Global warming is one of the most crippling issues affecting humankind. This unequivocal phenomenon has kept most of the scientific community engaged for more than 100 years. The driving force behind global warming is the emission of greenhouse gases. The most prominent heat trapping gas is CO$_2$ [18], whose concentration in the atmosphere is rapidly increasing which is evident from the existing data [12]. From the mid-18$^{th}$ century to 2018, the concentration of CO$_2$ has increased from 280 ppm to 406 ppm approximately. If this emission rate continues, the concentration of CO$_2$ is estimated to reach a dangerous level of 550 ppm by 2050 [17].

In accordance with the increase in the emission of CO$_2$ in the atmosphere, the global average temperature has shown an elevation of 0.74$^\circ$C during the 20$^{th}$ century. The rate of increase of global average temperature has been alarmingly high since 1970’s, at around 0.2$^\circ$C growth each decade [16]. There is almost a linearly increasing trend of the planet’s average temperature with respect to CO$_2$ concentration in the atmosphere [12, 15]. There is a broad scientific consensus that anthropogenic activities are leading to the warming of the world’s atmosphere at an unprecedented rate. Unless controlled, the global average temperature is predicted to show a rise of 3$^\circ$C to 4$^\circ$C by the end of 2100 as compared to the 1986 – 2005 average temperature [17].

The CO$_2$ concentration increases in the atmosphere due to natural as well as anthropogenic sources. The natural sources consist of volcanic eruptions, decay of plant and animal matter, the aerobic respiration, etc. The concentration of CO$_2$ emitted by these sources is reduced from the atmosphere through plantation of trees and by a number of oceanic processes such as the biological pumps etc. Further, since the beginning of the industrial age, human activities such as, burning of fossil fuels for power, burning of forests, industrialization, etc., have also produced an undesirable increase in the global average temperature [20].

Spread of carrier dependent infectious diseases is one of the direst impacts of global warming. The fourth report of IPCC gives strong evidence of this detrimental impact of global warming on human health [9]. The increase in the global temperature and conducive climate change causes an increase in the population of carriers such as, flies, cockroaches, mites, ticks, etc. For instance, house flies breed prolifically in warmer temperatures but they become
indolent as the mercury drops. The temperature also affects the rate at which the flies mature. These carriers transport infective agents excreted by infectives to the food and water of susceptible and thus play an important role in the increased spread of infectious diseases indirectly [1, 10, 13]. There is a general perception that these diseases mostly affect the underdeveloped areas located in equatorial zones, especially those regions which lack in sanitation. But even in developed countries of Europe, a variety of vector-borne diseases have surfaced, including West Nile fever, Leishmaniasis, Lyme disease and tick-borne encephalitis [4]. As the temperature rises, the invasive species responsible for the spread of these diseases such as ticks, sand flies and mosquitoes find favourable environment in UK [4]. It is rather intimidating that during 2030 to 2050, global warming is expected to cause around 250,000 more cases of casualties per year from carrier dependent infectious diseases [7,8].

Various investigators [2, 3, 6, 11, 19, 23, 24] have used mathematical modelling to study the spread of infectious diseases. It may be noted that very little attention has been paid to analyse the influence of globally rising temperature on the transmission of infectious diseases. Singh [25] had shed light on the effect of global warming on the proliferation of carrier dependent infectious diseases. Here, we bring to notice that, the role of immigration of carriers caused by temperature rise on the spread of infectious diseases in human habitat is very important but has not been studied so far in any of these literatures. To address this issue appropriately, it is crucial to comprehend the role of anthropogenic \( CO_2 \) and the corresponding temperature rise behind the increase of carrier population leading to the spread of infectious diseases. In this paper, therefore, we suggest and analyse a nonlinear SIS model using stability and basic differential sensitivity analysis to study the effect of global warming on the spread of carrier dependent infectious diseases caused by the immigration of carriers to the human habitat.

2. Mathematical Model

Let us consider a human habitat where the carrier population immigrates due to conducive temperature rise which is suitable for their growth and survival. The disease is assumed to spread directly by the infectives as well as indirectly by the carriers, transporting infective agents to the food and water of susceptible. The total population density \( N(t) \) at any time \( t \) is divided into two classes, susceptible class \( X(t) \) and infective class \( Y(t) \). It is assumed that the carrier population density \( C_r(t) \) grows logistically with given intrinsic growth rate \( s_0 \) and carrying capacity \( L \). Its growth rate also increases due to increase of global average temperature \( T(t) \) at the rate \( s_1 \). It is assumed that \( C(t) \), the concentration of \( CO_2 \) increases at constant emission rate \( Q_0 \) from natural sources and with a constant rate \( \delta_1 \) from anthropogenic sources, which increase with the increase in the human population [21]. Natural sinks uptake \( CO_2 \) at a rate \( \delta_0 C \) proportional to the atmospheric concentration of \( CO_2 \) [22]. It is observed that there is almost a linear correlation of the increase in global average temperature with the increased \( CO_2 \) concentration in the atmosphere (see Figure 2.1) [12, 15]. The variable \( T(t) \) represents the global average temperature at time \( t \) which is assumed to be proportional to the increased concentration of carbon dioxide \( C(t) \) in the atmosphere [12, 15]. In the model, the constant \( T_0 \) is the preindustrial global average temperature i.e. average temperature in the absence of anthropogenic \( CO_2 \) emissions [26] and \( C_0 = Q_0 / \delta_0 \) is the concentration of carbon dioxide in the absence of anthropogenic emissions [25].
With these considerations, the following nonlinear mathematical model is proposed to study the effect of global warming on the growth of carrier population and its impact on the spread of infectious diseases.

\[
\frac{dX}{dt} = A - \beta XY - \lambda XC_r - dX + \nu Y, \\
\frac{dY}{dt} = \beta XY + \lambda XC_r - (d + \alpha + \nu)Y, \\
\frac{dC_r}{dt} = s_0 C_r - \frac{s_0 C_r^2}{L} - d_1 C_r + s_1 (T - T_0), \\
\frac{dC}{dt} = Q_0 - \delta_0 C + \delta_1 (A - dN), \\
\frac{dT}{dt} = \theta (C - C_0) - \theta_0 (T - T_0),
\]

\[X(0) > 0, Y(0) \geq 0, N(0) > 0, C_r(0) \geq 0, C(0) > C_0, T(0) > T_0.\]

The different parameters present in this model are described as follows-

- \(A\) - the rate of immigration of human population
- \(d\) - natural death rate of human population
- \(\beta\) - transmission rate due to infectives
- \(\lambda\) - transmission rate due to carrier population
- \(\alpha\) - death rate of infected population due to diseases
- \(\nu\) - the rate of recovery of infective human population - \(s_0\) - intrinsic growth rate of carrier population
- \(s_1\) - the rate of growth of carrier population due to global warming
- \(d_1\) - the rate of depletion of carrier population by using control mechanism
- \(Q_0\) - the rate of emission of carbon dioxide from natural sources
- \(\delta_0\) - the rate of depletion of carbon dioxide due to natural sinks
- \(\delta_1\) - the rate of emission of carbon dioxide due to anthropogenic sources
- \(\theta\) - the rate of growth of temperature due to rise in carbon dioxide concentration
- \(\theta_0\) - the rate of natural depletion of coefficient of temperature

It should be noted that all these parameters are positive constants, except \(\lambda\), which is non-negative constant. In the third equation of model (2.1), we assume \(s_0 - d_1 = s > 0\).

Since, the total population density \(N = X + Y\), the model (2.1) can be reduced to the following equivalent form.

\[
\frac{dY}{dt} = \beta (N - Y)Y + \lambda (N - Y)C_r - (d + \alpha + \nu)Y, \\
\frac{dN}{dt} = A - dN - \alpha Y,
\]
Lemma 3.1. For equilibrium analysis of the model (2.2) we need the following lemma to establish the bounds of variables [14].

3. Equilibrium Analysis

Case 2. $E_{\text{back}}$ in equations (3.3) - (3.6) gives us the unique non trivial equilibrium $E^*$. Following equilibrium equations-

\[
\begin{align*}
   \frac{dC_r}{dt} &= sC_r - \frac{s_0 C_r^2}{L} + s_1 (T - T_0), \\
   \frac{dC}{dt} &= Q_0 - \delta_0 C + \delta_1 (A - dN), \\
   \frac{dT}{dt} &= \theta (C - C_0) - \theta_0 (T - T_0), \\
   X(0) &> 0, Y(0) \geq 0, N(0) > 0, C(0) \geq 0, C(0) > C_0, T(0) > T_0.
\end{align*}
\]

\[\sqrt{\lambda} = \sqrt{\frac{\beta \lambda}{d}} > 1.\]

Theorem 3.1. There are following two equilibria of the model (2.2) -

(i) $E_0 \equiv \{0, \frac{J}{L}, 0, C_0, T_0\}$ the trivial infective free, carrier free equilibrium.

(ii) $E^* = (Y^*, N^*, C^*, T^*)$ the unique non trivial equilibrium, which exists in a subset of $\Omega$ given by $\Omega_s = \{(Y, N, C, T) \in \mathbb{R}^4_+ : 0 < N < \frac{\lambda}{(s + \alpha d)}, N \geq \frac{\lambda}{(s + \alpha d)}, 0 \leq C \leq C_m, C_0 \geq C \leq C_m, T_0 \leq T \leq T_m\}$, provided the basic reproduction number $R_0 = \frac{\beta \lambda}{d(a + d + \alpha)} > 1$.

Proof. The existence of $E_0$ is obvious. Now the existence of $E^* = (Y^*, N^*, C^*, T^*)$ is established by solving the following equilibrium equations-

\[
\begin{align*}
   \beta (N - Y) Y + \lambda (N - Y) C - (d + \alpha + v) Y &= 0, \\
   A - dN - aY &= 0, \\
   sC_r - \frac{s_0 C_r^2}{L} + s_1 (T - T_0) &= 0, \\
   Q_0 - \delta_0 C + \delta_1 (A - dN) &= 0, \\
   \theta (C - C_0) - \theta_0 (T - T_0) &= 0.
\end{align*}
\]

Now we consider the following two cases

Case 1. $\lambda > 0$

Using equations (3.3) - (3.6) in equation (3.2) the following expression in $Y$ is obtained

\[
F(Y) = \frac{A - (\alpha + d)Y}{d} \beta Y + \lambda \frac{A - (\alpha + d)Y}{d} \frac{sL}{2s_0} \left(1 + \sqrt{1 + \frac{4s_0 s_1 \delta_1 \beta \lambda Y}{L \theta_0 \delta_0 \theta_0 s^2}}\right) = 0.
\]

Since

\[
F(0) = \frac{\lambda s A L}{2d s_0} > 0 \text{ and } F\left(\frac{A}{\alpha + d}\right) = -(\alpha + d + v)\left(\frac{A}{\alpha + d}\right) < 0.
\]

Therefore, ensures that there exists at least one root of $F(Y)$ in $\left(0, \frac{\lambda \alpha}{\alpha + d}\right)$. For uniqueness of root, we find $F'(Y)$ from equation (3.7) as follows

\[
F'(Y) = -\frac{\beta Y}{d} - \frac{\lambda s L}{2d Y s_0} (A - (\alpha + d)Y)
\]

\[
- \frac{\lambda (\alpha + d)}{d} \frac{sL}{2s_0} \left(1 + \sqrt{1 + \frac{4s_0 s_1 \delta_1 \beta \lambda Y}{L \theta_0 \delta_0 \theta_0 s^2}}\right) - \frac{\lambda}{d} (A - (\alpha + d)Y) \left(\frac{s^2 + 4s_0 s_1 \delta_1 \beta \lambda Y}{2\theta_0 \delta_0 s_0 s \sqrt{1 + \frac{4s_0 s_1 \delta_1 \beta \lambda Y}{L \theta_0 \delta_0 s^2}}\right).
\]

This shows that $F'(Y) < 0$. It proves that there is a unique root $Y^*$ of $F(Y)$ in $\left(0, \frac{\lambda \alpha}{\alpha + d}\right)$. This value $Y^*$ substituted back in equations (3.3) - (3.6) gives us the unique non trivial equilibrium $E^* = (Y^*, N^*, C^*, T^*)$ of model (2.2).

Case 2. $\lambda = 0$

Using equation (3.3) in equation (3.2), we get the following function of $Y$, $G(Y) = \beta \frac{\lambda}{(\alpha + d)} Y - (\alpha + d + v) Y = 0$. This gives us two values of $Y, Y = 0$ (trivial equilibrium $E_0$) and

\[
Y^* = \frac{\beta \lambda}{d(a + d + \alpha)} > 0 \text{ provided the basic reproduction number } R_0 = \frac{\beta \lambda}{d(a + d + \alpha)} > 1.
\]
Theorem 3.2. Now we shall show that $\frac{dY}{dT}|_{E^*}, \frac{dC}{dT}|_{E^*}, \frac{dC}{dT}|_{E^*}$ all are positive.

Proof. Using equations (3.3) in (3.5), and noting that $C_0 = \frac{Q_0}{\alpha_0}$ we get $C - C_0 = \frac{\beta_0 Y}{\delta_0}$. Hence $\frac{dY}{dT} = \frac{\delta_0}{\delta_0} > 0$

Differentiating equation (3.4) with respect to $T$ and simplifying, we get $rac{dC}{dT} = \frac{\delta_0}{\delta_0} > 0$.

Then using equations (3.3), (3.5) and (3.6) in (3.4), we get a relation between $C$, and $Y$, differentiating which we get $rac{dY}{dT} = \left(\frac{\alpha T}{\theta T} + \frac{\theta}{\delta T} C\right) \frac{\theta_0 y_0}{\alpha_0 y_0} > 0$ thus $\frac{dY}{dT} > 0$.

This shows that the spread of disease increases due to global average temperature as the number of infectives increases.

4. Stability Analysis

We study the stability criteria of equilibria $E_0$ and $E^*$. The local stability of equilibrium $E_0$ is investigated by obtaining the sign of eigen values of Jacobian matrix of linearized model system at $E_0$. The Jacobian matrix at $E_0$ is

$$M = \begin{pmatrix}
\beta \frac{d}{d} - (d + \alpha + \nu) & 0 & \frac{dA}{d} & 0 \\
-\alpha & -d & 0 & 0 \\
0 & 0 & s & 0 & s_1 \\
0 & -\delta_1 d & 0 & -s_0 & 0 \\
0 & 0 & 0 & \theta & -\theta_0
\end{pmatrix}$$

The characteristic polynomial of $M$ is

$$C(x) = -\left(\beta \frac{d}{d} - (d + \alpha + \nu) - x\right) (d + x)(s - x)(\delta_0 + x)(\theta_0 + x) + \alpha \lambda \theta \delta s_1 A,$$ (4.1)

where $s = s_0 - d_1 > 0$.

Since $C(s) = \alpha \lambda \theta \delta s_1 A > 0$ and $\lim_{x \to \infty} C(x) = -\infty$. Hence, there exists a positive eigen value of $M$. Thus, $E_0$ is unstable.

Using Lyapunov’s direct method to study the stability phenomena of $E^*$, we get the following results.

Theorem 4.1. The equilibrium $E^*$ is locally asymptotically stable if the following inequalities are satisfied:

$$\alpha \lambda^2 C_{r}^2 < \beta^2 Y^2,$$ (4.2)

$$\alpha \lambda^2 (N^* - Y^*)^2 L^2 \delta_0^2 \theta^2 \delta_1^2 d < \delta_0^2 \theta_0^2 \delta_0^2 C_{r}^2 \beta^2 Y^2.$$ (4.3)

For proof, see Appendix A.

Theorem 4.2. The global asymptotic stability of the equilibrium $E^*$ is established in $\Omega_\epsilon$ if the following two inequalities hold:

$$\alpha \lambda^2 C_{r m}^2 < \beta^2 Y^2,$$ (4.4)

$$\alpha \lambda^2 (N^* - Y^*)^2 L^2 \delta_0^2 \theta^2 \delta_1^2 d < \delta_0^2 \theta_0^2 \delta_0^2 C_{r}^2 \beta^2 Y^2.$$ (4.5)

For proof, see Appendix B.

5. Numerical Simulation

Now, we verify the analytical results numerically and plot solutions of model system (2.2) along with sensitivity functions of state variables through numerical simulation by assigning suitable values to parameters. Furthermore, while choosing the values of parameters, we try to be as rational as possible. From Singh [25], we choose the following values:

$$\beta = 5.1 \times 10^{-7}, \lambda = 6.1 \times 10^{-8}, \nu = 0.012, \alpha = 0.0005, d = 0.0004, s_0 = 0.9, s = 0.3,$$

$$L = 100000, T_0 = 13.5, Q_0 = 5, \delta_0 = 0.016, \theta = 0.001, \theta_0 = 0.25, C_0 = 312.50.$$

The remaining parameters are assumed to have values $A = 12, s_1 = 400, \delta_1 = 0.7$. With the above set of values of parameters, we get the non-trivial equilibrium as

$$C^* = 453.501, C_{r}^* = 34069.097, N^* = 21942.009, T^* = 14.064, Y^* = 6445.753.$$

The eigenvalues of the Jacobian matrix for the model (2.2) at $E_0$ and $E^*$ are obtained as

$$\{-0.2499999227, 0.002373232587, -0.00003684173444, -0.01600487427, 0.300000090\}$$

and

$$\{-0.00067229346, -0.01009814023, -0.01599158955, -0.2500000037, -0.3132438038\}$$

respectively. Since two eigenvalues are positive at $E_0$ and all eigenvalues are negative at $E^*$, hence, the trivial infective free, carrier free equilibrium $E_0$ is unstable while the nontrivial equilibrium $E^*$ is locally asymptotically stable. The
global stability conditions obtained in Theorems 4.1 and 4.2 are also satisfied by the above-mentioned set of parameter values.

In Figure 5.1, the solution trajectories are plotted in $Y - N - C_r$ space for four different initial conditions to show the convergence of different trajectories towards the equilibrium $E^*$ ($Y^*, N^*, C_r^*$). This clearly demonstrates the system’s global stability at $E^*$. Figures 5.2 and 5.3 are plotted to demonstrate the effects of the transmission rate due to infectives ($\beta$) and transmission rate due to carrier population ($\lambda$) on infected population and total population densities. Both $\beta$ and $\lambda$, when increased, cause increase in the infective population and decrease in total population densities. In Figure 5.4, it is shown that as the rate of growth of carrier population due to global warming ($s_1$) increases, the infected population and carrier population density increase. Figures 5.5, 5.6 and 5.7 demonstrate the effects of the rate of emission of carbon dioxide from natural sources ($Q_0$), the rate of emission of carbon dioxide due to anthropogenic sources ($\delta_1$) and the rate of growth of temperature due to rise in carbon dioxide concentration ($\theta$) on the dependent variables $Y, C_r, C, T$. It is evident that all these variables increase with increase in $Q_0, \delta_1$ and $\theta$.

![Figure 5.1](image1.png)

**Figure 5.1:** The global stability of $E^*$ in $Y - N - C_r$ space.

![Figure 5.2](image2.png)

**Figure 5.2:** Variation of infected population density and total population density with respect to time for different values of rate of transmission of disease due to infectives $\beta$. 
Figure 5.3: Variation of infected population density and total population density with respect to time for different values of rate of transmission of disease due to carriers $\lambda$.

Figure 5.4: Variation of infected population density and carrier population density with respect to time for different values of the rate of growth of carrier population due to global warming $s_1$. 
Figure 5.5: Variation of infected population density, carrier population density, concentration of CO$_2$ and average surface temperature with respect to time for different values of the rate of emission of CO$_2$ due to natural sources $Q_0$.

Figure 5.6: Variation of infected population density, carrier population density, concentration of CO$_2$ and average surface temperature with respect to time for different values of the rate of emission of CO$_2$ due to anthropogenic sources $\delta_1$. 
Figure 5.7: Variation of infected population density, total population density, carrier population density and average surface temperature with respect to time for different values of the rate of growth of temperature due to rise in carbon dioxide concentration $\theta$.

6. Sensitivity Analysis
For the parameters $\lambda, \beta, s, \theta, \delta_1$ and $\delta_0$, the basic differential sensitivity analysis of model system (2.2) is conducted following Bortz and Nelson [5], to better understand the impact of changes in these parameters on the model system behaviour. The sensitivity systems with respect to the parameters $\lambda, \beta, s, \theta, \delta_1$ and $\delta_0$ are given by the following sets of equations (6.1) - (6.6) respectively.

\[
\dot{Y}_A(t, \lambda) = \beta (N_A(t, \lambda) - Y_A(t, \lambda)) Y(t, \lambda) + \beta(N(t, \lambda) - Y(t, \lambda)) Y_A(t, \lambda) \\
+ \lambda (N_A(t, \lambda) - Y_A(t, \lambda)) C_I(t, \lambda) + \lambda(N(t, \lambda) - Y(t, \lambda)) C_I(t, \lambda) \\
+ \lambda(N(t, \lambda) - Y(t, \lambda) - C_I(t, \lambda)) - (d + \alpha + v) Y_A(t, \lambda) \\
\dot{N}_A(t, \lambda) = -d N_A(t, \lambda) - \alpha Y_A(t, \lambda), \\
\dot{C}_r(t, \lambda) = s C_r(t, \lambda) - 2s \frac{C_r(t, \lambda)}{L} C_r(t, \lambda) + s_1 T_c(t, \lambda) \\
\dot{C}_A(t, \lambda) = -\delta_0 C_A(t, \lambda) + \delta_1 (-d N_A(t, \lambda)) \\
\dot{T}_A(t, \lambda) = \theta C_A(t, \lambda) - \theta_0 T_A(t, \lambda) \\
\dot{Y}_B(t, \beta) = \beta \left(N_B(t, \beta) - Y_B(t, \beta)\right) Y(t, \beta) + \beta(N(t, \beta) - Y(t, \beta)) Y_B(t, \beta) \\
+ \lambda(N(t, \beta) - Y(t, \beta)) Y(t, \beta) + \lambda \left(N_B(t, \beta) - Y_B(t, \beta)\right) C_I(t, \beta) \\
+ \lambda(N(t, \beta) - Y(t, \beta)) C_I(t, \beta) - (d + \alpha + v) Y_B(t, \beta) \\
\dot{N}_B(t, \beta) = -d N_B(t, \beta) - \alpha Y_B(t, \beta).
\]  
(6.1)

\[
\dot{C}_r(t, \beta) = s C_r(t, \beta) - 2s \frac{C_r(t, \beta)}{L} C_r(t, \beta) + s_1 T_c(t, \beta) \\
\dot{C}_B(t, \beta) = -\delta_0 C_B(t, \beta) + \delta_1 (-d N_B(t, \beta)) \\
\dot{T}_B(t, \beta) = \theta C_B(t, \beta) - \theta_0 T_B(t, \beta)
\]  
(6.2)
\[-(d + \alpha + \nu)Y_{S1}(t, s_1)\]
\[\dot{N}_{S1}(t, s_1) = -dN_{S1}(t, s_1) - \alpha Y_{S1}(t, s_1).\]

\[\dot{C}_{r_1}(t, s_1) = sc_{r_1}(t, s_1) - \frac{2s_0C_r(t, s_1)}{L}C_{r_1}(t, s_1) + s_1T_{r_1}(t, s_1) + T(t, s_1) - T_0\]
\[C_{r_1}(t, s_1) = -\delta_0C_{r_1}(t, s_1) + \delta_1(-dN_{S1}(t, s_1))\]
\[T_{r_1}(t, s_1) = \theta C_{r_1}(t, s_1) - \theta_0T_{r_1}(t, s_1).\]

(6.3)

\[Y_{\theta}(t, \theta) = \beta(N_{\theta}(t, \theta) - Y_{\theta}(t, \theta))Y(t, \theta) + \beta(N(t, \theta) - Y(t, \theta))Y_{\theta}(t, \theta)\]
\[+ \lambda(N_{\theta}(t, \theta) - Y_{\theta}(t, \theta))C_r(t, \theta) + \lambda(N(t, \theta) - Y(t, \theta))C_{r_1}(t, \theta)\]
\[-(d + \alpha + \nu)Y_{\theta}(t, \theta)\]
\[N_{\theta}(t, \theta) = -dN_{\theta}(t, \theta) - \alpha Y_{\theta}(t, \theta).\]

(6.4)

\[\dot{C}_{r_1}(t, \delta_1) = sc_{r_1}(t, \delta_1) - \frac{2s_0C_r(t, \delta_1)}{L}C_{r_1}(t, \delta_1) + s_1T_{r_1}(t, \delta_1)\]
\[C_{r_1}(t, \delta_1) = -\delta_0C_{r_1}(t, \delta_1) + \delta_1(-dN_{\theta}(t, \delta_1)) + A - dN(t, \delta_1)\]
\[T_{r_1}(t, \delta_1) = \theta C_{r_1}(t, \delta_1) - \theta_0T_{r_1}(t, \delta_1).\]

(6.5)

\[Y_{\delta_0}(t, \delta_0) = \beta(N_{\delta_0}(t, \delta_0) - Y_{\delta_0}(t, \delta_0))Y(t, \delta_0) + \beta(N(t, \delta_0) - Y(t, \delta_0))Y_{\delta_0}(t, \delta_0)\]
\[+ \lambda(N_{\delta_0}(t, \delta_0) - Y_{\delta_0}(t, \delta_0))C_r(t, \delta_0)\]
\[+ \lambda(N(t, \delta_0) - Y(t, \delta_0))C_{r_1}(t, \delta_0) - (d + \alpha + \nu)Y_{\delta_0}(t, \delta_0)\]
\[N_{\delta_0}(t, \delta_0) = -dN_{\delta_0}(t, \delta_0) - \alpha Y_{\delta_0}(t, \delta_0).\]

(6.6)

Here, $Z_w(t, w)$ represents the sensitivity function of $Z$ with respect to the corresponding parameter $w$. In Figures 9 and 10, we have plotted semi-relative sensitivity solutions to show the impact of doubling of parameters $\lambda, \beta, s_1, \theta, \delta_1$ and $\delta_0$ on variables of the model system (2.2) [5]. From Figure 6.1, it is evident that between the two parameters $\lambda$ and $\beta$, the parameter $\beta$ has significant influence over the infected population and total population densities. In fifty years, the doubling of transmission rate due to infectives ($\beta$) causes more cases than the other parameter. On the other hand, in Figure 6.2, we see that among the parameters $s_1, \theta, \delta_1$ and $\delta_0$, the rate of growth of carrier population due to global warming ($s_1$) causes the maximum increase in the infected population and carrier population density, while the rate of depletion of carbon dioxide ($\delta_0$) can play a significant role in controlling the spread of disease.
7. Conclusions

Many infectious diseases including cholera, diarrhoea, dysentery, measles, gastroenteritis are spread by carriers such as flies, mites, ticks, cockroaches, etc. We have proposed and studied a nonlinear mathematical model for the spread of carrier dependent infectious diseases. In the proposed model, the carrier dependent infectious diseases are considered
to spread by the direct contact of susceptible and infective as well as by the indirect effect of increased carrier population, transporting infective agents.

A qualitative study of the proposed model is performed. The model is found to have two non-negative equilibria, a boundary equilibrium and an interior equilibrium. It is found that the boundary equilibrium is always unstable. Local and global stability conditions for the interior equilibrium have been obtained using Lyapunov’s direct method. The stability analysis of the non-trivial equilibrium shows that the growth rate of carrier population due to rise in average temperature has a destabilizing effect on the system. These results are confirmed by using numerical simulation and plotting various graphs.

Sensitivity analysis is conducted to show the comparative effect of doubling the key parameters on the dynamics of the model-system. The rate of transmission of diseases due to direct contacts of infectives and susceptible, immigration rate of carrier population due to global warming, the rate at which levels of atmospheric CO2 increase due to anthropogenic activities and the rate of depletion of carbon dioxide are found to be critical. It is observed that besides reducing CO2 emissions caused by anthropogenic activities, taking measures such as carbon capture, plantation, etc., to enhance CO2 sinks may also be helpful in controlling the spread of carrier dependent infectious diseases.

Appendices

Appendix A.

Proof of Theorem 4.1. To prove the theorem, we linearize the model system (2.2) using the transformation-

\[ Y = Y^* + y_1, N = N^* + n_1, C_r = C^*_r + c_{r1}, C = C^* + c_1, T = T^* + \zeta, \]

and choose the following positive definite function,

\[ U = \frac{k_0}{2} y_1^2 + \frac{k_1}{2} n_1^2 + \frac{k_2}{2} c_{r1}^2 + \frac{k_3}{2} c_1^2 + \frac{k_4}{2} S^2, \]

where \( k_0, k_1, k_2, k_3, k_4 \) are positive constants to be chosen appropriately later. Now differentiating \( U \) with respect to \( t \) and using linearised form of model system (2.2) and simplifying we get,

\[ \frac{dU}{dt} = -\left( \frac{k_0\beta Y^*}{2} y_1^2 - k_0\lambda C^*_r y_1 n_1 + \frac{k_1 d}{2} n_1^2 \right) - \left( \frac{k_0\beta Y^*}{2} y_1^2 - k_0\lambda (N^* - Y^*) y_1 c_{r1} + \frac{k_2 S c^*_r}{2L} c_{r1} \right) \\
- \left( \frac{k_1 d}{2} n_1^2 + (k_3 \delta d) n_1 c_1 + \frac{k_3 \delta_0}{2} c^2_1 \right) - \left( \frac{k_1 d}{2} n_1^2 - k_4 \theta c_1 + \frac{k_4 \theta_0}{2} \right) \\
- \left( \frac{k_4 \theta_0}{2} - k_2 s_1 c_{r1} \tau + \frac{k_2 s_0 c^*_r}{2L} c_{r1} \right) + (k_0 \beta Y^* - k_1 \lambda) y_1 n_1 \\
- \frac{k_2 \lambda (N^* - Y^*) C^*_r}{Y^*} y_1^2 - \frac{k_0 \lambda C^*_r y_1^2}{C^*_r} - \frac{k s_1 (T^* - T_0)}{C^*_r} c_{r1}^2. \]

Taking \( k_0 = \frac{\mu}{\beta Y^*} \) and \( k_1 = 1, \frac{dU}{dt} \) is negative definite if the following conditions hold-

\[ \alpha \lambda C^*_r y_1^2 < \beta^2 Y^2 d, \]

\[ k_2 > \alpha L \frac{\lambda^2 (N^* - Y^*)^2}{\beta^2 Y^2 S_0 C^*_r}, \]

\[ k_3 < \frac{\delta_0}{\delta^2 d}, \]

\[ k_4 < \frac{k_3 \delta_0 \theta_0}{\theta \tau}, \]

\[ k_2 < \frac{k_2 \delta_0 \theta_0 S_0 C^*_r}{L S_1^2}. \]

Combining the inequalities (A.5), (A.6), (A.7) and (A.8), we get

\[ \alpha \lambda^2 (N^* - Y^*)^2 L^2 \delta^2 \theta^2 \tau^2 d < \delta_0^2 \delta^2 \tau^2 C^*_r \beta^2 Y^2. \]

(A.4) and (A.9) are the required conditions for stability as stated in the Theorem 4.1 [see inequalities (4.2) and (4.3)].
Appendix B.

Proof of Theorem 4.2. To study global stability of \( E^* \) we consider the following positive definite function:

\[
V = m_0 \left( Y - Y^* \right) + \frac{m_1}{2} \left( N - N^* \right)^2 + m_2 \left( C_r - C_r^* \right) + \ln \left( \frac{C_r}{C_r^*} \right) + \frac{m_3}{2} \left( C - C^* \right)^2 + \frac{m_4}{2} (T - T^*). \tag{B.1}
\]

Differentiating with respect to \( t \) and using model system (2) and simplifying we get,

\[
\frac{dV}{dt} = -\left( \frac{m_0 \delta_0}{2} (C - C^*)^2 + m_3 \delta_1 (C - C^*)(N - N^*) + \frac{m_1}{2} \left( N - N^* \right)^2 \right)
- \left( \frac{m_3 \delta_0}{2} (C - C^*)^2 + m_3 \delta_1 d (C - C^*)(N - N^*) + \frac{m_1 d}{2} \left( N - N^* \right)^2 \right)
- \left( m_4 \delta_0 (T - T^*) - \frac{m_1 \omega}{2d} (T - T^*) \right)
- \left( \frac{m_0 \omega N C_r}{YY^*} (Y - Y^*)^2 + (m_1 \alpha - m_1 \alpha Y^* - N^*) (Y - Y^*) + \frac{m_2 \delta_0}{2L} (C_r - C_r^*)^2 \right)
- \frac{m_3 \delta_1}{C_r C_r^*} (T - T^*). \tag{B.2}
\]

Taking \( m_0 = \frac{\alpha}{\beta} \) and \( m_1 = 1 \) the conditions for \( \frac{dV}{dt} \) to be negative definite are

\[
\alpha \lambda^2 C_r^2 < \beta Y^* d, \tag{B.3}
\]

\[
m_2 > \alpha L \frac{\lambda^2 (N^* - Y^*)^2}{\beta Y^*^2 s_0}, \tag{B.4}
\]

\[
m_3 < \frac{\delta_0}{\delta_1 d}, \alpha \lambda^2 C_r^2 < \beta Y^* d, \tag{B.5}
\]

\[
m_4 < \frac{m_3 \delta_0 \delta_1}{\delta_1 d}, \alpha \lambda^2 C_r^2 < \beta Y^* d, \tag{B.6}
\]

\[
m_5 < \frac{m_3 \delta_0 \delta_1 s_0}{\lambda^2 s_1^2}, \alpha \lambda^2 C_r^2 < \beta Y^* d. \tag{B.7}
\]

Combining the inequalities (B.4), (B.5), (B.6) and (B.7), we get

\[
\alpha \lambda^2 (N^* - Y^*)^2 \frac{L^2 \delta_1^2 \theta^2 \lambda^2 d}{\delta_0 \delta_0^2 \lambda^2 C_r^2} \beta^2 Y^* \alpha \lambda^2 C_r^2 \beta^2 Y^* d < \delta_0 \delta_0^2 \lambda^2 C_r^2 \beta^2 Y^* \alpha \lambda^2 C_r^2 \beta^2 Y^* d. \tag{B.8}
\]

Also, on taking upper bound for \( C_r \) in equation (B.3), we get

\[
\alpha \lambda^2 C_r^2 < \beta^2 Y^* d \alpha \lambda^2 C_r^2 < \beta^2 Y^* d, \tag{B.9}
\]

where \( C_r = \frac{\alpha L}{2s_0} \left[ 1 + \sqrt{1 + \frac{4m_3 \delta_0 \delta_1 s_0}{\lambda^2 L s_1^2}} \right] \).

Thus (B.8) and (B.9) are the required inequalities for stability conditions as stated in Theorem 4.2 [see inequalities (4.4) and (4.5)]

References


