$$\lim_{t\to\infty} \sup(F_{s2}) \leq \frac{(\psi - \psi_1)(Q_0 + \theta_1 A) + \psi_s \theta_0}{\psi_0 \theta_0} = (F_{s2m}) \text{ (say)}.$$

■

3. Equilibrium Analysis

In this section, the existence of equilibrium points of the model system (2) is being investigated by equating right hand side of system (2) to zero. We obtain the following twelve non-negative equilibria:

1. $E_0(0, \frac{A}{d}, 0, \frac{Q_0}{\theta_0}, 0, 0)$. This is a 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 present in the environment, no increased sanitation effort is required to be applied. In such a case, the human population and environmental degradation will always remain at its equilibrium $\frac{A}{d}$ and $\frac{Q_0}{\theta_0}$, respectively.

2. $E_1(\overline{Y}, \overline{N}, 0, \overline{E}_m, 0, 0)$. This is a 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 $\overline{Y}$ with human population and environmental degradation maintained

at its reduced equilibrium $\overline{N}$ and $\overline{E}_m$, respectively, where, $\overline{N} = \frac{A-\alpha Y}{d}$, $\overline{E}_m = \frac{Q_0+\theta_1\alpha Y}{\theta_0}$, $\overline{Y} = \frac{\beta A-d(d+\alpha+\nu)}{\beta(\alpha+d)}$ which exists if $\beta A > d(d+\alpha+\nu)$.

3. $E_2(0, \frac{A}{d}, 0, \frac{Q_0}{\theta_0}, \frac{\phi_s}{\phi_0}, 0)$. This is also disease-free equilibrium with no carriers, the existence of which is obvious, where N=$\frac{A}{d}$, E_m=$\frac{Q_0}{\theta_0}$ and F_{s1}=$\frac{\phi_s}{\phi_0}$. It implies that since no carrier population is present in the system and disease also does not persist, the human population and environmental degradation will remain at their equilibrium. 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.

4. $E_3(0, \overline{\overline{N}}, 0, \overline{\overline{E}}_m, 0, \overline{\overline{F}}_{s2})$. As above, this is also disease-free equilibrium with no carriers, the existence of which is obvious, where $\overline{\overline{N}} = \frac{A}{d}$, $\overline{\overline{E}}_m = \frac{-(\theta_0\psi_0+\theta_2\psi_s)+\sqrt{(\theta_0\psi_0+\theta_2\psi_s)^2+4\theta_2 Q_0\psi_0(\psi-\psi_1)}}{2\theta_2(\psi-\psi_1)}$ and $\overline{\overline{F}}_{s2} = \frac{(\psi-\psi_1)E_m+\psi_s}{\psi_0}$. It implies that since no carrier population is present in the system and disease also does not persist, the human population and environmental degradation will remain at their equilibrium. In the absence of carrier population, degradation of environment remains at its equilibrium and hence the sanitation effort is neither consumed in cleaning the environment nor it increases due to degradation of environment and hence it remains at its natural level $\overline{\overline{F}}_{s2}$.

5. $E_4(\tilde{Y}, \tilde{N}, \tilde{C}, \tilde{E}_m, 0, 0)$. 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 and environmental degradation remains at their reduced equilibrium $\tilde{N}$ and $\tilde{E}_m$ with carrier population at its carrying capacity $\tilde{C}$. Its existence is discussed in Section 3.1.

6. $E_5(\tilde{\tilde{Y}}, \tilde{\tilde{N}}, 0, \tilde{\tilde{E}}_m, \tilde{\tilde{F}}_{s1}, 0)$. In this equilibrium carrier population is not present, thus, infective population, human population, environmental degradation and sanitation effort applied to control carrier population remains at their equilibrium point with $\tilde{\tilde{Y}} = \frac{\beta A-d(d+\alpha+\nu)}{\beta(\alpha+d)}$ (which exists if $\beta A > d(d+\alpha+\nu)$), $\tilde{\tilde{N}} = \frac{A-\alpha Y}{d}$, $\tilde{\tilde{E}}_m = \frac{Q_0+\theta_1\alpha Y}{\theta_0}$ and $\tilde{\tilde{F}}_{s1} = \frac{\phi_s}{\phi_0}$.

7. $E_6(\dot{Y}, \dot{N}, 0, \dot{E}_m, 0, \dot{F}_{s2})$. In this equilibrium carrier population is not present, thus, infective population, human population and environmental degradation remains at their equilibrium point with $\dot{Y} = \frac{\beta A-d(d+\alpha+\nu)}{\beta(\alpha+d)}$ which exists if $\beta A > d(d+\alpha+\nu)$, $\dot{N} = \frac{A-\alpha Y}{d}$ and $\dot{E}_m = \frac{-(\theta_0\psi_0+\theta_2\psi_s)+\sqrt{(\theta_0\psi_0+\theta_2\psi_s)^2+4\theta_2(\psi-\psi_1)(Q_0\psi_0+\theta_1\alpha\psi_0 Y)}}{2\theta_2(\psi-\psi_1)}$. Due to the absence of carrier population the degradation of environment remains at its equilibrium $\dot{E}_m$, and therefore, sanitation effort applied to control the degradation of environment also remains at its equilibrium $\dot{F}_{s2} = \frac{(\psi-\psi_1)E_m+\psi_s}{\psi_0}$.

8. $E_7(0, \ddot{N}, 0, \ddot{E}_m, \ddot{F}_{s1}, \ddot{F}_{s2})$. This equilibrium is free from infective population and carrier population. Thus, human population, environmental degradation and both sanitation effort applied are at equilibrium given by $\ddot{N} = \frac{A}{d}$, $\ddot{E}_m = \frac{-(\theta_0\psi_0+\theta_2\psi_s)+\sqrt{(\theta_0\psi_0+\theta_2\psi_s)^2+4\theta_2 Q_0\psi_0(\psi-\psi_1)}}{2\theta_2(\psi-\psi_1)}$, $\ddot{F}_{s1} = \frac{\phi_s}{\phi_0}$ and $\ddot{F}_{s2} = \frac{(\psi-\psi_1)E_m+\psi_s}{\psi_0}$.

9. $E_8(\dddot{Y}, \dddot{N}, \dddot{C}, \dddot{E}_m, \dddot{F}_{s1}, 0)$. This equilibrium is free from sanitation effort applied to control

degradation of environment. Its existence is discussed in Section 3.2.

10. $E_9(\hat{Y}, \hat{N}, \hat{C}, \hat{E}_m, 0, \hat{F}_{s2})$. This equilibrium is free from sanitation effort applied to control carrier population. Its existence is discussed in Section 3.3.

11. $E_{10}(\mathring{Y}, \mathring{N}, 0, \mathring{E}_m, \mathring{F}_{s1}, \mathring{F}_{s2})$. This equilibrium is free from carrier population, thus, infective population, human population, environmental degradation and both the sanitation efforts applied remains at their equilibrium point with $\mathring{N} = \frac{A-\alpha Y}{d}$, $\mathring{Y} = \frac{\beta A - d(d+\alpha+\nu)}{\beta(\alpha+d)}$ which exists if $\beta A > d(d+\alpha+\nu)$, $\mathring{F}_{s1} = \frac{\phi_s}{\phi_0}$, $\mathring{F}_{s2} = \frac{(\psi-\psi_1)E_m+\psi_s}{\psi_0}$ and $\mathring{E}_m = \frac{-(\theta_0\psi_0+\theta_2\psi_s)+\sqrt{(\theta_0\psi_0+\theta_2\psi_s)^2+4\theta_2(\psi-\psi_1)(Q_0\psi_0+\theta_1\alpha\psi_0 Y)}}{2\theta_2(\psi-\psi_1)}$.

12. $E_{11}(Y^*, N^*, C^*, E_m^*, F_{s1}^*, F_{s2}^*)$. This is endemic equilibrium.

3.1. Existence of $E_4(\tilde{Y}, \tilde{N}, \tilde{C}, \tilde{E}_m, 0, 0)$

The value of $\tilde{Y}$, $\tilde{N}$, $\tilde{C}$ and $\tilde{E}_m$ is obtained by solving the following set of algebraic equations,

$$(N-Y)(\beta Y + \lambda C) - (d+\alpha+\nu)Y = 0, \tag{3}$$

$$A - dN - \alpha Y = 0, \tag{4}$$

$$(s - s_1) - s_0\frac{C}{L} + s_3 E_m = 0, \tag{5}$$

$$Q_0 - \theta_0 E_m + \theta_1 \alpha Y = 0. \tag{6}$$

From Equation (4), (5) and (6) we have,

$\tilde{N} = \frac{A-\alpha Y}{d}$, $\tilde{C} = \frac{L((s-s_1)+s_3 E_m)}{s_0}$ and $\tilde{E}_m = \frac{Q_0+\theta_1 Y}{\theta_0}$.

Now, using the above values of $\tilde{N}$, $\tilde{C}$ and $\tilde{E}_m$ in Equation (3) we have,

$$a_1\tilde{Y}^2 - a_2\tilde{Y} - a_3 = 0. \tag{7}$$

where,

$a_1 = \beta(\alpha+d) + \frac{\lambda L\theta_1\alpha s_3(\alpha+d)}{\theta_0 s_0} > 0$,
$a_2 = (\beta A - d(d+\alpha+\nu)) + \frac{\lambda AL\theta_1\alpha s_3}{\theta_0 s_0} - \frac{\lambda L(\alpha+d)}{\theta_0 s_0}((s-s_1)+s_3 Q_0)$, and
$a_3 = \frac{\lambda AL}{\theta_0 s_0}((s-s_1)+s_3 Q_0) > 0$.

a_1 and a_3 are always positive. Hence, from Equation (7), $\tilde{Y}$ has at least one positive root by Descartes's Rule of Signs.

From the value of $\tilde{Y}$, we can find the value of $\tilde{N}$, $\tilde{E_m}$ and $\tilde{C}$.

3.2. Existence of $E_8(\dddot{Y}, \dddot{N}, \dddot{C}, \dddot{E}_m, \dddot{F}_{s1}, 0)$

The values of $\dddot{Y}$, $\dddot{N}$, $\dddot{C}$, $\dddot{E}_m$ and $\dddot{F}_{s1}$ are obtained by solving the following set of algebraic equations,

$$\beta Y(N-Y) + \lambda C(N-Y) - (d+\alpha+\nu)Y = 0, \tag{8}$$

$$A - dN - \alpha Y = 0, \tag{9}$$

$$(s - s_1) - \frac{s_0 C}{L} - s_2 F_{s1} + s_3 E_m = 0, \tag{10}$$

$$Q_0 - \theta_0 E_m + \theta_1 (A - dN) = 0, \tag{11}$$

$$(\phi - \phi_1)C - \phi_0 F_{s1} + \phi_s = 0. \tag{12}$$

From Equations (9), (11) and (12), respectively, we have,

$$\dddot{N} = \frac{A - \alpha Y}{d}, \tag{13}$$

$$\dddot{E}_m = \frac{Q_0 + \theta_1 \alpha Y}{\theta_0}, \tag{14}$$

$$\dddot{F}_{s1} = \frac{(\phi - \phi_1)C + \phi_s}{\phi_0}. \tag{15}$$

Now, using Equation (12) in Equation (11) we have,

$$\dddot{C} = \frac{L(\theta_0(\phi_0(s - s_1) - s_2\phi_s) + s_3\phi_0(Q_0 + \theta_1\alpha Y))}{\theta_0(\phi_0 s_0 + s_2 L(\phi - \phi_1)}. \tag{16}$$

Now, using the value of $\dddot{C}$ from Equation (16) in Equation (8) we have,

$$p_1 \dddot{Y}^2 - p_2 \dddot{Y} - p_3 = 0. \tag{17}$$

where,

$p_1 = \beta(\alpha + d) + \frac{\lambda L \theta_1 \alpha(\alpha+d)}{\theta_0(\phi_0 s_0 + s_2 L(\phi - \phi_1))} > 0,$

$p_2 = \beta A - d(d + \alpha + \nu) + \frac{\lambda A L \theta_1 \alpha}{\theta_0(\phi_0 s_0 + s_2 L(\phi - \phi_1)} - \frac{\lambda L(\alpha+d)(\theta_0(\phi_0(s-s_1) - s_2\phi_s) + s_3\phi_0 Q_0)}{\theta_0(\phi_0 s_0 + s_2 L(\phi - \phi_1))}$, and

$p_3 = \frac{\lambda A L(\theta_0(\phi_0(s-s_1) - s_2\phi_s)}{\theta_0(\phi_0 s_0 + s_2 L(\phi - \phi_1))} > 0,$

since $p_1 > 0$ and $p_3 > 0$. Thus, by using Descartes's Rule of Signs, it is found that $\dddot{Y}$ has at least one positive root. From the value of $\dddot{Y}$,s we can find the value of $\dddot{N}$, $\dddot{C}$, $\dddot{E_m}$ and $\dddot{F_{s1}}$.

3.3. Existence of $E_9(\hat{Y}, \hat{N}, \hat{C}, \hat{E}_m, 0, \hat{F}_{s2})$

The values of $\hat{Y}$, $\hat{N}$, $\hat{C}$, $\hat{E}_m$ and $\hat{F}_{s2}$ are obtained by solving the following set of algebraic equations,

$$\beta Y(N-Y) + \lambda C(N-Y) - (d+\alpha+\nu)Y = 0, \tag{18}$$

$$A - dN - \alpha Y = 0, \tag{19}$$

$$(s - s_1) - \frac{s_0 C}{L} + s_3 E_m = 0, \tag{20}$$

$$Q_0 - \theta_0 E_m + \theta_1(A - dN) - \theta_2 E_m F_{s2} = 0, \tag{21}$$

$$(\psi - \psi_1)E_m - \psi_0 F_{s2} + \psi_s = 0. \tag{22}$$

From Equations (19), (20) and (22), respectively, we have,

$$N = \frac{A - \alpha Y}{d}, \tag{23}$$

$$C = \frac{L((s - s_1) + s_3 E_m)}{s_0}, \tag{24}$$

$$F_{s2} = \frac{(\psi - \psi_1)E_m + \psi_s}{\psi_0}. \tag{25}$$

Using the value of F_{s2} from Equation (25) in Equation (21) we get,

$$\theta_2(\psi - \psi_1)E_m^2 + (\theta_0\psi_0 + \theta_2\psi_s)E_m - (Q_0\psi_0 + \theta_1\psi_0\alpha Y) = 0. \tag{26}$$

From Equation (18) we have

$$F(Y) = \beta(\alpha+d)Y^2 - (\beta A - d(d+\alpha+\nu))Y + \lambda(\alpha+d)CY - \lambda AC = 0, \tag{27}$$

$F(0) = -\lambda AC < 0$, and

$F(\frac{A}{(\alpha+d)}) = \frac{Ad(d+\alpha+\nu)}{\alpha+d} > 0$,

$F'(Y) = \beta(\alpha+d)Y + \frac{\lambda AC}{Y} - \lambda(A - (\alpha+d)Y)\frac{dC}{dY}$,

since, $\frac{dC}{dY} = \frac{Ls_3\theta_1\alpha\psi_0}{s_0(2\theta_2(\psi-\psi_1)E_m + (\theta_0\psi_0+\theta_2\psi_s))} > 0$.

Thus, $F'(Y) > 0$ if $(\beta(\alpha+d)Y + \frac{\lambda AC}{Y}) > (\lambda(A - (\alpha+d)Y)\frac{dC}{dY})$.

Thus, we can find one positive root of $\hat{Y}$, and hence, the values of $\hat{N}$, $\hat{C}$, $\hat{E}_m$ and $\hat{F}_{s2}$ can be obtained.

3.4. Existence of $E_{11}(Y^*, N^*, C^*, E_m^*, F_{s1}^*, F_{s2}^*)$

We prove the existence of equilibrium by setting right hand side of equations in the model (2) to zero and by solving the resulting algebraic equations, as given below,

$$\beta Y(N-Y) + \lambda C(N-Y) - (d+\alpha+\nu)Y = 0, \tag{28}$$

$$A - dN - \alpha Y = 0, \tag{29}$$

$$sC - \frac{s_0 C^2}{L} - s_1 C - s_2 C F_{s1} + s_3 C E_m = 0, \tag{30}$$

$$Q_0 - \theta_0 E_m + \theta_1(A - dN) - \theta_2 E_m F_{s2} = 0, \tag{31}$$

$$\phi C F_{s1} - \phi_0 F_{s1}^2 - \phi_1 C F_{s1} + \phi_s F_{s1} = 0, \tag{32}$$

$$\psi E_m F_{s2} - \psi_0 F_{s2}^2 - \psi_1 E_m F_{s2} + \psi_s F_{s2} = 0. \tag{33}$$

From Equations (29), (32) and (33) we have,

$$N = \frac{A - \alpha Y}{d}, \tag{34}$$

$$F_{s1} = \frac{(\phi - \phi_1)C + \phi_s}{\phi_0}, \tag{35}$$

$$F_{s2} = \frac{(\psi - \psi_1)E_m + \psi_s}{\psi_0}. \tag{36}$$

Using Equation (35) in Equation (30)

$$C = \frac{L(\phi_0(s - s_1) - s_2\phi_s) + s_3\phi_0 E_m}{s_0\phi_0 + s_2 L(\phi - \phi_1)}. \tag{37}$$

Using Equation (36) in Equation (31) we have,

$$\theta_2(\psi - \psi_1)E_m^2 + (\theta_0\psi_0 + \theta_2\psi_s)E_m - (Q_0\psi_0 + \theta_1\psi_0(A - dN)) = 0. \tag{38}$$

From Equation (28) we have

$$F(Y) = \beta(\alpha+d)Y^2 - (\beta A - d(d+\alpha+\nu))Y + \lambda(\alpha+d)CY - \lambda AC = 0, \tag{39}$$

$F(0) = -\lambda AC < 0$, and

$F(\frac{A}{(\alpha+d)}) = \frac{Ad(d+\alpha+\nu)}{\alpha+d} > 0$,

$F'(Y) = \beta(\alpha+d)Y + \frac{\lambda AC}{Y} - \lambda(A - (\alpha+d)Y)\frac{dC}{dY}$,

since, $\frac{dC}{dY} = \frac{Ls_3\phi_0\theta_1\alpha\psi_0}{(s_0\phi_0+s_2L(\phi-\phi_1))(2\theta_2(\psi-\psi_1)E_m+(\theta_0\psi_0+\theta_2\psi_s))} > 0$.

Thus, $F'(Y) > 0$ if $(\beta(\alpha+d)Y + \frac{\lambda AC}{Y}) > (\lambda(A-(\alpha+d)Y)\frac{dC}{dY})$.

Thus, we can find one positive root of Y^*, and hence, the values of N^*, C^*, E_m^*,F_{s1}^* and F_{s2}^* can be obtained.

4. Stability Analysis

The stability behavior of equilibrium points $E_0 - E_{11}$ is presented here. The stability behavior of $E_i, (i = 0-10)$ is analyzed by Jacobian matrix method and of E_{11} is analyzed by Lyapunov method.

Theorem 4.1.

The equilibria $E_i, (i = 0-10)$ are unstable and the endemic equilibrium E_{11} is locally asymptotically stable provided the following condition is satisfied,

$$s_2 L\lambda^2(N^*-Y^*)^2 < \frac{2}{3}s_0(\phi-\phi_1)F_{s1}^*\left(\beta Y^* + \frac{\lambda C^* N^*}{Y^*}\right), \tag{40}$$

$$s_3^2\theta_2 L(\phi-\phi_1)E_m^* F_{s1}^* < \frac{4}{9}s_0 s_2(\psi-\psi_1)(\theta_0+\theta_2 F_{s2}^*)F_{s2}^*, \tag{41}$$

$$\alpha\theta_1^2 d(\psi-\psi_1)F_{s2}^* < \frac{2}{3}\theta_2 E_m^*(\beta Y^* + \lambda C^*)(\theta_0+\theta_2 F_{s2}^*). \tag{42}$$

Proof:

The general variational matrix M of the model (2) is as follows,

$$M = \begin{bmatrix} \overline{A} & \beta Y+\lambda C & \lambda(N-Y) & 0 & 0 & 0 \\ -\alpha & -d & 0 & 0 & 0 & 0 \\ 0 & 0 & \overline{B} & 0 & -s_2 C & 0 \\ 0 & -\theta_1 d & 0 & -\theta_0-\theta_2 F_s & 0 & -\theta_2 E_m \\ 0 & 0 & (\phi-\phi_1)F_{s1} & 0 & (\phi-\phi_1)C-2\phi_0 F_{s1}+\phi_s & 0 \\ 0 & 0 & 0 & (\psi-\psi_1)F_{s2} & 0 & \overline{C} \end{bmatrix},$$

where, $\overline{A} = \beta N - 2\beta Y - \lambda C - (d+\alpha+\nu)$, $\overline{B} = (s-s_1) - \frac{2s_0 C}{L} - s_2 F_{s1} + s_3 E_m$ and $\overline{C} = (\psi-\psi_1)E_m - 2\psi_0 F_{s2} + \psi_s$.

The variational matrix $M_i, (i = 0-10)$ of model (2) corresponding to equilibria $E_i, (i = 0-10)$ are found having positive roots, and therefore, the equilibria $E_i, (i = 0-10)$ are unstable.

A positive-definite function is considered to establish the local stability of endemic equilibrium E_{11},

$$U_1 = \frac{1}{2}(m_0 y^2 + m_1 n^2 + m_2 c^2 + m_3 e_m^2 + m_4 f_{s1}^2 + m_5 f_{s2}^2),$$

where m_i $(i = 0, 1, 2, 3, 4, 5)$ are positive constants to be chosen appropriately and y, n, c, e_m, f_{s1}, f_{s2} are small perturbations about E_{11}, defined as follows:

$$Y = Y^* + y, N = N^* + n, C = C^* + c, E_m = E_m^* + e_m, F_{s1} = F_{s1}^* + f_{s1}, F_{s2} = F_{s2}^* + f_{s2}.$$

Differentiating the above equation with respect to "t" and using linearized system of model system (2) around E_{11}, we get,

$$\begin{aligned}\frac{dU_1}{dt} = &-m_0\left(\beta Y^* + \frac{\lambda C^* N^*}{Y^*}\right)y^2 - m_1 dn^2 - m_2\frac{s_0 C^*}{L}c^2 - m_3(\theta_0 + \theta_2 F_{s2}^*)e_m^2 \\ &- m_4\phi_0 F_{s1}^* f_{s1}^2 - m_5\psi_0 F_{s2}^* f_{s2}^2 + [m_0(\beta Y^* + \lambda C^*) - m_1\alpha]ny + m_0\lambda(N^* - Y^*)cy \\ &+ m_2 s_3 C^* e_m c + [-m_2 s_2 C^* + m_4(\phi - \phi_1)F_{s1}^*]cf_{s1} - m_3\theta_1 dne_m \\ &+ [-m_3\theta_2 E_m^* + m_5(\psi - \psi_1)F_{s2}^*]e_m f_{s2}.\end{aligned}$$

On choosing m_0 = 1, m_1= $\dfrac{\beta Y^* + \lambda C^*}{\alpha}$, m_2=$\frac{(\phi-\phi_1)F_{s1}^*}{s_2 C^*}$, m_3= $\frac{(\psi-\psi_1)F_{s2}^*}{\theta_2 E_m^*}$, $m_4 = 1$ and $m_5 = 1$, we get $\dfrac{dU_1}{dt}$ to be negative definite showing that U_1 is a Lyapunov's function, and hence, E_{11} is locally asymptotically stable provided the conditions (40), (41) and (42) are satisfied. ■

Theorem 4.2.

The endemic equilibrium E_{11} is nonlinearly asymptotically stable in the region Ω provided the following conditions are satisfied:

$$4\alpha\left(\beta + \frac{\lambda C_m}{Y^*}\right) < \beta d, \tag{43}$$

$$s_2(\phi - \phi_1) < \frac{4}{3}\frac{s_0\phi_0}{L}, \tag{44}$$

$$\theta_2\left(\frac{Q_0 + \theta_1 A}{\theta_0}\right)(\psi - \psi_1) < \frac{1}{3}\psi_0(\theta_0 + \theta_2 F_{s2}^*), \tag{45}$$

$$\alpha\theta_0\theta_1^2 d(\psi - \psi_1) < \frac{2}{3}\theta_2\left(\beta + \frac{\lambda C_m}{Y^*}\right)(\theta_0 + \theta_2 F_{s2}^*)(Q_0 + \theta_1 A). \tag{46}$$

Proof:

Consider the following positive-definite function to establish the nonlinear stability of endemic equilibrium E_{11},

$$\begin{aligned}U_2 = &\, k_0\left(Y - Y^* - Y^*\ln\frac{Y}{Y^*}\right) + \frac{k_1}{2}(N - N^*)^2 + k_2\left(C - C^* - C^*\ln\frac{C}{C^*}\right) \\ &+ \frac{k_3}{2}(E_m - E_m)^2 + k_4\left(F_{s1} - F_{s1}^* - F_{s1}^*\ln\frac{F_{s1}}{F_{s1}^*}\right) + k_5\left(F_{s2} - F_{s2}^* - F_{s2}^*\ln\frac{F_{s2}}{F_{s2}^*}\right),\end{aligned}$$

where k_i $(i = 0, 1, 2, 3, 4, 5)$ are positive constants to be chosen appropriately and y, n, c, e_m, f_{s1}, f_{s2} are small perturbations about E_{11}, defined as follows:

$$Y = Y^* + y, N = N^* + n, C = C^* + c, E_m = E_m^* + e_m, F_{s1} = F_{s1}^* + f_{s1}, F_{s2} = F_{s2}^* + f_{s2}.$$

Differentiating the above equation with respect to "t" and using linearized system of model system (2) around E_{11}, we get,

$$\begin{aligned}\frac{dU_2}{dt} = &-k_0\left(\beta + \frac{\lambda CN}{YY^*}\right)(Y - Y^*)^2 - k_1 d(N - N^*)^2 - k_2\frac{s_0}{L}(C - C^*)^2 \\ &- k_3(\theta_0 + \theta_2 F_{s2}^*)(E_m - E_m^*)^2 - k_4\phi_0(F_{s1} - F_{s1}^*)^2 - k_5\psi_0(F_{s2} - F_{s2}^*)^2 \\ &+ (k_0\left(\beta + \frac{\lambda C}{Y^*}\right) - k_1\alpha)(Y - Y^*)(N - N^*) + k_0\lambda\left(\frac{N^* - Y^*}{Y^*}\right)(Y - Y^*)(C - C^*) \\ &+ (-k_2 s_2 + k_4(\phi - \phi_1))(C - C^*)(F_{s1} - F_{s1}^*) + k_2 s_3(C - C^*)(E_m - E_m^*) \\ &- k_3\theta_1 d(N - N^*)(E_m - E_m^*) + (-k_3\theta_2 E_m + k_5(\psi - \psi_1))(E_m - E_m^*)(F_{s2} - F_{s2}^*).\end{aligned}$$

On choosing k_0 = 1, k_1= $\frac{1}{\alpha}(\beta + \frac{\lambda C_m}{Y^*})$, k_2=$\frac{(\phi-\phi_1)}{s_2}$, k_3= $\frac{(\psi-\psi_1)\theta_0}{\theta_2(Q_0+\theta_1 A)}$, $k_4 = 1$ and $k_5 = 1$, we get $\frac{dU_2}{dt}$ to be negative definite showing that U_2 is a Lyapunov's function and hence E^* is locally asymptotically stable provided the conditions (43), (44), (45) and (46) are satisfied. ∎

5. Numerical Simulation

We give here the numerical solution of the mathematical model system (2) to show the existence of equilibrium values and to check the feasibility of stability conditions.

For this, we integrate the system (2) by fourth-order Runge-Kutta method using MATLAB with the following set of parameter values:

A = 120, β = 0.0005, λ = 0.00002, ν = 0.2, d = 0.15, α = 0.03, s=0.4, s_0=0.9, L=500, s_1=0.2, s_2=0.0004, s_3=0.0002, Q_0=20, θ_1 = 0.02, θ_0 = 0.1, θ_2 = 0.002, ϕ = 0.6, ϕ_0 = 0.3, ϕ_1 = 0.005, ξ_1=0.4, ξ_2=0.004, ψ = 0.5, ψ_0 = 0.26, ψ_1 = 0.004, ζ_1=0.3, ζ_2=0.003.

The equilibrium values of endemic equilibrium are computed as,

Y^* = 64.490, N^* = 787.102, C^* = 83.419, E_m = 84.307, F_{s1}=166.768, F_{s2}=161.975.

The eigenvalues corresponding to the variational matrix of endemic equilibrium are: -41.7862, -0.7515, -0.0645, -0.1381, -0.2166, -49.9640.

Since all the eigenvalues are found to be negative, therefore, for the above set of parameter values the endemic equilibrium is locally asymptotically stable. The results of the model are displayed graphically in Figures 1 through 12. From Figure 1, we see that the endemic equilibrium E^* is non-linearly asymptotically stable. This shows that for the given set of parameter values the curves of

total human population (N), infective population (Y) and carrier population density (C) approach to the equilibrium points regardless of the initial values of N, Y and C.

The initial starts of all trajectories to reach the equilibrium point are given below:

(1) $Y(0)$= 100 $N(0)$= 450 $C(0)$= 200 $E_m(0)$= 248 $F_{s1}(0)$= 250 $F_{s2}(0)$= 200,
(2) $Y(0)$= 150 $N(0)$= 500 $C(0)$= 250 $E_m(0)$= 248 $F_{s1}(0)$= 250 $F_{s2}(0)$= 200,
(3) $Y(0)$= 200 $N(0)$= 550 $C(0)$= 300 $E_m(0)$= 248 $F_{s1}(0)$= 250 $F_{s2}(0)$= 200,
(4) $Y(0)$= 250 $N(0)$= 600 $C(0)$= 350 $E_m(0)$= 248 $F_{s1}(0)$= 250 $F_{s2}(0)$= 200.

Figure 2 shows the variation of the infective population with time t for distinct values of λ. It is seen from the figure that with an increase in the value of λ, the infective population increases. Thus, to control this increment in the indirect transmission of infectious disease due to the carrier population density present in the conducive degraded environment, some suitable sanitation strategies should be applied to clean our environment.

The decline of carrier population density present in the degraded environment and infective population as a result of suitable sanitation effort applied is shown in Figure 3 and Figure 4, respectively, with time t for distinct values of s_2 (rate of depletion of carrier population due to sanitation effort applied). It is seen in the figure that with an increase in the sanitation effort carrier population decreases (Figure 3) which consequently decreases the infective population (Figure 4).

The decline of carrier population density present in the degraded environment and infective population as a result of suitable sanitation effort applied is shown in Figure 7 and Figure 8, respectively, with time t for distinct values of θ_2 (rate of depletion of carrier population due to sanitation effort applied). It is seen in the figure that with an increase in the sanitation effort carrier population decreases (Figure 7) which consequently decreases the infective population (Figure 8).

The effect of sanitation effort on infective population and carrier population with time t is shown in Figure 9 and 10, respectively, for different value of ϕ, the rate of sanitation effort applied in direct proportion to the carrier population density. It is observed that with increase in the rate of sanitation effort, the carrier population decreases (Figure 9). This decrease in the infective population is due to decline in the population of carrier in the environment as a result of increasing the sanitation effort (Figure 10). It is, therefore, speculated that the prevalence of infectious disease can be controlled by minimizing the carrier population in the environment if sanitation effort is suitably applied.

The effect of sanitation effort is shown in Figure 11 and 12 on carrier population density and infective population with time t, respectively, for different values of ψ, the rate of sanitation effort applied in direct proportion to environmental degradation. It is found that by applying the suitable sanitation effort, the degradation of the environment will decrease. This will make our environment clean, and hence, the carriers which carry infected bacteria and virus from the degraded environment will decrease (Figure 11). This ultimately decreases the infective population (Figure 12). Thus, to control the spread of infectious diseases due to carrier present in the conducive degraded environment proper sanitation effort should be applied.

REFERENCES

Brauer, F. and Castillo-Chavez, C. (2012). *Mathematical Models in Population Biology and Epidemiology*, Springer, Second Edition.

Dubey, B., Patra, A., Srivastava, P.K. and Dubey, U.S. (2013). Modeling and analysis of an $SEIR$ model with different types of nonlinear treatment rates, J. Biol. Syst., Vol. 21. doi:10.1142/S021833901350023X

Ghosh, M., Chandra, P., Sinha, P. and Shukla, J. (2005). Modelling the spread of bacterial disease: Effect of service providers from an environmentally degraded region, Applied Mathematics and Computation, Vol. 60, pp. 615–647.

Ghosh, M., Chandra, P., Sinha, P. and Shukla, J. (2006). Modelling the spread of bacterial infectious disease with environmental effect in a logistically growing human population, Nonlinear Analysis: Real World Applications, Vol. 7, pp. 341–363.

Gonzalez-Guzman, J. (1989). An epidemiological model for direct and indirect transmission of typhoid fever, Math. Biosci., Vol. 96, pp. 33–46.

Graczyk, T.K., Knight, R., Gilman, R.H. and Cranfield, M.R. (2001). The role of non-biting flies in the epidemiology of human infectious diseases, Microbes Infect., Vol. 3, pp. 231–235.

Harry, D.P. and Kent, S.L. (1961). Ticks of public health importance and their control, US Department of Health, Education and Welfare, Communicable Disease Center, Atlanta, Georgia.

Hsu, S. and Zee, A. (2004). Global spread of infectious diseases, Journal of Biological Systems, Vol. 12, pp. 289–300.

Keeling, M.J. and Rohani, P. (2008). *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press, New Jersey.

Kiss, I.Z., Cassell, J., Recker, M. and Simon, P.L. (2010). The impact of information transmission on epidemic outbreaks, Mathematical Biosciences, Vol. 225, pp. 1–10.

Kumari, N. and Sharma, S. (2018). Modeling the dynamics of infection disease under the influence of environmental pollution, International Journal of Applied and Computational Mathematics, Vol. 4, pp. 1–24.

Lakshmikantham, V. and Leela S. (1969). *Differential and Integral Inequalities: Theory and Applications*, Academic Press, New York, USA.

Lanzas, C., Lu, Z. and Gröhn, Y. T. (2011). Mathematical modeling of the transmission and control of foodborne pathogens and antimicrobial resistance at preharvest, Foodborne Pathog. Dis., Vol. 8, pp. 1–10.

Ma, Z. and Li, J. (2009). *Dynamical Modeling and Analysis of Epidemics*, World Scientific. doi:10.1142/6799

McMichael, A.J., Campbell-Lendrum, D.H. et al. (2003). Climate Change and Human Health-Risk and Responses, WHO.

Misra, A.K., Mishra, S.N., Pathak, A.L., Misra, P. and Naresh, R. (2012). Modeling the effect of time delay in controlling the carrier dependent infectious disease-Cholera, App. Math. Comp., Vol. 218, pp. 11547–11557.

Misra, A.K., Singh, V. and Shukla, J.B. (2011). Modeling the spread of an infectious disease with

bacteria and carriers in the environment, Nonlinear Analysis: Real World Applications, Vol. 12, pp. 2541–2551.

Naresh, R. and Pandey, S. (2009). Modeling the cumulative effect of ecological factors in the habitat on the spread of tuberculosis, International Journal of Biomathematics, Vol. 2, pp. 339–355.

Naresh, R., Pandey, S. and Misra, A. (2008). Analysis of vaccination model for carrier dependent infectious disease with environmental effects, Nonlinear Analysis: Modelling and Control, Vol. 13, pp. 331–350.

Nthiiri, J.K. et al. (2016). Mathematical modelling of typhoid fever disease incorporating protection against infection, British Journal of Mathematics & Computer Science, Vol. 14, pp. 1-10.

Shukla, J.B., Singh, V. and Misra, A. (2011). Modeling the spread of an infectious disease with bacteria and carriers in the environment, Nonlinear Analysis: Real World Applications, Vol. 12, pp. 2541–2551.

Toomey, J.A. et al. (1947). Can Drosophila flies carry Poliomyelitis virus?, J. of Infect. Dis., Vol. 81, pp. 135–138.

Appendix: Figures

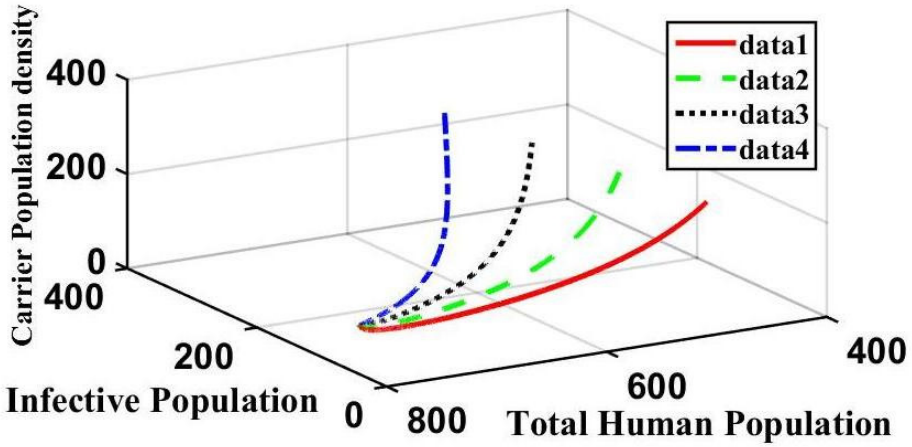

Figure 1. Variation of total human population with infective population and carrier population density

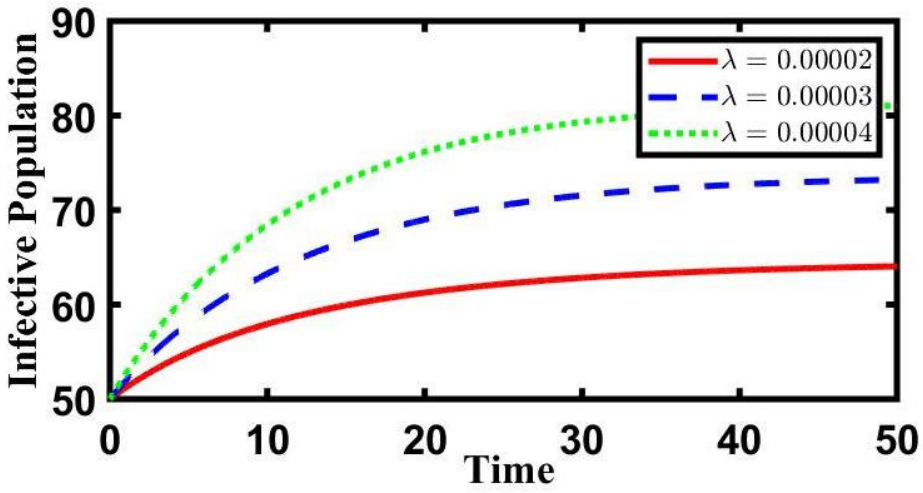

Figure 2. Variation of infective population with time for distinct values of λ

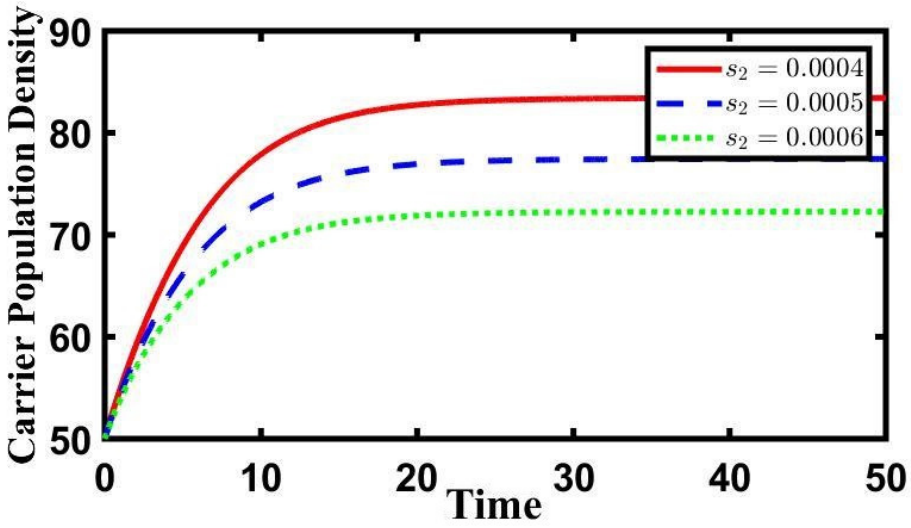

Figure 3. Variation of carrier population density with time for distinct values of s_2

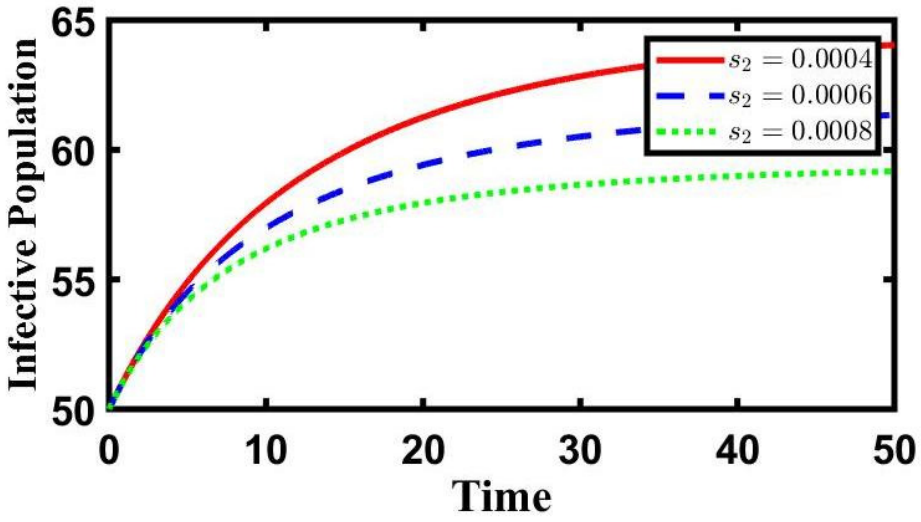

Figure 4. Variation of infective population with time for distinct values of s_2

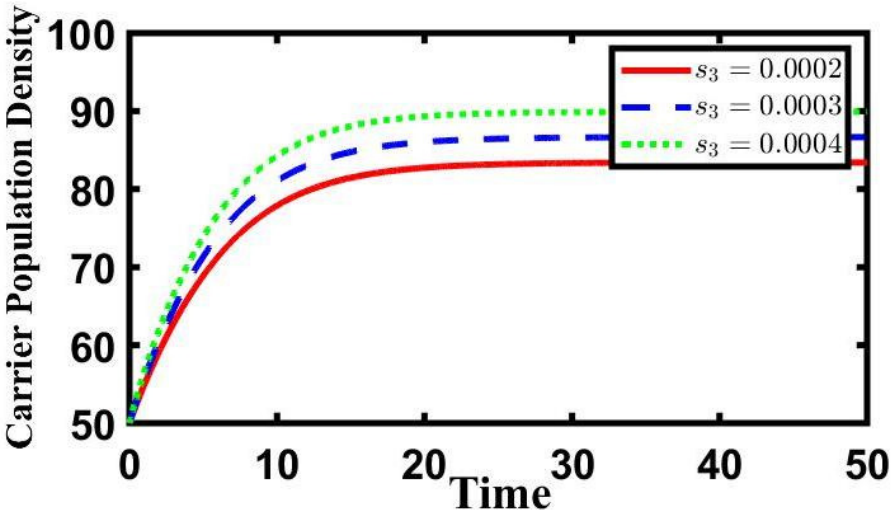

Figure 5. Variation of carrier population density with time for distinct values of s_3

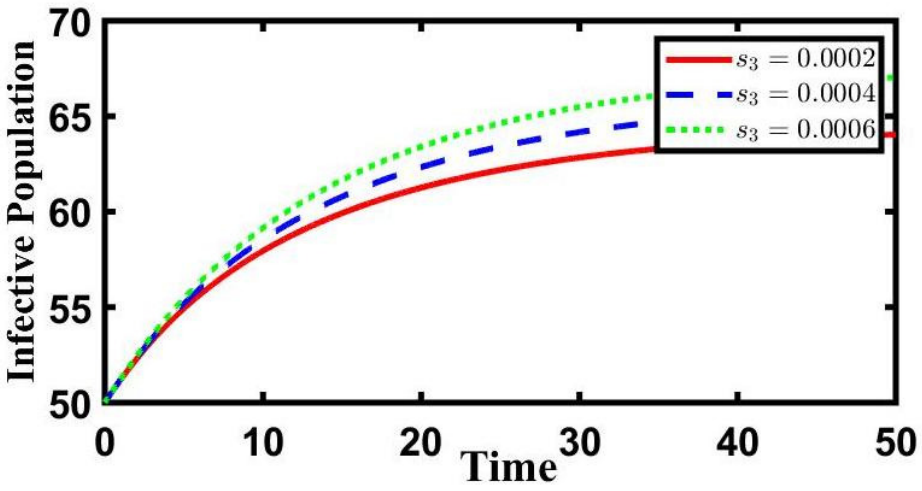

Figure 6. Variation of infective population with time for distinct values of s_3

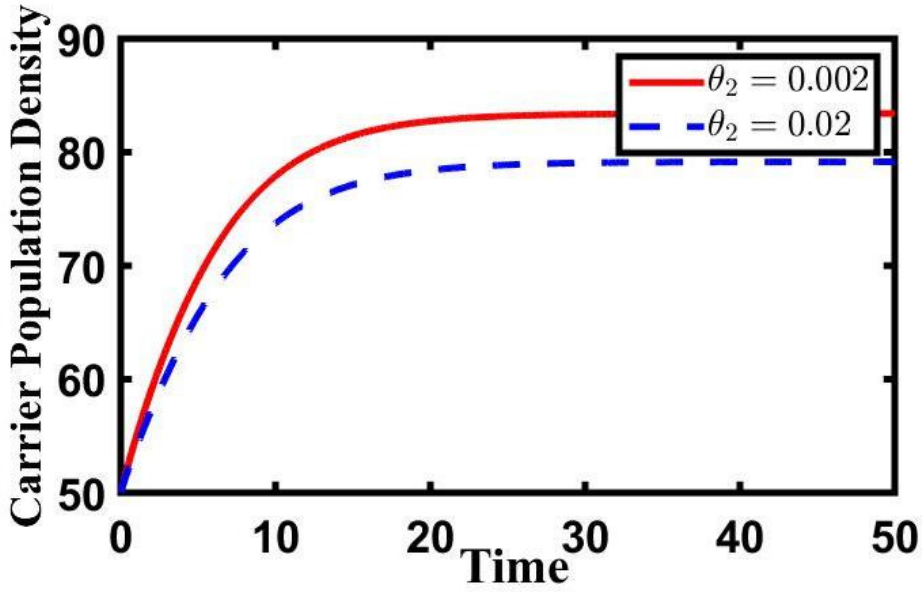

Figure 7. Variation of carrier population density with time for distinct values of θ_2

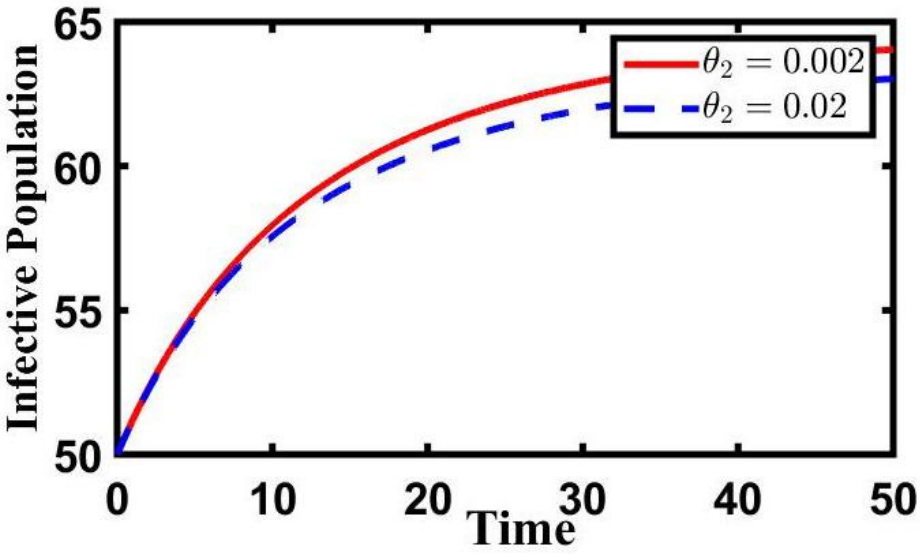

Figure 8. Variation of infective population with time for distinct values of θ_2

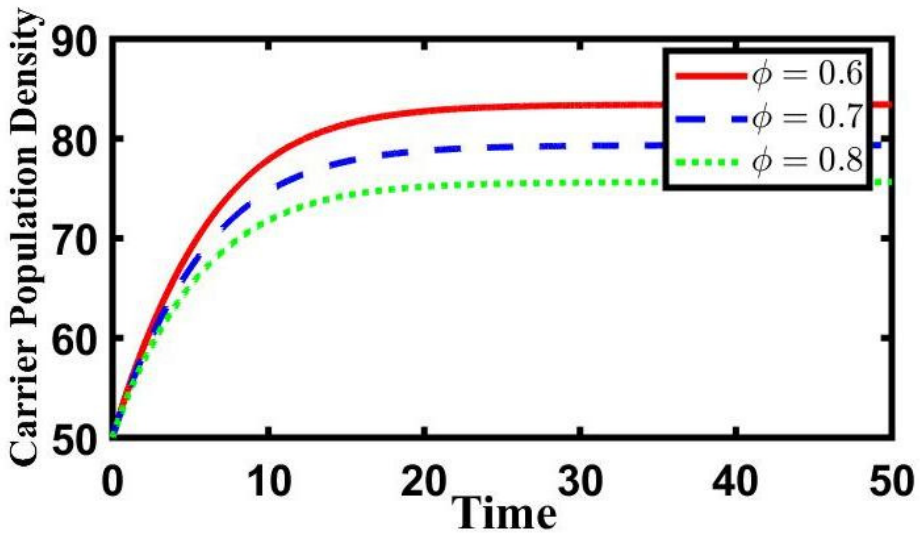

Figure 9. Variation of carrier population density with time for distinct values of ϕ

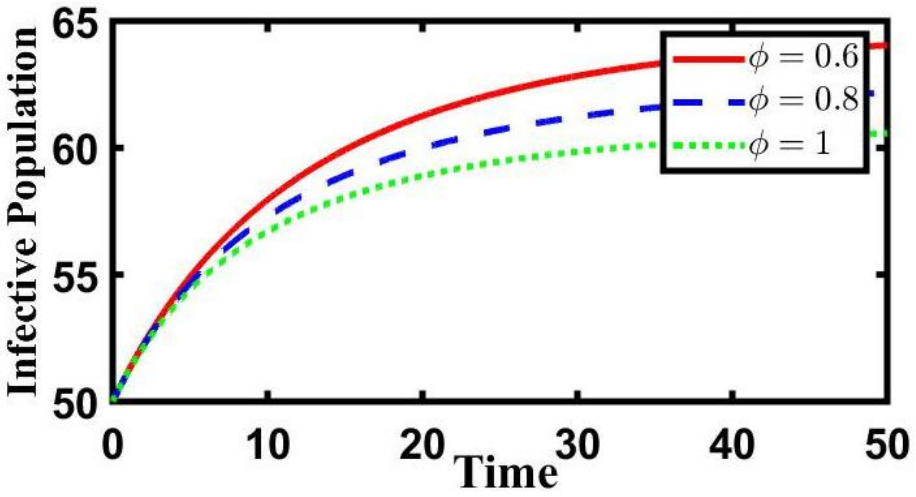

Figure 10. Variation of infective population with time for distinct values of ϕ

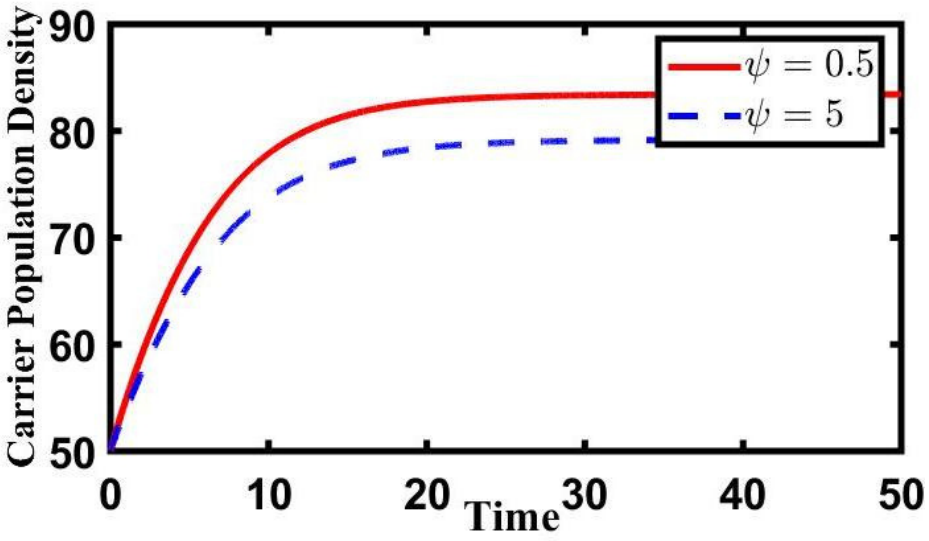

Figure 11. Variation of carrier population density with time for distinct values of ψ

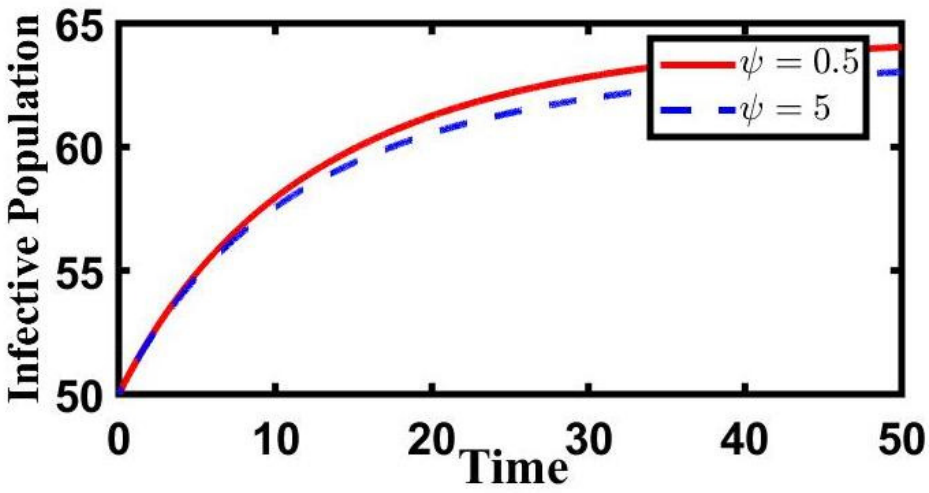

Figure 12. Variation of infective population with time for distinct values of ψ