Article

Modeling the effect of global warming on the spread of vector borne infectious diseases

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Received 20 May 2023; *Accepted 28 May 2023*; *Published online 5 June 2023*; *Published 1 December 2023*

Abstract

Climate change, the primarily human-caused rise in the average temperature of the Earth's climate system, is referred to as global warming. In this paper a non-linar mathematical model is proposed and analysed to study the effect of global warming global warming temperature on the transmission dynamics of vector borne diseases. In modeling the process, the total host population is divided into subclasses of susceptible host and infective host; and the vector population is divided into subclasses of susceptible vector and infective vector. The model is analysed using the stability theory. The analysis of model shows that as the temperature due to global warming increases, the vector population as well as the infective host population is increased. The rise in Infective host population results in the fast spread of vector borne diseases.

Keywords host-vector transmission; global warming; carbon dioxide; numerical simulation.

Computational Ecology and Software
ISSN 2220-721X
URL: http://www.iaees.org/publications/journals/ces/online-version.asp
RSS: http://www.iaees.org/publications/journals/ces/rss.xml
E-mail: ces@iaees.org
Editor-in-Chief: WenJun Zhang
Publisher: International Academy of Ecology and Environmental Sciences

1 Introduction

Global warming is one of the most difficult challenges for experts to study in this century. The World Health Organization considers global warming to be one of the most serious hazards to human health. The term "global warming" refers to the influence of human activities on the climate, particularly the use of fossil resources (coal, oil, and gas) and broad range deforestation, which produce significant amounts of "greenhouse gases" into the atmosphere, the most important of which is carbon dioxide. The average global temperature that has changed in recent times has been exceptional throughout the centuries (Mann et al., 1999; Stocker, 2014) and research (Field, 2014) suggest that the reason is growing anthropogenic greenhouse gas emissions. Vector-borne diseases are spread by the pathogens carried by arthropods. The primary vectors are mosquitoes and ticks. Global warming has an indirect impact on vector-borne infectious diseases. The geographical distribution and activity of vectors are affected by global warming. Arthropod vectors are to accountable for the spread of some of the most deadly infectious diseases affecting both people and animals. More than three

billion people now live in endemic regions and are susceptible to vector-borne diseases and parasites. These infections kill millions of people each year and have far-reaching implications owing to the intrinsic morbidity they induce, leading to incalculable protracted illnesses and life-long risks (Oliveira et al., 2016; Lorenz et al., 2017). The rise in temperature due to global warming will not only impact the growth of arthropod vector population, but also the spread of disease by reducing the latent (incubation) period of pathogens (Chan and Johansson, 2012). The increased frequency and severity of natural disasters caused by global warming should also be included in the epidemiology of arthropod vector-borne diseases. As catastrophes and tropical storms become more frequent and intense, vectors can move to different places, creating an abundance of fresh breeding grounds for their growth (Caillouet et al., 2008; Patricola and Wehner, 2018). The escalation of urbanisation and rise in temperature are global trends that are likely not possible to alter in the coming years. consequently, some extra regions may become comprehensively appropriate for vector breading and amplification, boosting the chance of disease transmission carried by arthropod vectors (Aguiar et al., 2018).

Despite the fact that warmer temperature conditions are more favourable to growth of vectors, their intimate interaction with human beings, along with their ability to survive in urbanized environments, is even more significant factor than climate alone (Rochlin et al., 2013). There are several instances of increased disease transmission by vectors. Dengue fever, the most common arbovirus in the world, is an outstanding example (Benelli and Mehlhornxl, 2016; Akter et al., 2018). According to estimates, 390 million individuals globally are infected with the dengue disease every year and this number is likely to rise in the future (Bhatt et al., 2013; Wunderlich et al., 2018). Moreover, reports of epidemics in previously unaffected regions are becoming frequently regular, such as the Zika virus outbreak in the America, which had almost 700,000 cases reported in 2016 (Faria et al., 2017) and over 150 cases were recorded in the first severe mosquito-borne disease epidemic in a temperate region (Poletti et al., 2011). In addition, the frequency of yellow fever in Brazil increased significantly, with over 1500 reported cases and 400 deaths (Rodhain, 2022) , as did the prevalence of West Nile virus and Lyme disease in North America (Rosenberg et al., 2018).

Various researches have investigated mathematical modelling and analysis of infectious diseases (May and Anderson, 1979; Hsu and Zee, 2004; Ma et al., 2009; Siettos and Russo, 2013; Su and Wang, 2015; Agaba et al., 2017; Banerjee, 2017; Nasution et al., 2020; Verma et al., 2020; Zhang et al., 2020; Naresh et al., 2021; Tyagi et al., 2021). Conser (2015) investigated the role of movement of host on the vector borne diseases and suggested that processes of economic advancement and proliferation make mobility more significant in the future. A vector-host system in which the host population is organised into communities that engage with non-mobile vectors that live in different environments is analysed by using Lagrangian perspective (Bichara and Chavez, 2016). The regional and seasonal spread of vector borne infectious disease is influenced by environmental changes. The seasonal change in disease distribution to determine the effect of climate on the contact incidence of disease is studied by Dangbe et al. (2017). To understand the influence of environmental disaster like as hurricanes on the spread of infectious disease dynamics is considerable issue. Chowell et al. (2019) studied the impact of severe rainfall on vector borne disease transmission in the subtropical parts of the world (southern coastal portions of the United States) and analysed the response of heavy rainfall on the dynamics of vector breeding capacity. Furthermore, despite human migration, vector movement should not be neglected in disease transmission.Traore (2020) studied non-linear mathematical model to investigate the stability of vector host disease model by considering the long-distance vector and human migration. Mishra (2021) studied vector host model to explain the impact of media on disease transmission. The model is analysed using two switching components based on the susceptible host and infected vector population and the impact of media on transmission rate is assumed to be dependent on number of cases and rate of change of cases. The effect of global warming temperature on the spread of carrier dependent (Singh, 2017) and bacteria-

dependent (Arora, 2021) diseases is studied and analysed.

It is worth to mention here that the impact of global warming temperature on the spread of vector borne infectious diseases has not been studied yet. In this article, we have analysed a mathematical model to study the effect of global warming on the spread of vector born diseases . Our aim is to explore the impact of global warming temperature on the vector host transmission dynamics by assuming that the vector population follows the logistic model which has intrinsic growth rate δ_0 and K, the carrying capacity of environment.

2 Mathematical Model

In this section we assume that total population size at time t, given by $N_1(t)$, is denoted into subclasses of individuals who are Susceptible population of size $S(t)$ and Infective population of size $I(t)$. Thus $N_1(t) = S(t) + I(t) + R(t)$. Furthermore, the total vector population size at time t is assumed to be $N_2(t)$ and is divided into subclasses of vectors that are Susceptible vector of size $M(t)$ and Infective vector of size $V(t)$. So we have $N_2(t) = M(t) + V(t)$. The mathematical model can be represented by the following non-linear differential equations

$$\frac{dE}{dt} = b_1 - \lambda_1 SI - \lambda_2 SV - \mu_1 S, \tag{1}$$

$$\frac{dI}{dt} = \lambda_1 SI + \lambda_2 SV - (\gamma + \mu_1)I, \tag{2}$$

$$\frac{dN_1}{dt} = b_1 - \gamma I - \mu_1 N_1, \tag{3}$$

$$\frac{dM}{dt} = \delta_0 \left(1 - \frac{N_2}{K}\right) N_2 - \beta MI + \psi N_2 (T - T_0) - \mu_2 M, \tag{4}$$

$$\frac{dV}{dt} = \beta MI - \mu_2 V, \tag{5}$$

$$\frac{dN_2}{dt} = \delta_0 \left(1 - \frac{N_2}{K}\right) N_2 + \psi N_2 (T - T_0) - \mu_2 N_2, \tag{6}$$

$$\frac{dT}{dt} = \alpha_1 (C - C_0) - \alpha_0 (T - T_0), \tag{7}$$

$$\frac{dC}{dt} = E_0 + \phi_1 (b_1 - \mu_1 N_1) - \phi_0 C, \tag{8}$$

with initial conditions $S(t) \geq 0, I(t) \geq 0, N_1(t) \geq 0, M(t) \geq 0, V(t) \geq 0, N_2(t) \geq 0, T(t) \geq 0, C(t) \geq 0$, where $N_1(t)$ is total population size at time t, and is divided into subclasses of individuals who are Susceptible host population of size $E(t)$ and Infective host population of size $I(t)$. Thus $N_1(t) = S(t) + I(t)$. Furthermore, $N_2(t)$, is the total vector population size at time t and is divided into subclasses of vectors that are Susceptible vector of size $M(t)$ and Infective vector of size $V(t)$. So we have $N_2(t) = M(t) + V(t)$.

We consider λ_1 as the rate of direct transmission for new infections. λ_2 is the biting rate of the infective vector on the susceptible host. The susceptible vectors become infected after biting the infective host at a rate β. We assume that the susceptible vectors after becoming infective host will remain infective throughout the life and will carry pathogen for whole life. The rate of emission of CO_2 from natural resources is denoted by E_0 and from anthropogenic sources is ϕ_1. The rate of natural depletion of CO_2 is denoted by ϕ_0. α_1 is the Temperature growth rate coefficient resulting from increase in CO_2 in the region. α_0 is the rate of natural depletion of temperature. δ_0 is the intrinsic growth rate of vector population and K is the carrying capacity of the environment. ψ denotes the rate of vector population growth due to temperature of the region. b_1 is the

birth rate for host population. γ is the disease induced death rate. μ_1 and μ_2 natural death rates of host and vector population respectively.

We assume that $\delta_0 - \mu_2 = \delta$ for equation (6). The equilibrium concentrations of Temperature and CO_2 are denoted by T_0 and C_0 respectively. The analysis of model $(1-8)$ is carried out by considering the reduced model using $N_1(t) = S(t) + I(t)$ and $N_2(t) = M(t) + V(t)$ as

$$\frac{dI}{dt} = \lambda_1(N_1 - I)I + \lambda_2(N_1 - I)V - (\gamma + \mu_1)I, \tag{9}$$

$$\frac{dN_1}{dt} = b_1 - \gamma I - \mu_1 N_1, \tag{10}$$

$$\frac{dV}{dt} = \beta(N_2 - V)I - \mu_2 V, \tag{11}$$

$$\frac{dN_2}{dt} = \delta N_2 - \delta_0 \frac{N_2^2}{K} + \psi N_2(T - T_0), \tag{12}$$

$$\frac{dT}{dt} = \alpha_1(C - C_0) - \alpha_0(T - T_0), \tag{13}$$

$$\frac{dC}{dt} = E_0 + \phi_1(b_1 - \mu_1 N_1) - \phi_0 C. \tag{14}$$

The following closed set Ω defines the region of attraction for model system (9-14).

$$\Omega = \{(I, N_1, V, N_2, T, C) \in \mathbf{R}_+^6 | 0 \le I \le N_1 \le \frac{b_1}{\mu_1}, 0 \le V \le N_2 \le \frac{K\delta}{\delta_0}, T_0 \le T \le T_{max}, C_0 \le C \le C_{max}\}$$

where $T_{max} = T_0 + \frac{b_1\phi_1\alpha_1}{\phi_0\alpha_0}$ and $C_{max} = C_0 + \frac{\gamma\phi_0}{\phi_1}$.

It can be verified that Ω is positively invariant with respect to the system $(9-14)$.

3 Equilibrium Analysis

In this section, the equilibrium analysis for the model (9-14) is carried out. The results of the equilibrium analysis are given in the following theorem. We obtain the following three feasible non-negative equilibria:
The equilibria for the model (9-14), can be obtained by setting right hand side of model (9-14) equal to zero.

The model clearly has a unique disease free equilibrium point E_0 in the region Ω, given by $E_0 = \left(\frac{b_1}{\mu_1}, 0, 0\right)$.

Theorem-1 There exists following three equilibria of the system (9-14).

Disease free equilibrium $E_1(0, N_1, 0, N_2, C, T)$, where $N_1 = \frac{b_1}{\mu_1}$, $N_2 = \frac{L\delta}{\delta_0}$, $C = C_0$,$T = T_0$.

Vector free equilibrium $E_2(0, \bar{N}_1, 0, 0, \bar{N}_2, \bar{C}, \bar{T})$.
Endemic equilibrium $E_3(I^*, N_1^*, V^*, N_2^*, C^*, T^*)$.
The existence of first equilibrium $E_1(0, N_1, 0, N_2, C, T)$ obvious.

3.1 Existance of $E_2(0, \bar{N}_1, 0, \bar{N}_2, \bar{C}, \bar{T})$

From equation (10) we have

$$\bar{I} = \frac{b_1 - \mu_1\bar{N}_1}{\gamma} \tag{15}$$

from equation (9) we have

$$\bar{N}_1 = \frac{b_1\lambda_1 + \gamma(\mu_1 + \gamma)}{\gamma}, \tag{16}$$

using (16) in (15) we have

$$\bar{I} = \frac{b_1\lambda_1 - \mu_1(\mu_1+\gamma)}{\lambda_1(\mu_1+\gamma)} \tag{17}$$

which exists if $b_1\lambda_1 - \mu_1(\mu_1 + \gamma) > 0$.

We define the Basic Reproduction number as

$$\mathcal{R}_0 = \frac{b_1\lambda_1}{\mu_1(\mu_1+\gamma)},$$

from equation (14) we have

$$\bar{C} = \frac{E_0}{\phi_0} + \frac{\phi_1(b_1-\mu_1\bar{N}_1)}{\phi_0} = C_0 + \frac{\phi_1(b_1-\mu_1\bar{N}_1)}{\phi_0}, \tag{18}$$

from equation (13) we have

$$\bar{T} = T_0 + \frac{\alpha_1\phi_1(b_1-\mu_1\bar{N}_1)}{\phi_0\alpha_0}. \tag{19}$$

Thus, the Vector free equilibrium point $E_2(0, \bar{N}_1, 0, \bar{N}_2, \bar{C}, \bar{T})$ exists if $\mathcal{R}_0 = \frac{b_1\lambda_1}{\mu_1(\mu_1+\gamma)} > 1$.

3.2 Existance of $E_3(I^*, N_1^*, V^*, N_2^*, C^*, T^*)$

After setting the equations (9-14), the non-trivial equilibrium $E_3(I^*, N_1^*, V^*, N_2^*, C^*, T^*)$ can be obtained as:
We have, from equation (14)

$$C = C_0 + \frac{\phi_1(b_1-\mu_1N_1^*)}{\phi_0}. \tag{20}$$

Substituting value of C from equation (20) in equation (13),

$$T = T_0 + \frac{\alpha_1\phi_1(b_1-\mu_1N_1^*)}{\phi_0\alpha_0}. \tag{21}$$

From equation (12) we have

$$N_2^* = \frac{K}{\delta_0}\big(\delta - \psi(T - T_0)\big), \tag{22}$$

substituting equation (21) in equation (22) we get,

$$N_2^* = \frac{K\big(\delta\alpha_0\phi_0 - \psi\alpha_1\phi_1(b_1-\mu_1N_1^*)\big)}{\delta_0\phi_0\alpha_0}. \tag{23}$$

Substituting equation (23) in equation (11) we get,

$$V^* = \frac{\beta K(\delta\alpha_0\phi_0 - \psi\alpha_1\phi_1\gamma I^*)}{\delta_0\phi_0\alpha_0(\beta I^* + \mu_2)}. \tag{24}$$

from equation (10), we have

$$N_1^* = \frac{b_1 - \gamma I^*}{\mu_1} \tag{25}$$

Now, substituting equations (24) and (25) in equation (9), we get the value of I^* from the following cubic equation

$$a_3I^{*3} + a_2I^{*2} + a_1I^* + a_0 = 0, \tag{26}$$

where

$$a_3 = \lambda_1\delta_0\phi_{\alpha_0}\beta(\gamma + \mu_1) > 0,$$
$$a_2 = -\big[\beta K\lambda_2\gamma\phi_1\alpha_1\psi\gamma(\gamma + \mu_1) + \lambda_1\delta_0\phi_0\alpha_0\big(b_1\beta - \mu_2(\gamma + \mu_1)\big)\big],$$
$$a_1 = \beta K\lambda_2\big(\phi_1\alpha_1\psi\gamma b_1 + \delta\phi_0\alpha_0(\gamma + \mu_1)\big) - \big(b_1\mu_2\lambda_1\delta_0\phi_0\alpha_0 + \mu_1\delta_0\phi_0\alpha_0\beta(\gamma + \mu_1)\big),$$
$$a_0 = -\big(\beta\lambda_2b_1\delta\phi_0\alpha_0K + \mu_1\mu_2\delta_0\phi_0\alpha_0(\gamma + \mu_1)\big) < 0.$$

We state the theorem given in (Burnside and Panton, 1935) to determine the existence of endemic equilibrium.

Theorem-2 Every equation of an odd degree has at least one real root of a sign opposite to that of its last term

and every equation of an odd degree has at least one real root of a sign opposite to that of its last term.

Since, $a_0 < 0$, therefore atleast one positive of equation (26) exists. Now, $a_3 > 0$ and $a_2 < 0$ if $b_1\beta - \mu_2(\gamma + \mu_1) > 0$.

Also $a_1 < 0$ if $\beta K\lambda_2\left(\phi_1\alpha_1\psi\gamma b_1 + \delta\phi_0\alpha_0(\gamma + \mu_1)\right) > \left(b_1\mu_2\lambda_1\delta_0\phi_0\alpha_0 + \mu_1\delta_0\phi_0\alpha_0\beta(\gamma + \mu_1)\right)$. Hence, if $a_1 < 0$ and $b_1\beta > \mu_2(\gamma + \mu_1)$, then by using Descartes' Rule the endemic equilibrium point $E_3(I^*, N_1^*, V^*, N_2^*, C^*, T^*)$ exists uniquely.

4 Stability Analysis

4.1 Local stability analysis

In this section, the local stability of equilibria E_1, E_2 and E_3 are stated. The local stability of equilibrium points E_1 and E_2 are studied by determining the sign of eigen values of corresponding Jacobian matrix and the local stability of endemic equilibrium E_3 is investigated by using Lapyunov's theory.

Let $\mathcal{J}_i$ be the Jacobian matrix evaluated at equilibrium points $E_i(i = 1,2)$. The variational matrix $\mathcal{J}_i$ for the model (9-14) is given by

$$\mathcal{J}_i = \begin{pmatrix} \lambda_1 N_1 - 2\lambda_1 I - \lambda_2 V - (\mu_1 + \gamma) & \lambda_1 I - \lambda_2 V & \lambda_2 N_1 - \lambda_2 I & 0 & 0 & 0 \\ -\gamma & -\mu_1 & 0 & 0 & 0 & 0 \\ \beta(N_2 - V) & 0 & -\beta I - \mu_2 & \beta I & 0 & 0 \\ 0 & 0 & 0 & \delta - \frac{2\delta_0 N_2}{K} + \psi(T - T_0) & 0 & \psi N_2 \\ 0 & \phi_1\mu_1 & 0 & 0 & -\phi_0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_1 & -\alpha_0 \end{pmatrix},$$

The variational matrix $\mathcal{J}_1$ corresponding to the model $(9 - 14)$ at equilibrium point E_1 is given as:

$$\mathcal{J}_1 = \begin{pmatrix} \frac{b_1\lambda_1}{\mu_1} - (\mu_1 + \gamma) & 0 & \lambda_2\frac{b_1}{\mu_1} & 0 & 0 & 0 \\ -\gamma & -\mu_1 & 0 & 0 & 0 & 0 \\ \frac{\delta\beta K}{\delta_0} & 0 & -\mu_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\delta & 0 & \frac{\psi\delta K}{\delta_0} \\ 0 & \phi_1\mu_1 & 0 & 0 & -\phi_0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_1 & -\alpha_0 \end{pmatrix},$$

The characteristic polynomial is given as

$$(-\mu_1 - \xi)(-\delta - \xi)(-\phi_0 - \xi)(-\alpha_0 - \xi),$$

$$\left(\xi^2 + \left(\mu_1 + \mu_2 + \gamma - \frac{\lambda_1 b_1}{\mu_1}\right)\xi - \left(\frac{b_1}{\mu_1}\left(\mu_2\lambda_1 + \frac{\lambda_2\beta}{\delta_0}\right) - (\mu_1 + \gamma)\right)\right) = 0.$$

The eigen value are : $-\mu_1, -\delta, -\phi_0, -\alpha_0$ and the remaining two eigen values can be obtained from following quadratic equation

$$\xi^2 + e_1\xi + e_0 = 0, \tag{27}$$

where

$$e_1 = \left(\mu_1 + \mu_2 + \gamma - \frac{\lambda_1 b_1}{\mu_1}\right),$$

$$e_0 = \left((\mu_1 + \gamma) - \frac{b_1}{\mu_1}\left(\mu_2\lambda_1 + \frac{\lambda_2\beta}{\delta_0}\right)\right).$$

If both the roots of equation (27) are negative or have negative real parts then the equilibrium point E_1 is

stable otherwise unstable. Now $e_1 > 0$ if $\mu_1 + \mu_2 + \gamma > \frac{\lambda_1 b_1}{\mu_1}$) and $e_0 > 0$ if $\mu_1 + \gamma > \frac{b_1}{\mu_1}\left(\mu_2\lambda_1 + \frac{\lambda_2\beta}{\delta_0}\right)$.

Using Routh-Hurwitz criterion, a second-degree polynomial with all the coefficients positive will have negative roots. Then the equilibrium point E_1 is stable if $\mu_1 + \mu_2 + \gamma > \frac{\lambda_1 b_1}{\mu_1}$) and $\mu_1 + \gamma > \frac{b_1}{\mu_1}\left(\mu_2\lambda_1 + \frac{\lambda_2\beta}{\delta_0}\right)$ otherwise unstable.

The variational matrix $\mathcal{J}_2$ corresponding to the model $(9-14)$ at equilibrium point E_2 is given as :

$$\mathcal{J}_2 = \begin{pmatrix} \lambda_1\bar{N}_1 - 2\lambda_1\bar{I} - (\mu_1+\gamma) & \lambda_1\bar{I} & \lambda_2\bar{N}_1 - \lambda_2\bar{I} & 0 & 0 & 0 \\ -\gamma & -\mu_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\beta\bar{I} - \mu_2 & \beta\bar{I} & 0 & 0 \\ 0 & 0 & 0 & \delta + \psi(\bar{T} - T_0) & 0 & 0 \\ 0 & \phi_1\mu_1 & 0 & 0 & -\phi_0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_1 & -\alpha_0 \end{pmatrix},$$

The Jacobian matrix $\mathcal{J}_2$ has one positive eigen value $\delta + \psi(\bar{T} - T_0)$, therefore, the equilibrium point E_2 is locally unstable. Further, we study the linear stability of endemic equilibrium point E_3 by using Lyapunov's direct method. We thus state the following theorem

Theorem-3 The endemic equilibrium E_3 is locally asymptotically stable provided the following conditions hold

$$\gamma\lambda_2^2 V^{*2} < \lambda_1\mu_2(\lambda_1 I^{*2} + \lambda_2 N_1^* V^*) \tag{28}$$

$$16\beta\gamma K\psi^2\mu_1^2\phi_1^2\lambda_2^2 K(N_2^* - I^*) < \delta_0\mu_2\lambda_1\beta\phi_0\alpha_0^2 N_2^2. \tag{29}$$

Proof. we linearise the system (9-14), using the transformation $I = I^* + x_1$, $N_1 = N_1^* + x_2$, $V = V^* + x_3$, $N_2 = N_2^* + x_4$, $C = C^* + x_5$ and $T = T^* + x_6$

Consider the following positive definite function

$$U = \frac{m_1}{2}x_1^2 + \frac{m_2}{2}x_2^2 + \frac{m_3}{2}x_3^2 + \frac{m_4}{2}x_4^2 + \frac{m_5}{2}x_5^2 + \frac{m_6}{2}x_6^2 \,. \tag{30}$$

Now, differentiating equation (30) w.r.t 't' and using linearised system (9-14) corresponding to E_4, $\frac{dU}{dt}$ can be written as

$$\frac{dU}{dt} = -m_1\left(\lambda_1 I^* + \lambda_2 V^* + \lambda_2\frac{(N_1^* - I^*)}{I^*}V^*\right)x_1^2 - m_2\mu_2 x_2^2 - m_3\beta I^* x_3^2 - m_4\frac{\delta_0 N_2^*}{K}x_4^2 - m_5\alpha_0 x_5^2$$
$$-m_6\phi_0 x_6^2 + m_1\lambda_2(N_2^* - I^*)x_1x_3 + (m_1\lambda_1 I^* - \mu_2\gamma)x_1x_2 + m_1\lambda_2 V^* x_1x_2 + m_3\beta I^* x_3x_4$$
$$+m_5\alpha_1 x_5x_6 - m_6\phi_1\mu_1 x_2x_6 + m_4\psi x_4x_5 \,.$$

After choosing $m_2 = 1$, $m_2 = \frac{\gamma}{\lambda_1 I^*}$, we have the following inequalities for $\frac{dU}{dt}$ to be negative definite:

$$\gamma\lambda_2^2 V^{*2} < \lambda_1\mu_2(\lambda_1 I^{*2} + \lambda_2 N_1^* V^*)\,, \tag{31}$$

$$m_3 > \frac{\gamma\lambda_2^2(N_2 - I^*)^2}{\lambda_1\beta I^*(\lambda_1 I^{*2} + \lambda_2 N_1^* V^*)}\,, \tag{32}$$

$$m_4 < \frac{\delta_0\alpha_0 N_2^*}{\psi^2}m_5\,, \tag{33}$$

$$m_5 < \frac{\alpha_0\phi_0}{\alpha_1}m_6\,, \tag{34}$$

$$m_6 < \frac{\mu_2}{\phi_1^2\mu_1^2}\,, \tag{35}$$

$$m_3 < \frac{\delta_0 N_2^2}{\beta K I^*}m_4\,, \tag{36}$$

Further, we choose $m_4 = \frac{\delta_0\phi_0\mu_2\alpha_0^2N_2}{8\psi^2\phi_1^2\mu_1^2}$, $m_5 = \frac{\alpha_0\phi_0\mu_2}{4\phi_1^2\mu_1^2}$ and $m_6 = \frac{\mu_2}{2\phi_1^2\mu_1^2}$, m_3 can be choosen by using (32) and (36) if the following inequality is satisfied:

$$16\beta\gamma K\psi^2\mu_1^2\phi_1^2\lambda_2^2K(N_2^* - I^*) < \delta_0\mu_2\lambda_1\beta\phi_0\alpha_0^2N_2^2 \tag{37}$$

which proves the Theorem (3).

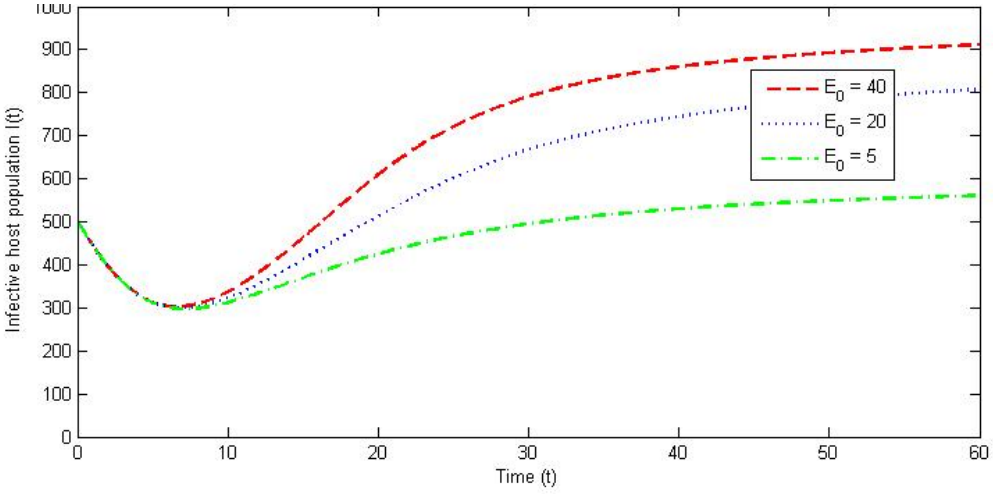

Fig. 1 Variation of Infective host population with respect to time t for distinct values of E_0.

4.2 Non-linear stability analysis

The result of non-linear stability analysis of endemic equilibrium E_3 is presented in the following theorem.

Theorem-4 The endemic equilibrium E_3 is non-linearly asymptotically stable in the region provided the following conditions are satisfied:

$$\gamma\lambda_2^2V^2 < \mu_1\lambda_1^2I^2, \tag{38}$$

$$16\phi_1^2\alpha_1^2\psi^2K^2V^2\left(\lambda_2\gamma V^*(N_1^* - I^*) + \lambda_1\beta I^*(N_1^* - I^*)\right)^2 < \delta_0\phi_0^2\alpha_1^2\lambda_1^2\gamma V^*I^{*2}N_2^2 \tag{39}$$

Proof. Consider a positive definite function as:

$$W = n_1\left(I - I^* - I^*\ln\frac{I}{I^*}\right) + \frac{n_2}{2}(N_1 - N_1^*)^2 + n_3\left(V - V^* - V^*\ln\frac{V}{V^*}\right) + n_4\left(N_2 - N_2^* - N_2^*\ln\frac{N_2}{N_2^*}\right)$$
$$+ \frac{n_5}{2}(T - T^*)^2 + \frac{n_6}{2}(C - C^*)^2, \tag{40}$$

where, $n_i(i = 1,2,\ldots 6)$ are positive chosen suitably. Differentiating (40) and using the using linearised system (9-14), after some algebraic calculations, we get

$$\frac{dW}{dt} = (n_1\lambda_1 - n_2\gamma)(N_1 - N_1^*)(I - I^*) - n_1\frac{\lambda_2VN_1}{II^*}(I - I^*)^2 - \left[\frac{n_1}{2}\lambda_1(I - I)^2 - n_1\frac{\lambda_2V}{I^*}(N_1 - N_1^*)(I - I^*)\right.$$
$$\left. + \frac{n_2}{2}\mu_1(N_1 - N_1^*)^2\right] - \left[\frac{n_1}{2}\lambda_1(I - I^*)^2 - \left(n_1\frac{\lambda_2V}{I^*}(N_1^* - I^*) + n_2\frac{\beta}{V^*}(N_2^* - V^*)\right)(I - I^*)(V - V^*)\right.$$
$$\left. + \frac{n_3}{2}\frac{\beta N_2I}{VV^*}(V - V^*)^2\right] - \left[\frac{n_4}{2}\frac{\delta_0}{K}(N_2 - N_2^*)^2 - n_3\frac{\beta I}{V}(N_2 - N_2^*)(V - V^*) + \frac{n_3}{2}\frac{\beta N_2I}{VV^*}(V - V^*)^2\right] -$$
$$\left[\frac{n_4}{2}\frac{\delta_0}{K}(N_2 - N_2^*)^2 - n_4\psi(T - T^*)(N_2 - N_2^*)^2 + \frac{n_5}{2}\alpha_0(T - T^*)^2\right] - \left[\frac{n_6}{2}\phi_0(C - C^*)^2 - n_5\alpha_1\right.$$

$$(C-C^*)^2(T-T^*)^2+\frac{n_5}{2}\alpha_0(T-T^*)^2\Big]-\Big[\frac{n_2}{2}\mu_1(N_1-N_1^*)^2+n_6\phi_1\mu_1(N_1-N_1^*)(C-C^*)$$

$$+\frac{n_6}{2}\phi_0(C-C^*)^2\Big]$$

Now, using Sylvester criteria in the above expression and after choosing $n_1=\frac{\gamma}{\lambda_1}$, $n_2=1$, $n_4=\frac{\delta_0\phi_0^2\alpha_0^2}{16\phi_1^2\alpha_1^2\psi^2K}$, $n_5=\frac{\phi_0^2\alpha_0}{4\phi_1^2\alpha_1^2}$ and $n_6=\frac{\phi_0}{2\phi_0^2}$.

We get

$$\gamma\lambda_2^2V^2<\mu_1\lambda_1^2I^2$$

and

$$\frac{\gamma V\left(\lambda_2\gamma V^*(N_1^*-I^*)+\lambda_1\beta I^*(N_1^*-I^*)\right)^2}{\lambda_1^2\gamma^2N_2V^*I^{*2}}<n_3<\frac{\delta_0^2\phi_0^2\alpha_0^2N_2V^*}{16\phi_1^2\alpha_1^2\psi^2K^2V}.$$

i.e.

$$16\phi_1^2\alpha_1^2\psi^2K^2V^2\left(\lambda_2\gamma V^*(N_1^*-I^*)+\lambda_1\beta I^*(N_1^*-I^*)\right)^2<\delta_0\phi_0^2\alpha_1^2\lambda_1^2\gamma V^*I^{*2}N_2^2.$$

which proves the Theorem (4).

5 Numerical Simulation

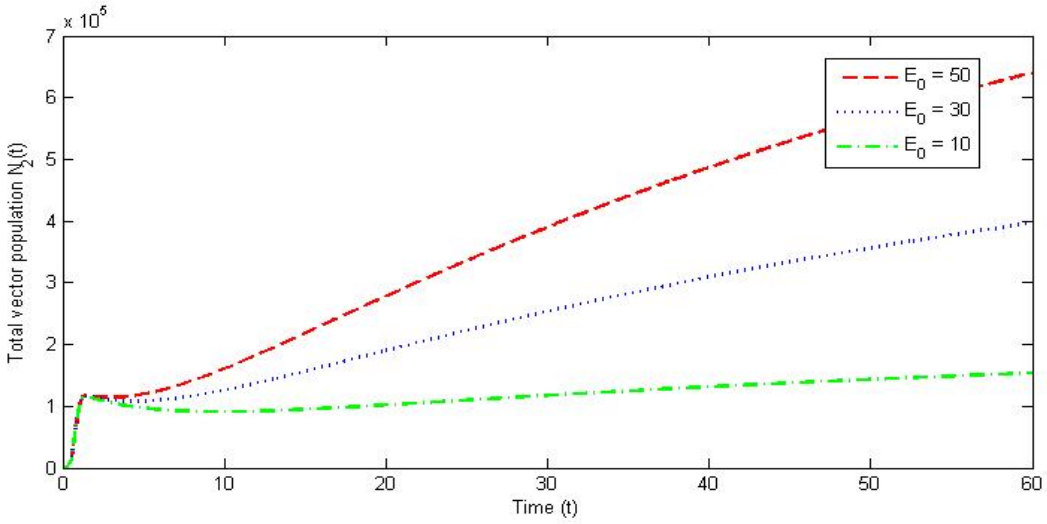

Fig. 2 Variation of Total vector population with respect to time t for distinct values of E_0

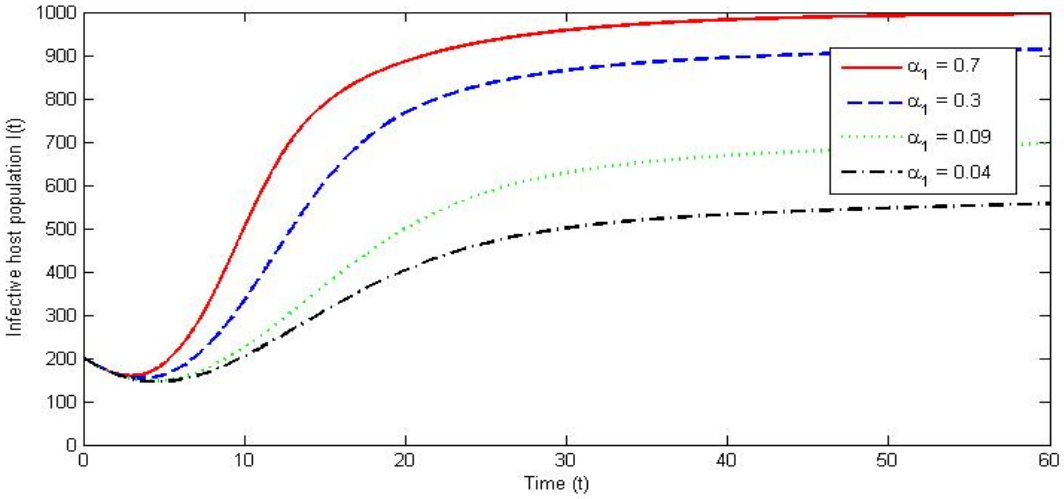

Fig. 3 Variation of Infective host population with respect to time t for distinct values of α_1.

In this section numerical simulation is presented to study the dynamic behaviour of the system (9-14) and to explain the applicability of the results discussed above. The parameter values considered for simulation are as: $b_1 = 120, \lambda_1 = 2.1 \times 10^{-5}, \lambda_2 = 1.1 \times 10^{-6}, \beta = 5.1 \times 10^{-4}, \gamma = 0.012, \psi = 0.04, K = 10^3, \mu_1 = 0.1, \mu_2 = 0.004, \delta = 0.3, T_0 = 14, C_0 = 2.5, \delta_0 = 0.6, \alpha_1 = 0.10, \alpha_0 = 0.1, \phi_0 = 0.016, \phi_1 = 0.576 \times 10^{-3}, E_0 = 5$. The equilibrium values for endemic equilibrium are computed as $E_3 = 6821.8157, 7627.6422, 597.5471, 598.2341, 15.4735, 315.4470$. The eigenvalues corresponding to variational matrix of endemic equilibrium are :

$$-0.015996, -0.018797, -0.130198, -0.2, -0.35894, -3.483126.$$

Since, all the eigenvalues corresponding to E_3 are negative for the above set of parameter values, therefore, the endemic equilibrium E_3 is locally stable. The results of the system (9-14) are shown graphically in Figs 1-6.

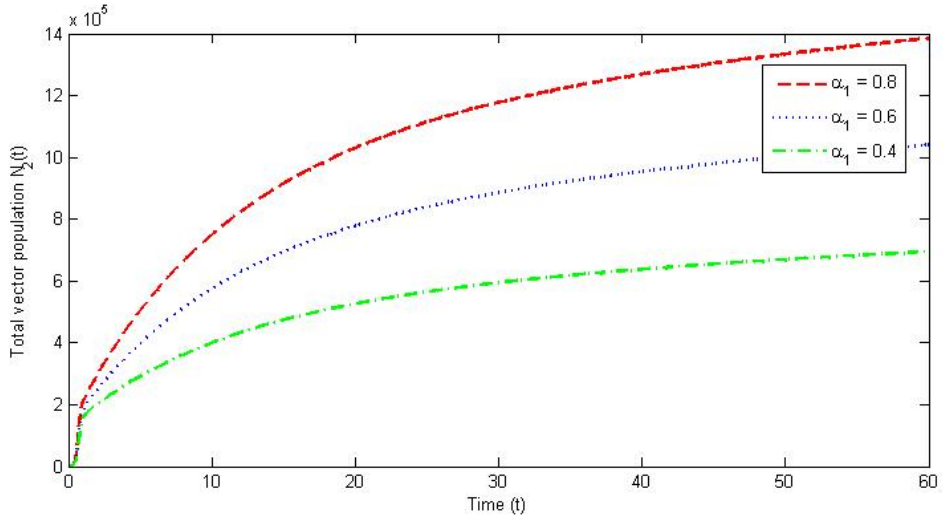

Fig. 4 Variation of Total vector population with respect to time t for distinct values of α_1

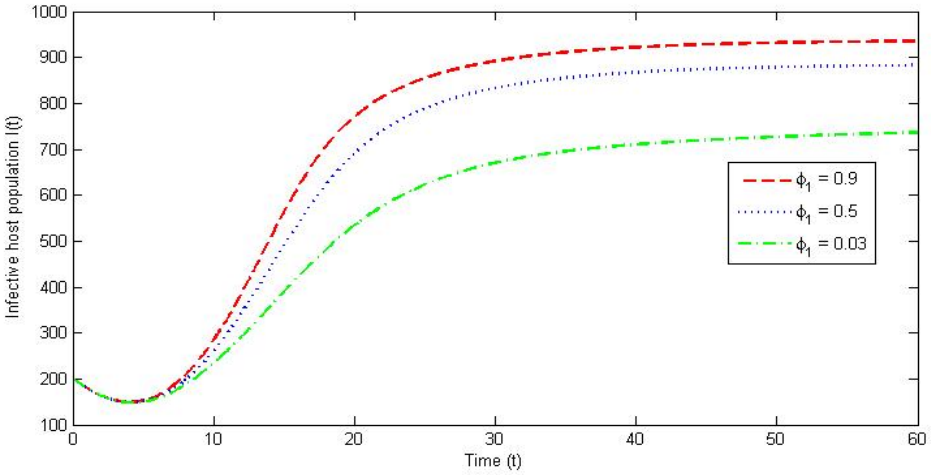

Fig. 5 Variation of Infective host population with respect to time t for distinct values of ϕ_0

Fig. 1 and Fig. 2 shows the variation of infective host and total vector population with time, respectively, for different values of E_0, the rate of emission of CO_2 from natural sources. It is clear that as the rate of emission of CO_2 increases, the infective host population increases (Fig. 1). This increase in infective population is due to increase in vector population in the environment (Fig. 2). α_1 is the Temperature growth rate coefficient resulting from increase in CO_2 in the region. The effect of Temperature growth rate coefficient resulting from increase in CO_2 on infective host and total vector population with time t is shown in Fig. 3 and Fig.4, respectively, for different value of α_0, the growth rate coefficient of Temperature resulting from increase in CO_2 . It is seen that as the rate of emission of CO_2 from anthropogenic sources increase ϕ_1, the infective host population increases Fig. 5. This increase in the infective host population is due to increase of vector population in the environment as a result of increase in emission of CO_2 from anthropogenic sources. From Fig. 6, it is clear that the total vector population increases with increase in emission of CO_2 from anthropogenic sources. It is, therefore, observed that the prevalence of vector borne diseases can be reduced if the rate of emission of CO_2 from anthropogenic sources ϕ_1 and natural sources E_0; and Temperature growth rate coefficient resulting from increase in carbon dioxideα_1, in the region can be reduced.

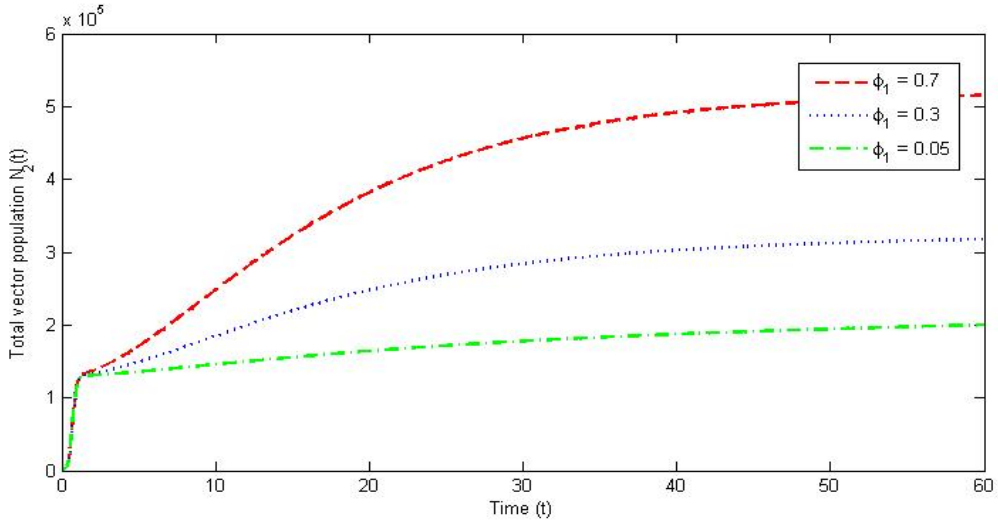

Fig. 6 Variation of Total vector population with respect to time t for distinct values of ϕ_1

6 Conclusion

In this paper, a non-linear mathematical model is proposed to study the effect of global warming on the vector borne diseases. In modeling the process, the total human population is divided into subclasses of susceptible host and infective host; and the vector population is divided into subclasses of susceptible vector and infective vector. The model exhibits three equilibria; namely disease free equilibrium, vector free equilibrium and endemic equilibrium. We have assumed that, in addition to natural sources,the human related activities are also responsible for increased temperature , due to increase in concentration of CO_2. The analysis of the model reviles that the increase in temperature, due to increase in CO_2 concentration in environment, leads to fast spread of vector borne disease. The rise in temperature results in increase of vector population and this increase in vector population results in fast spread of vector born disease. The model analysis suggests that if the emission of CO_2 both from natural and human related activities is reduced, the spread of vector borne disease can be reduced. The model analysis shows that as the CO_2 concentration in the environment increases, giving rise to the increase in temperature, then not only the vector population increases but also the infective host population increases, resulting in the fast spread of disease.

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