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MATHEMATICAL MODELING AND SIMULATING OF *HELICOBACTER PYLORI* TREATMENT AND TRANSMISSION IMPLICATIONS ON STOMACH CANCER DYNAMICS

GLORY KAWIRA MUTUA[1,*], CYRUS GITONGA NGARI[2], GRACE GAKII MUTHURI[3], DOMINIC MAKAA KITAVI[1]

[1]Department of Mathematics and Statistics, University of Embu, Kenya

[2]Department of Pure and Applied Science, Kirinyaga University, Kenya

[3]Department of mathematics, Meru University of Science and Technology, Kenya

Abstract: Deterministic mathematical model of the nonlinear first-order differential equation is proposed considering both direct and indirect contact transmission to capture some of the control measures such as treatment in limiting the infection. Six compartments are considered in the model that is; susceptible humans, exposed humans, infected humans with *H. pylori*, treated humans, infected humans with stomach cancer and the bacteria concentration from the environment. The qualitative behavior of the model was performed including, the existence of nonnegative invariant solution, boundness region, equilibria (both disease-free as well as endemic) and stabilities of two-equilibrium point. Moreover, control reproduction number and bifurcation analysis were also studied. Based on the analysis of sensitivity, the method of partial rank correlation coefficient (PRCC) and Latin hypercube sampling (LHS) was studied to find out which parameters are useful for the model. Furthermore, sensitivity analysis of some parameters was also studied based on the control reproduction number. The simulation results show that increasing the *Helicobacter pylori* infections treatment rate, has a vital role in the reduction of infections and stomach cancer in the community. Therefore, we concluded that effective treatment rate and low contact rate are most significant to eradicate stomach cancer from

*Corresponding author

E-mail address: glorykawiramutua@yahoo.com

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the community.

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1. INTRODUCTION

Helicobacter pylori (*H. pylori)* is a microaerophilic, gram-negative and spiral shaped bacterium which was first discovered in 1982 [1]. It is the most serious human bacterial, which colonizes the host gastric of more than half of human population globally, especially in adulthood [2,3]. *H. pylori* infection has been discussed as the primary risk factor associated with stomach cancer and it significantly increases chances of developing peptic ulcers and cancer diseases in the stomach [3].

Gastric cancer mainly begins with lesions which develop into inflammation, gastric gastritis, gastric atrophy and then followed by gastric cancer. Gastric cancer commonly is ranked to be the 5^{th} cancer globally after lung, breast , colorectal and prostate cancer [4] and 3^{rd} most leading cancer death globally [5], after lung and colorectal cancer with estimated death cases of about 783,000 [6] and commonly in developing countries than developed countries [7]. In Africa, gastric cancer is ranked as 12^{th} most prevalent stomach cancer with estimated death rate as 3.8 to 100,000 and estimated incidence rate as 4 to 100,000. The incidence and death rate are higher in Africa than in developed countries like United State [8]. According to GLOBACAN 2020 gastric cancer is ranked to be 5^{th} causes of death in Kenya for about 1,501 death cases and about 1,781 new cases of gastric cancer.

Helicobacter Pylori is commonly transmitted directly from infected to a susceptible person through oral-to-oral route or faecal to oral route which includes kissing, vomiting, oral sex, breastfeeding. Also, the human pathogen can be transmitted by environmental factors such as using contaminated water and contaminated food [6]. The common risk factors involved with *H. pylori* includes overcrowding conditions, poor social economic status, inadequate sanitations, water-borne and food transmission, migration to highly vulnerable regions and infection status of the family members. The most common symptoms associated with the *H. pylori* includes Abdominal pains, bloating and fullness, ingestion and nausea [9].

Infection with *H. pylori* is endemic mainly in countries which are developing and despite current progress in public health goals such as safe water, improved sanitation and proper medical care

coverage, remains a common public health problem. Understanding the important mechanism of transmission of disease is more vital for preventive and also intervention strategies against stomach cancer. In the previous studies, dynamics of *H. pylori* infections have been studied for example Cousins et al. [10] formulated a model on dynamics of *campylobacter* considering direct and indirect pathway which assumed the insect vector as a mechanical vector for disease transmission. Rupnow et al. [11] applied SEIR compartmental model on dynamics of infection by *H. pylori* in United State. The results stressed on the trends of infection in the future. No environment to human contact was considered in this study. Moreover, Siewe et al. [12] proposed SEIR model on the role of assymptomatic infection which emphasized on different stages of assymptomatic state of the disease. Therefore, this study aims to incorporate the role of treatment as a control strategy and also consider the implications of *H. pylori* in stomach cancer dynamics. This raises a vital question, does *H. pylori* affect the spread of stomach cancer? The qualitative behaviour, sensitivity analysis and the numerical simulations of the mathematical model are performed in this study.

2. MODEL FORMULATION

The model considers human-human and environment-human transmission. The total population of individuals is categorized into five classes which comprises of, Susceptible S(t): these are humans who are at high risk of getting the infection sometimes in the future, Exposed E(t): are individuals who have not developed symptoms of *H. pylori* infection but can transmit the infection, Infected I(t): are humans infected with *H. pylori*. Treated T(t): includes humans who have received treatment at a time $t > 0$ after the infection with *H. pylori*. Infected C(t): these are individuals who have been infected with stomach cancer. The forces of infection of the model are; $\lambda_1 = \beta_1(E + \eta_1 I + \eta_2 T + \eta_3 C)$, where β_1 is the ingestion rate of *H. pylori* through human-human interaction and $\eta_1 \ldots \eta_3 < 1$ accounts for infectiousness among individuals with *H. pylori* infection and $\lambda_2 = \frac{(1-\varepsilon)\beta_2 H}{K+H}$, where β_2 *H. pylori* ingestion rate through environment-human, K is the concentration of *Helicobacter pylori* in waters or food, ε is the use of hygiene, safe water or sanitation and $\frac{H}{K+H}$ is the probability of humans consuming contaminated water or foods with infection causing bacteria. The assumptions of the study are as follows, the strongest

risk factor associated with stomach cancer is *Helicobacter pylori* infection [13]. The size of human population is constant. All the parameters are non-negative. All individuals are susceptible in the community, since *Helicobacter pylori* infection spread to all parts of the community. The birth and the death rate are not equal. There is no significance to the recovery from natural immunity. It is possible to determine the area.

Table 1: Parameter description

Parameter	**Description**
π	Individuals birth rate
μ	Natural individual mortality rate
λ_1	Human induced force of infection
λ_2	Environment induced force of infection
ρ	Exposed individual infectious rate
ω	Treatment rate of I (t)
θ	Susceptible rate of individuals who have treated
δ	Stomach cancer induced mortality rate
γ	Rate at which infected I(t) individuals gets stomach cancer
α	Rate at which treated individuals gets stomach cancer
τ	Natural death of *H. pylori*
ψ_1	Discharge of *H. pylori* from exposed class
ψ_2	Discharge of *H. pylori* from infected class
ψ_3	Discharge of *H. pylori* from treated class
ψ_4	Discharge of *H. pylori* from stomach cancer class

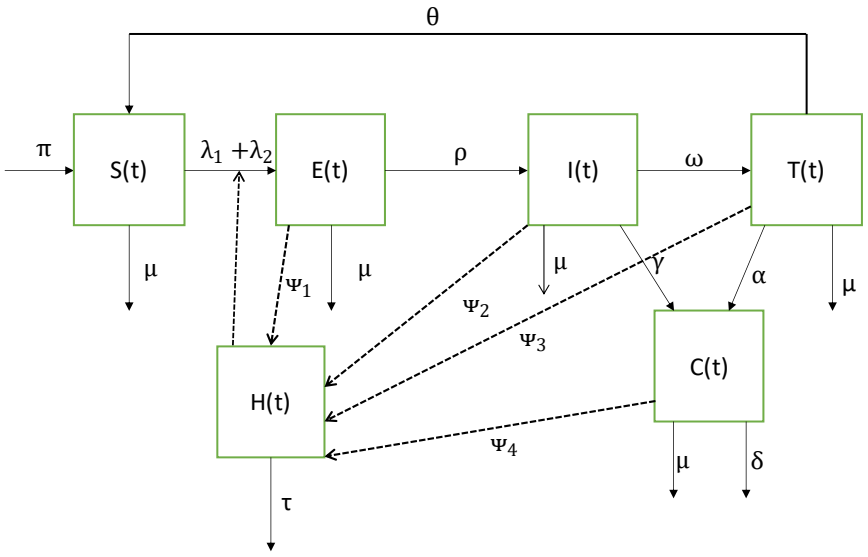

Figure 1: Flow diagram of the model

2.1 Model equations

$$\frac{dS}{dt} = \pi + \theta T - (\mu + \lambda_1 + \lambda_2)S \tag{1}$$

$$\frac{dE}{dt} = (\lambda_1 + \lambda_2)S - (\mu + \rho)E \tag{2}$$

$$\frac{dI}{dt} = \rho E - (\mu + \omega + \gamma)I \tag{3}$$

$$\frac{dT}{dt} = \omega I - (\alpha + \mu + \theta)T \tag{4}$$

$$\frac{dC}{dt} = \gamma I + \alpha T - (\mu + \delta)C \tag{5}$$

$$\frac{dH}{dt} = \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H \tag{6}$$

3. MODEL ANALYSIS

3.1 Positivity

Theorem 1. Let $R=\{S(t),E(t),I(t),T(t),C(t),H(t)\}\in R_{+}^{6}$:

$S(0)\geq 0,E(0)\geq 0,I(0)\geq 0,T(0)\geq 0,C(0)\geq 0,H(0)\geq 0$ }; then the solutions of $\{S,E,I,T,C,H\}$ are nonnegative for $t\geq 0$.

Proof. The prove of the theorem follows that,

From the first differential equation (1):

$$\frac{dS}{dt}=\pi+\theta T-(\mu+\lambda_1+\lambda_2)S$$

$$\frac{dS}{dt}\geq-(\mu+\lambda_1+\lambda_2)S \tag{7}$$

$$\int\frac{dS}{S}\geq-\int_0^t(\mu+\lambda_1+\lambda_2)dr \tag{8}$$

Solving (8) using separation of variable, we get,

$$S(t)\geq e^{c}\,exp-\int_0^t(\mu+\lambda_1+\lambda_2)dr \tag{9}$$

Applying initial condition, (9) becomes,

$$S(t)\geq S(0)exp-\int_0^t(\mu+\lambda_1+\lambda_2)dr\geq 0 \tag{10}$$

From equation (2), we have

$$\frac{dE}{dt}=(\lambda_1+\lambda_2)S-(\mu+\rho)E$$

$$\frac{dE}{dt}\geq-(\mu+\rho)E \tag{11}$$

$$\int\frac{dE}{E}\geq-\int(\mu+\rho)dt \tag{12}$$

$$E(t)\geq E(0)exp-(\mu+\rho)t\geq 0 \tag{13}$$

Similarly, it follows that from the third to sixth differential equations of the model, we take similar steps to get

$$I(t) \geq I(0)\exp-\left(\mu+\omega+\gamma\right)t \geq 0 \tag{14}$$

$$T(t) \geq T(0)\exp-(\alpha+\mu+\theta)t \geq 0 \tag{15}$$

$$C(t) \geq C(0)\exp-\left(\mu+\delta\right)t \geq 0 \tag{16}$$

$$H(t) \geq H(0)exp-(\tau)t \geq 0 \tag{17}$$

Thus, the theorem is proved.

3.2. Boundness of the solution

The model can be divided into two main parts; Human population R_N and concentration of bacteria from the environment R_H such that,

$R_N = \{S(t), E(t), I(t), T(t), C(t)\} \in \mathbb{R}_+^5 : S + E + I + T + C = N$ and

$R_H = \{H(t)\} \in \mathbb{R}_+^1$ respectively.

We first consider the boundness of system (1) to (5) at time t, given as

$$N(t) = S(t) + E(t) + I(t) + T(t) + C(t) \tag{18}$$

By differentiating (18), gives

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dC}{dt} \tag{19}$$

Substituting system (1)-(5) into (19), we have

$$\frac{dN}{dt} = \pi - \mu N - \delta C \tag{20}$$

If there is no infectious human with Stomach cancer, it indicates that $(\delta = 0)$ then (20) becomes

$$\frac{dN}{dt} \leq \pi - \mu N(t) \tag{21}$$

By using method of separation of variables of inequality, we have

$$\frac{-1}{\mu}\ln\left(\pi - \mu N(t)\right) \leq t + c$$

$$\pi - \mu N(t) \geq De^{-\mu t} \tag{22}$$

Solving (22) and evaluating as t approaches to ∞, we have:

$$\lim_{t\to\infty} N(t) = \frac{\pi}{\mu} \tag{23}$$

Implying that,

$$0 \le N(t) \le \frac{\pi}{\mu} \tag{24}$$

Therefore, the model is bounded in the domain

$$R_N = \left\{ (S,E,I,T,C) \in \mathbb{R}^5_+ : 0 \le N(t) \le \frac{\pi}{\mu} \right\}.$$

We then consider the boundness solution for the bacteria concentration at time t

From (6), we have

$$\frac{dH}{dt} = \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H$$

$$\frac{dH}{dt} + \tau H = \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C \tag{25}$$

Let $M = \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C$ which is the recruitment rate of the bacteria from infectious classes. We then obtain a differential inequality

$$\frac{dH}{dt} + \tau H \le M \tag{26}$$

By use of integration factor method, we obtained

$$H \le \frac{M}{\tau} + ce^{-\tau t} \tag{27}$$

Where c is the constant.

As t goes to ∞, we have:

$$\lim_{t\to\infty} H(t) = \frac{M}{\tau} \tag{28}$$

Which implies that,

$$0 \le H(t) \le \frac{M}{\tau} \tag{29}$$

$$R_H = \left\{ H(t) \in \mathbb{R}^1_+ : 0 \le H(t) \le \frac{M}{\tau} \right\}$$

Therefore, the model is bounded as;

$$R = \left[S, E, I, T, C, H \ge 0 : N(t) \le \frac{\pi}{\mu}, H(t) \le \frac{M}{\tau} \right]$$

Thus, the model makes biological sense and it is well posed.

3.3 Disease free equilibrium point (DFE)

DFE of the system (1) – (6) is obtained by setting $E = 0$, $T = 0$, $C = 0$ and $H = 0$ Therefore, after solving. We obtained

$$B^0 = (S^0, E^0, I^0, T^0, C^0, H^0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right)$$

3.4. The control reproduction number (R_C)

To obtain control reproduction number, we used next generation method. Non-negative matrix F and non-singular matrix V represents new infections terms and transfer of infections terms respectively (At DFE). Thus,

$$f = \begin{bmatrix} (\lambda_1 + \lambda_2)S \\ 0 \\ 0 \\ 0 \\ \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C \end{bmatrix} \tag{30}$$

$$v = \begin{bmatrix} (\mu + \rho)E \\ -\rho E + (\mu + \omega + \gamma)I \\ -\omega I + (\mu + \alpha + \theta)T \\ -\gamma I - \alpha T + (\mu + \delta)C \\ \tau H \end{bmatrix} \tag{31}$$

Where, $k_1 = \mu + \rho$, $k_2 = \mu + \omega + \gamma$, $k_3 = \mu + \alpha + \theta$ and $k_4 = \mu + \delta$

At DFE point, Jacobian matrices of f and v was evaluated to find out Matrices F and V respectively.

$$F=\begin{bmatrix} S^0\beta_1 & S^0\beta_1\eta_1 & S^0\beta_1\eta_2 & S^0\beta_1\eta_3 & \frac{S^0(1-\epsilon)\beta_2}{K} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \Psi_1 & \Psi_2 & \Psi_3 & \Psi_4 & 0 \end{bmatrix} \quad (32)$$

$$V=\begin{bmatrix} k_1 & 0 & 0 & 0 & 0 \\ -\rho & k_2 & 0 & 0 & 0 \\ 0 & -\omega & k_3 & 0 & 0 \\ 0 & -\gamma & -\alpha & k_4 & 0 \\ 0 & 0 & 0 & 0 & \tau \end{bmatrix}$$

Inverse of V is evaluated and given by

$$V^{-1}=\begin{bmatrix} \frac{1}{k_1} & 0 & 0 & 0 & 0 \\ \frac{\rho}{k_1k_2} & \frac{1}{k_2} & 0 & 0 & 0 \\ \frac{\rho\omega}{k_1k_2k_3} & \frac{\omega}{k_2k_3} & \frac{1}{k_3} & 0 & 0 \\ \frac{\alpha\rho\tau\omega+\gamma\rho\tau k_3}{\tau k_1k_2k_3k_4} & \frac{\alpha\tau\omega k_1+\gamma\tau k_1k_3}{\tau k_1k_2k_3k_4} & \frac{\alpha}{k_3k_4} & \frac{1}{k_4} & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\tau} \end{bmatrix} \quad (33)$$

The dominant eigenvalue represents the control reproduction number, which is given as;

$$R_C = \frac{1}{2K\tau k_1k_2k_3k_4}(KS^0\tau\beta_1(k_4(k_3(k_2+\rho\eta_1)+\rho\omega\eta_2)+\rho(\alpha\omega+\gamma k_3)\eta_3)+Y \quad (34)$$

where,

Y=

$$\sqrt{KS^0\tau(KS^O\tau\beta_1^2(k_4(k_3(k_2+\rho\eta_1)+\rho\omega\eta_2)+\rho(\alpha\omega+\gamma k_3)\eta_3)^2-4(-1+\epsilon)k_1k_2k_3k_4\beta_2(k_4(k_3(k_2\Psi_1+\rho\Psi_2)+\rho\omega\Psi_3)+\rho(\alpha\omega+\gamma k_3)\Psi_4)}$$

And the control reproduction number for reduced system (1) to (5) is given as;

$$R_C^* = \frac{\beta_1S^0\left(k_2k_3k_4+\eta_1\rho k_3k_4+\eta_2\rho\omega k_4+\eta_3\gamma\rho k_3+\eta_3\alpha\rho\omega\right)}{k_1k_2k_3k_4}$$

3.5 Local stability of disease-free equilibrium point

Theorem 2. The DFE of the system (1) - (6) is locally asymptomatic stable if $R_C < 1$.

Proof. The Jacobian matrix at DFE is obtained and written as:

$$J(E^0)=\begin{bmatrix} -\mu & -S^0\beta_1 & -S^0\beta_1\eta_1 & \theta - S^0\beta_1\eta_2 & -S^0\beta_1\eta_3 & \frac{S^0(1-\epsilon)\beta_2}{K} \\ 0 & S^0\beta_{1-}k_1 & S^0\beta_1\eta_1 & S^0\beta_1\eta_2 & S^0\beta_1\eta_3 & \frac{S^0(1-\epsilon)\beta_2}{K} \\ 0 & \rho & -k_2 & 0 & 0 & 0 \\ 0 & 0 & \omega & -k_3 & 0 & 0 \\ 0 & 0 & \gamma & \alpha & -k_4 & 0 \\ 0 & \Psi_1 & \Psi_2 & \Psi_3 & \Psi_4 & -\tau \end{bmatrix} \quad (35)$$

From Jacobian matrix above, the first eigenvalue is -μ. Therefore, matrix reduces to,

$$\begin{bmatrix} S^0\beta_1 - k_1 & S^0\beta_1\eta_1 & S^0\beta_1\eta_2 & S^0\beta_1\eta_3 & \frac{S^0(1-\epsilon)\beta_2}{K} \\ \rho & -k_2 & 0 & 0 & 0 \\ 0 & \omega & -k_3 & 0 & 0 \\ 0 & \gamma & \alpha & -k_4 & 0 \\ \Psi_1 & \Psi_2 & \Psi & \Psi_4 & -\tau \end{bmatrix} \quad (36)$$

The characteristic polynomial of the above matrix (36) is obtained as;

$$a_1x^5 + a_2x^4 + a_3x^3 + a_4x^2 + a_5x + a_6 = 0 \quad (37)$$

Where constant a_1, a_2, a_3, a_4, a_5 and a_6 are determined using Mathematica software as;

$a_1 = 1 > 0$

$a_2 = \tau + k_1 + k_2 + k_3 + k_4 - S^O\beta_1$

$a_3 = \tau k_1 + \tau k_2 + k_1k_2 + \tau k_3 + k_1k_3 + k_2k_3 + \tau k_4 + k_1k_4 + k_2k_4 + k_3k_4 - S^0\tau\beta_1 -$

$S^0k_2\beta_1 - S^0k_3\beta_1 - S^0k_4\beta_1 - S^0\rho\beta_1\eta_1 - \frac{S^0\beta_2\Psi_1}{K} + \frac{S^0\epsilon\beta_2\Psi_1}{K}$

$a_4 = \tau k_1k_2 + \tau k_1k_3 + \tau k_2k_3 + k_1k_2k_3 + \tau k_1k_4 + \tau k_2k_4 + k_1k_2k_4 + \tau k_3k_4 + k_1k_3k_4 +$

$k_2k_3k_4 - S^0\tau k_2\beta_1 - S^0\tau k_3\beta_1 - S^0k_2k_3\beta_1 - S^0\tau k_4\beta_1 - S^0k_2k_4\beta_1 - S^0k_3k_4\beta_1 - S^0\rho\tau\beta_1\eta_1 -$

$S^0\rho k_3\beta_1\eta_1 - S^0\rho k_4\beta_1\eta_1 - S^0\rho\omega\beta_1\eta_2 - S^0\gamma\rho\beta_1\eta_3 - \frac{S^0k_2\beta_2\Psi_1}{K} + \frac{S^0\epsilon k_2\beta_2\Psi_1}{K} - \frac{S^0k_3\beta_2\Psi_1}{K} +$

$\frac{S^0\epsilon k_3\beta_2\Psi_1}{K} - \frac{S^0k_4\beta_2\Psi_1}{K} + \frac{S^0\epsilon k_4\beta_2\Psi_1}{K} - \frac{S^0\rho\beta_2\Psi_2}{K} + \frac{S^0\epsilon\rho\beta_2\Psi_2}{K}$ (38)

$a_5 = \tau k_1k_2k_3 + \tau k_1k_2k_4 + \tau k_1k_3k_4 + \tau k_2k_3k_4 + k_1k_2k_3k_4 - S^0\tau k_2k_3\beta_1 - S^0\tau k_2k_4\beta_1 -$

$S^0\tau k_3k_4\beta_1 - S^0k_2k_3k_4\beta_1 - S^0\rho\tau k_3\beta_1\eta_1 - S^0\rho\tau k_4\beta_1\eta_1 - S^0\rho k_3k_4\beta_1\eta_1 - S^0\rho\tau\omega\beta_1\eta_2 -$

$S^0\rho\omega k_4\beta_1\eta_2 - S^0\gamma\rho\tau\beta_1\eta_3 - S^0\alpha\rho\omega\beta_1\eta_3 - S^0\gamma\rho k_3\beta_1\eta_3 - \frac{S^0k_2k_3\beta_2\Psi_1}{K} + \frac{S^0\epsilon k_2k_3\beta_2\Psi_1}{K} -$

$\frac{S^0k_2k_4\beta_2\Psi_1}{K}+\frac{S^0\epsilon k_2k_4\beta_2\Psi_1}{K}-\frac{S^0k_3k_4\beta_2\Psi_1}{K}+\frac{S^0\epsilon k_3k_4\beta_2\Psi_1}{K}-\frac{S^0\rho k_3\beta_2\Psi_2}{K}+\frac{S^0\epsilon\rho k_3\beta_2\Psi_2}{K}-\frac{S^0\rho k_4\beta_2\Psi_2}{K}+$

$\frac{S^0\epsilon\rho k_4\beta_2\Psi_2}{K}-\frac{S^0\rho\omega\beta_2\Psi_3}{K}+\frac{S^0\epsilon\rho\omega\beta_2\Psi_3}{K}-\frac{S^0\gamma\rho\beta_2\Psi_4}{K}+\frac{S^0\gamma\epsilon\rho\beta_2\Psi_4}{K}$

$a_6=\tau k_1k_2k_3k_4-S^0\tau k_2k_3k_4\beta_1-S^0\rho\tau k_3k_4\beta_1\eta_1-S^0\rho\tau\omega k_4\beta_1\eta_2-S^0\alpha\rho\tau\omega\beta_1\eta_3-$

$S^0\gamma\rho\tau k_3\beta_1\eta_3-\frac{S^0k_2k_3k_4\beta_2\Psi_1}{K}+\frac{S^0\epsilon k_2k_3k_4\beta_2\Psi_1}{K}-\frac{S^0\rho k_3k_4\beta_2\Psi_2}{K}+\frac{S^0\epsilon\rho k_3k_4\beta_2\Psi_2}{K}-\frac{S^0\rho\omega k_4\beta_2\Psi_3}{K}+$

$\frac{S^0\epsilon\rho\omega k_4\beta_2\Psi_3}{K}-\frac{S^0\alpha\rho\omega\beta_2\Psi_4}{K}+\frac{S^0\alpha\epsilon\rho\omega\beta_2\Psi_4}{K}-\frac{S^0\gamma\rho k_3\beta_2\Psi_4}{K}+\frac{S^0\gamma\epsilon\rho k_3\beta_2\Psi_4}{K}$

By Routh-Hurwitz criteria for stability, for $R_C<1$ the system (1) – (6) is locally asymptomatically stable at disease free equilibrium (B^0) if and only if $a_2>0, a_4>0, a_6>0, b_1>0, c_1>0, d_1>0, e_1>0$ are satisfied and otherwise unstable.

where, $b_1=\frac{a_1a_2-a_3}{a_1}$, $b_2=\frac{a_1a_4-a_5}{a_1}$, $b_3=a_6$, $c_1=\frac{b_1a_3-a_1b_2}{b_1}$, $c_2=\frac{b_1a_5-a_1b_3}{b_1}$,

$d_1=\frac{c_1b_2-b_1c_2}{c_1}$, $d_2=b_3$ and $e_1=\frac{d_1c_2-c_1d_2}{d_1}$

3.6 Global stability of disease-free equilibrium

To obtain the global stability of DFE, Castillo-Chavez method was used.

Theorem 3. Point $\hat{P}=(X^*,0)=(\frac{\pi}{\mu},0,0,0,0)$ is globally asymptomatically stable if and only if $R_C^*<1$ and conditions (T_1) and (T_2) are satisfied in system (1) – (5).

Proof. We can rewrite the reduced system as:

$\frac{dX}{dt}=F(X,Z);\ \frac{dZ}{dt}=G(X,Z).$

Here, $X=(S)$ represents the class of uninfected population while

$Z=(E,I,T,C)$ represents the population which is infected.

$G(X,0)=0, \hat{P}=(X^*,0)=(\frac{\pi}{\mu},0,0,0,0)$ denote the DFE of the system (1) – (5).

In order to generate global asymptotically stability, (T_1) and (T_2) conditions must be satisfied.

(T_1) for $\frac{dX}{dt}=F(X,0), X^*$ is globally asymptotically stable.

(T2), $G(X,Z) = AZ - \hat{G}(X,Z)$, $\hat{G}(X,Z) \geq 0$ for $(X,Z) \in \mathbb{R}^5_+$

Where, $A = D_Z G(X,Z)$ is an M matrix.

In this case, $F(X,0) = \pi - \mu S$

$$A = \begin{bmatrix} \beta_1 S^0 - k_1 & \eta_1 \beta_1 S^0 & \eta_2 \beta_1 S^0 & \eta_3 \beta_1 S^0 \\ \rho & -k_2 & 0 & 0 \\ 0 & \omega & -k_3 & 0 \\ 0 & \gamma & \alpha & -k_4 \end{bmatrix} \tag{39}$$

$$AZ = \begin{bmatrix} (\beta_1 S^0 - k_1)E + \eta_1 \beta_1 S^0 I + \eta_2 \beta_1 S^0 T + \eta_3 \beta_1 S^0 C \\ \rho E - k_2 I \\ \omega I - k_3 T \\ \gamma I + \alpha T - k_4 C \end{bmatrix} \tag{40}$$

$$G(X,Z) = \begin{bmatrix} \lambda_1 S - k_1 E \\ \rho E - k_2 I \\ \omega I - k_3 T \\ \gamma I + \alpha T - k_4 C \end{bmatrix} \tag{41}$$

But $\hat{G}(X,Z) = AZ - G(X,Z)$,

$$\hat{G}(X,Z) = \begin{bmatrix} \hat{G}_1(X,Y) \\ \hat{G}_2(X,Y) \\ \hat{G}_3(X,Y) \\ \hat{G}_4(X,Y) \end{bmatrix} = \begin{bmatrix} \beta_1 (E + \eta_1 I + \eta_2 T + \eta_3 C)(S^0 - S) \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{42}$$

Thus, if $\hat{G}(X,Z) \geq 0$ then DFE is globally asymptomatic stable and unstable otherwise. The susceptible is bounded as, $S \leq S^0$ .Therefore $\hat{G}_1(X,Y) \geq 0$, $\hat{G}_2(X,Y) = 0$, $\hat{G}_3(X,Y) = 0$ and $\hat{G}_4(X,Y) = 0$. Thus DFE, is globally asymptomatically stable when $R_C^* < 1$

3.7 Existence of Endemic Equilibrium Point (EEP)

$B^* = (S^*, E^*, I^*, T^*, C^*)$ denote the EEP of system (1) – (5) and when the disease persists, it occurs in the community [14]. B^* is evaluated by equating the system to zero. To get,

$$S^* = \frac{\pi + \theta T^*}{\mu + \lambda_1^*}, \quad E^* = \frac{\lambda_1^* S^*}{k_1}, \quad I^* = \frac{\rho E^*}{k_2}$$

$$T^* = \frac{\omega I^*}{k_3}, \quad C^* = \frac{\gamma I^* + \alpha T^*}{k_4}$$

Where, $k_1 = \mu + \rho$, $k_2 = \mu + \omega + \gamma$, $k_3 = \mu + \alpha + \theta$ and $k_4 = \mu + \delta$

Lemma 1. For $R_C^* > 1$ there exists a unique EEP B^* and otherwise does not exist.

Proof. For the endemic disease $\frac{dE}{dt} > 0$, $\frac{dI}{dt} > 0$, $\frac{dT}{dt} > 0$ and $\frac{dC}{dt} > 0$ that is

$$\frac{dE}{dt} = \lambda_1 S - (\mu + \rho)E > 0 \tag{43}$$

$$\frac{dI}{dt} = \rho E - (\mu + \omega + \gamma)I > 0 \tag{44}$$

$$\frac{dT}{dt} = \omega I - (\alpha + \mu + \theta)T > 0 \tag{45}$$

$$\frac{dC}{dt} = \gamma I + \alpha T - (\mu + \delta)C > 0 \tag{46}$$

From inequalities of (43) – (46),

$$E < \frac{\beta_1 \left(E + \eta_1 I + \eta_2 T + \eta_3 C\right) S^0}{k_1} \tag{47}$$

$$I < \frac{\rho E}{k_2} \tag{48}$$

$$T < \frac{\rho \omega E}{k_2 k_3} \tag{49}$$

$$C < \frac{\gamma \rho E}{k_2 k_4} + \frac{\alpha \rho \omega E}{k_2 k_3 k_4} \tag{50}$$

Where, $k_1 = \mu + \rho$, $k_2 = \mu + \omega + \gamma$, $k_3 = \mu + \alpha + \theta$ and $k_4 = \mu + \delta$

Substituting (48), (49) and (50) to (47) we obtained,

$$E < \frac{\beta_1 S^0}{k_1}\left(E + \eta_1 \frac{\rho E}{k_2} + \eta_2 \frac{\rho \omega E}{k_2 k_3} + \eta_3 \left(\frac{\gamma \rho E}{k_2 k_4} + \frac{\alpha \rho \omega E}{k_2 k_3 k_4}\right)\right) \tag{51}$$

By cancelling the E on both sides, we obtained

$$1 \le \frac{\beta_1 S^0 \left(k_2 k_3 k_4 + \eta_1 \rho k_3 k_4 + \eta_2 \rho \omega k_4 + \eta_3 \gamma \rho k_3 + \eta_3 \alpha \rho \omega\right)}{k_1 k_2 k_3 k_4} = R_C^{\,*} \tag{52}$$

$$R_C^{\,*} > 1$$

Therefore, there is unique endemic equilibrium existence when $R_C^{\,*}$ is greater than unity.

3.8 Global stability of endemic equilibrium

Theorem 4**.** Given $R_C > 1$ then, EEP B^* of the system (1) – (6) is globally asymptomatically stable.

Proof. Lyapunov function L is defined as;

$$L = \left(S - S^* + S^* ln\frac{S^*}{S}\right) + \left(E - E^* + E^* ln\frac{E^*}{E}\right) + \left(I - I^* + I^* ln\frac{I^*}{I}\right) + \left(T - T^* + T^* ln\frac{T^*}{T}\right) + \left(C - C^* + C^* ln\frac{C^*}{C}\right) + \left(H - H^* + H^* ln\frac{H^*}{H}\right) \tag{53}$$

The derivative of (53) is taken with respect to t, which gives

$$\frac{dL}{dt} = \left(\frac{S - S^*}{S}\right)\frac{dS}{dt} + \left(\frac{E - E^*}{E}\right)\frac{dE}{dt} + \left(\frac{I - I^*}{I}\right)\frac{dI}{dt} + \left(\frac{T - T^*}{T}\right)\frac{dT}{dt} + \left(\frac{C - C^*}{C}\right)\frac{dC}{dt} + \left(\frac{H - H^*}{H}\right)\frac{dH}{dt} \tag{54}$$

By replacing

$$\frac{dS}{dt}, \frac{dE}{dt}, \frac{dI}{dt}, \frac{dT}{dt}, \frac{dC}{dt} and \frac{dH}{dt}$$

into (54) and putting all positive and negative terms together we have,

$$\frac{dL}{dt} = Q - W$$

Where,

$$Q = \pi + \theta T + \frac{\theta T^* S^*}{S} + \left(\lambda_1 + \lambda_2\right) S + \frac{\left(\lambda_1 + \lambda_2\right) S^* E^*}{E} + \rho E + \frac{\rho E^* I^*}{I} + \omega I + \frac{\omega I^* T^*}{T} + \gamma I + \alpha T + \frac{\gamma I^* C^*}{C} + \frac{\alpha T^* C^*}{C} + \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C + \frac{\psi_1 E^* H^*}{H} + \frac{\psi_2 I^* H^*}{H} + \frac{\psi_3 T^* H^*}{H} + \frac{\psi_4 C^* H^*}{H}$$

$$W = \theta T^* + \frac{\pi S^*}{S} + \frac{\theta T S^*}{S} + (\lambda_1 + \lambda_2) S^* + \frac{(\lambda_1 + \lambda_2) S E^*}{E} + \rho E^* + \frac{\rho E I^*}{I} + \omega I^* + \frac{\omega I T^*}{T} \gamma I^* + \alpha T^* + \frac{\gamma I C^*}{C} + \frac{\alpha T C^*}{C} + \psi_1 E^* + \psi_2 I^* + \psi_3 T^* + \psi_4 C^* + \frac{\psi_1 E H^*}{H} + \frac{\psi_2 I H^*}{H} + \frac{\psi_3 T H^*}{H} + \frac{\psi_4 C H^*}{H} + \frac{(S - S^*)^2}{S}[\mu + \lambda_1 + \lambda_2] + \frac{(E - E^*)^2}{E}[\mu + \rho] + \frac{(I - I^*)^2}{I}[\mu + \omega + \gamma] + \frac{(T - T^*)^2}{T}[\alpha + \mu + \theta] + \frac{(C - C^*)^2}{C}[\alpha + \delta] + \frac{\tau (H - H^*)^2}{H} \tag{55}$$

If Q<W, then $\frac{dL}{dt} \leq 0$;

Note that, $\frac{dL}{dt} = 0$ if and only if $S = S^*, E = E^*, I = I^*, T = T^*, C = C^*, H = H^*$ Thus, the largest compact invariant set in $\{(S^*, E^*, I^*, T^*, C^*, H^*) \in \mathbb{R}^6_+ : \frac{dL}{dt} = 0\}$ is the singleton B^*, where B^* is the EEP of the system (1) – (6).

By LaSalle's invariant principle, it shows that B^* is globally asymptomatically stable in $\mathbb{R}^6_+$ if Q<W.

3.9 Bifurcation analysis

The possibility of bifurcation in human population was studied using center manifold theory. For simplicity the renaming of variables is made by letting;

$S = x_1$, $E = x_2$, $I = x_3$, $T = x_4$, $C = x_5$

Using vector notation:

$x = (x_1, x_2, x_3, x_4, x_5)^T$. The system (1) – (5) is written as,

$$\frac{dx}{dt} = F(x)$$

Where, $F = (f_1, f_2, f_3, f_4, f_5)^T$ it follows that;

$$\frac{dx_1}{dt} = f_1 = \pi + \theta x_4 - (\mu + \lambda_1) x_1 \tag{56}$$

$$\frac{dx_2}{dt} = f_2 = \lambda_1 x_1 - k_1 x_2 \tag{57}$$

$$\frac{dx_3}{dt} = f_3 = \rho x_2 - k_2 x_3 \tag{58}$$

$$\frac{dx_4}{dt} = f_4 = \omega x_3 - k_3 x_4 \tag{59}$$

$$\frac{dx_5}{dt} = f_5 = \gamma x_3 + \alpha x_4 - k_4 x_5 \tag{60}$$

With $\lambda_1 = \beta_1 \left(x_2 + \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5 \right)$

Jacobian solved at DFE,

$B^0 = (S^0, E^0, I^0, T^0, C^0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0 \right)$. Gives

$$J(B^0) = \begin{bmatrix} -\mu & -\beta_1 S^0 & -\beta_1 \eta_1 S^0 & \theta - \beta_1 \eta_2 S^0 & -\beta_1 \eta_3 S^0 \\ 0 & \beta_1 S^0 - k_1 & \beta_1 \eta_1 S^0 & \beta_1 \eta_2 S^0 & \beta_1 \eta_3 S^0 \\ 0 & \rho & -k_2 & 0 & 0 \\ 0 & 0 & \omega & -k_3 & 0 \\ 0 & 0 & \gamma & \alpha & -k_4 \end{bmatrix} \tag{61}$$

Consider the case, where $R_C^* = 1$. Further, suppose that $\beta_1 = \beta_1^*$ is a bifurcation parameter. Solving for β_1^* from $R_C^* = 1$ we get,

$$\beta_1^* = \frac{k_1 k_2 k_3 k_4}{S^0 (k_2 k_3 k_4 + \rho k_3 k_4 \eta_1 + \rho \omega k_4 \eta_2 + \alpha \rho \omega \eta_3 + \gamma \rho k_3 \eta_3)} \tag{62}$$

By use of Mathematica software, Jacobian of the system $\frac{dx}{dt} = F(x)$ of DFE with $\beta_1 = \beta_1^*$ has one of the eigenvalues as simple zero. Thus, the system dynamics near $\beta_1 = \beta_1^*$ was analysed by theory of center manifold.

Jacobian matrix near $\beta_1 = \beta_1^*$, has a right eigenvector linked with zero eigenvalue written as $w = \left(w_1, w_2, w_3, w_4, w_5 \right)^T$ from the system.

Solving the system of the equations we obtained

$$w_1 = \frac{-\left(\beta_1 S^0 w_2 + \beta_1 \eta_1 S^0 w_3 + (\beta_1 \eta_2 S^0 - \theta) w_4 + \beta_1 \eta_3 S^0 w_5\right)}{\mu}$$

$$w_2 = w_2 > 0, \quad w_3 = \frac{\rho w_2}{k_2} > 0, \quad w_4 = \frac{\omega w_3}{k_3} > 0 \tag{63}$$

$$w_5 = \frac{\rho w_3 + \alpha w_4}{k_4} > 0$$

The left eigenvector at $\beta_1 = \beta_1^*$ associated with zero eigenvalues is given by $v = (v_1, v_2, v_3, v_4, v_5)^T$ from the system.

Solving the system of the equations we have;

$$v_1 = 0, \quad v_2 = v_2 > 0, \quad v_3 = \frac{\beta_1 \eta_1 S^0 v_2 + \omega v_4 + \gamma v_5}{k_2} > 0$$

$$v_4 = \frac{\beta_1 \eta_2 S^0 v_2 + \alpha v_5}{k_3} > 0, \quad v_5 = \frac{\beta_1 \eta_3 S^0 v_2}{k_4} > 0 \tag{64}$$

From the system the computation of a and b can be shown as:

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \frac{\partial^2 f_2}{\partial x_2 \partial x_1} = \beta_1, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_3} = \frac{\partial^2 f_2}{\partial x_3 \partial x_1} = \beta_1 \eta_1 \tag{65}$$

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = \beta_1 \eta_2, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_5} = \frac{\partial^2 f_2}{\partial x_5 \partial x_1} = \beta_1 \eta_3$$

$$a = 2 v_2 \left\{ w_1 w_2 \beta_1 + w_1 w_3 \beta_1 \eta_1 + w_1 w_4 \beta_1 \eta_2 + w_1 w_5 \beta_1 \eta_3 \right\} < 0 \tag{66}$$

Also, computation of b can be shown that;

$$\frac{\partial^2 f_2}{\partial x_1 \partial \beta_1} = x_2 + \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5, \qquad \frac{\partial^2 f_2}{\partial x_2 \partial \beta_1} = x_1, \qquad \frac{\partial^2 f_2}{\partial x_3 \partial \beta_1} = \eta_1 x_1$$

$$\frac{\partial^2 f_2}{\partial x_4 \partial \beta_1} = \eta_2 x_1, \quad \frac{\partial^2 f_2}{\partial x_5 \partial \beta_1} = \eta_3 x_1 \tag{67}$$

$$b = v_2 \left\{ w_1 (x_2 + \eta_1 x_3 + \eta_2 x_4 + \eta_3 x_5) + w_2 x_1 + w_3 \eta_1 x_1 + w_4 \eta_2 x_1 + w_5 \eta_3 x_1 \right\} > 0 \tag{68}$$

At $R_C^* = 1$ bifurcation of system (1) – (5) has a forward direction.

4. SENSITIVITY ANALYSIS OF THE PARAMETERS

Sensitivity analysis was done by the technique identified in based on control reproduction number of some given parameters on reduced system (1) – (5), let say ρ given as:

$$\Lambda_{\rho}^{R_C^*} = \frac{\partial R_C^*}{\partial \rho} x \frac{\rho}{R_C^*} \tag{69}$$

Table 2: Table of sensitivity indices

Parameter	Sensitivity indices
β_1	1
η_1	0.524301
η_2	0.252245
η_3	0.206807
ω	-0.311424
γ	-0.211066
δ	-0.20634
α	-0.146564
θ	-0.104878
ρ	-0.0166177
μ	-0.00310901

The sensitivity indices on control reproduction number with positive parameters $(\beta_1, \eta_1, \eta_2, \eta_3)$ indicate a vital role in expansion of the infections in the community when their values are increased. This is because increase in their values increases the control reproduction number. While negative sensitivity indices of given parameters $(\delta, \mu, \omega, \rho, \gamma, \alpha, \theta)$ have a significance in lowering the burden of infection in the community given that their values are increased as the rest remain fixed. This is due to reason that increase in their value will lead to decrease in the control reproduction number, resulting to decrease in the endemicity of the infections in the community.

5. PARTIAL RANK CORRELATION COEFFICIENT

The PRCC is an efficient tool which is usually used in uncertainity analysis with aim of exploring the parameter space of the model entirely. The strength of inputs and output of the model can be measured using PRCC through LHS method. The main aim of LHS/PRCC analysis is to identify parameters which are more important in uncertainity contributions to rank them by their significance and prediction to this imprecision [15] . The PRCC always ranges from -1 to +1 and its absolute value describe how the relationship between x and y variable is strong. If the value is close to -1 or +1 then the linear relationship is stronger between x and y and if the value is far much from -1 or +1 then the weaker relationship between x and y. From Figure 2, the two values on the top [x, y] in each plot, represents the PRCC and p-value respectively. The parameters with large PRCC value and corresponding small p-value are more significant in the model. For easier analysis, the result obtained from figure 2, are summarized in table 3. Moreso, the PRCC values for all model parameters are represented in the diagram shown in figure 3. Therefore, the most significant parameters are given as follows; birth rate, π. Natural mortality rate of individuals, μ. Environment to human force of infection, λ_2. Infectious rate of exposed individuals, ρ and treatment rate, ω. The five parameters $\pi, \mu, \lambda_2, \rho, \omega$ in the model will be significant contributors to uncertainity. From table 3, we note that highly correlated parameters are $\pi, \mu, \lambda_2, \rho$ with corresponding values 0.99629, -0.88649, 0.87202 and -0.86981 respectively compared to ω with PRCC value of 0.55332. We can conclude that parameters $\pi, \mu, \lambda_2, \rho$ are most influential in the model.

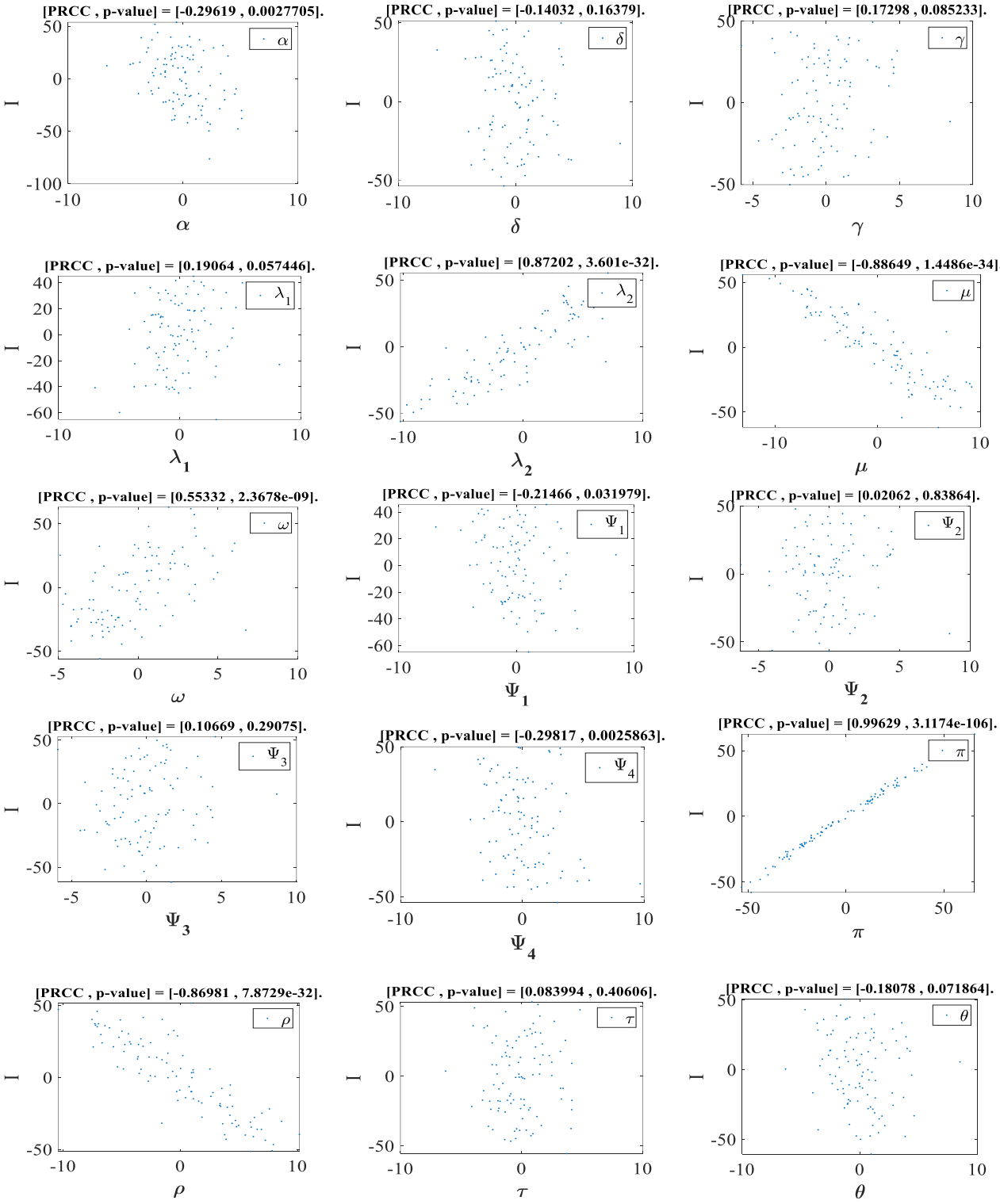

Figure 2. The PRCC plot for Objective Functional

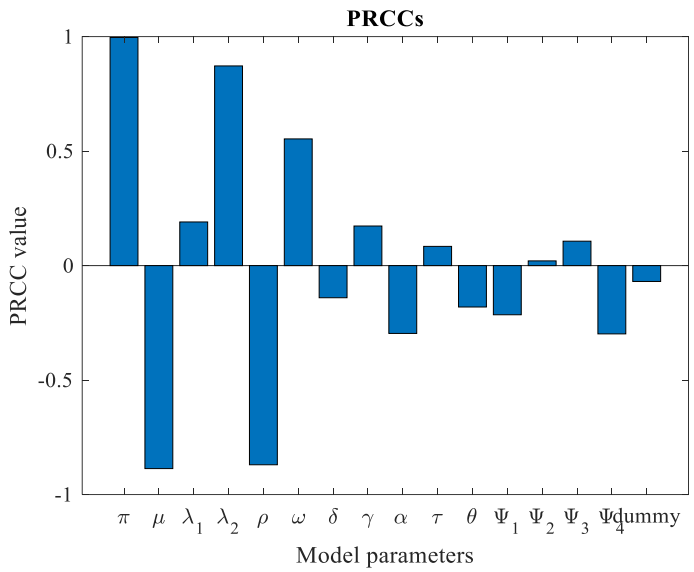

Figure 3. The PRCC diagram for model parameters

Table 3. Output from PRCC Analysis

Parameter symbol	**Objective functional**	
	PRCC	**p-value**
π	0.99629	3.1174e-106
μ	-0.88649	1.4486e-34
λ_1	0.19064	0.057446
λ_2	0.87202	3.601e-32
ρ	-0.86981	7.8729e-32
ω	0.55332	2.3678e-09
θ	-0.18078	0.071864
δ	-0.14032	0.16379
γ	0.17298	0.085233
α	-0.29619	0.0027705
τ	0.083994	0.40606
ψ_1	-0.21466	0.031979
ψ_2	0.02062	0.83864
ψ_3	0.10669	0.29075
ψ_4	-0.29817	0.0025863

6. NUMERICAL SIMULATIONS

MATLABR2019a was used in numerical simulation to demonstrate the dynamical behaviour of non-linear ODE in the system (1) – (6). The simulations were carried out using initial conditions and parameters values in Table 4 which were derived from the literature review and graphically presented.

Table 4: Parameter and values of the model

Parameter	Value	Source
π	10	Assumed
μ	0.000019	[10]
λ_1	0.02	Assumed
λ_2	0.1075	Estimated
ρ	0.65	[10]
ω	0.0073	[16]
θ	0.002485	Estimated
δ	0.00839	[17]
γ	0.003	[11]
α	0.0066	[16]
τ	0.001	[18]
ψ_1	0.0009	Assumed
ψ_2	0.0008	Assumed
ψ_3	0.0004	Assumed
ψ_4	0.0001	Assumed

6.1 Numerical simulation of the full model

In Figure 4, displays the trend of the model as all parameter values of the model remains unchanged. The number of exposed populations decreases with time due to use of clean water, hygiene which leads to low infection rate. The infectious class I(t) increases exponentially and then decreases gradually until stationary point is attained. The number of treated humans increases slowly with time up to a certain point until the steady state is established and also, we can see that concentration of the bacteria increases very slowly with time up to a maximum point where equilibrium point is attained.

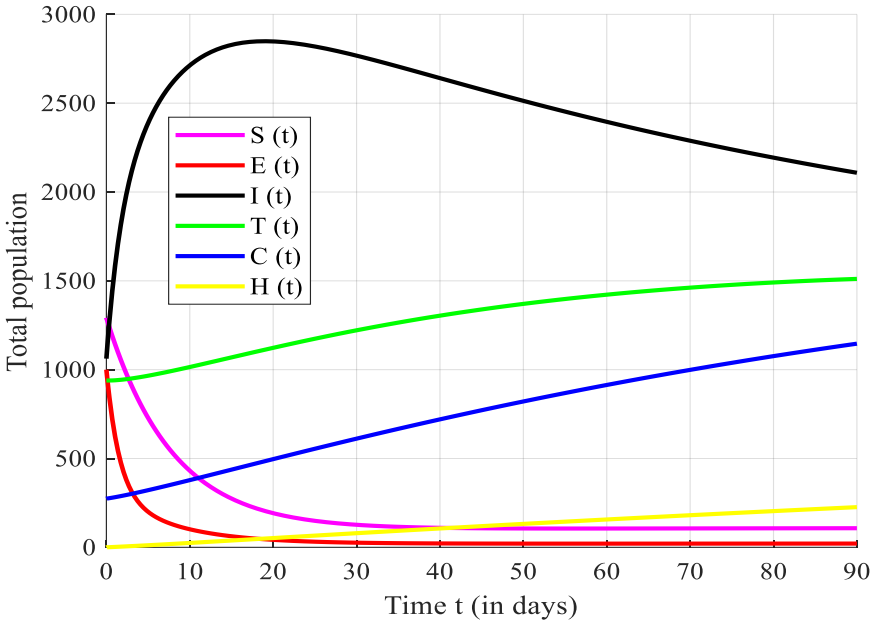

Figure 4. Numerical simulation of the full model

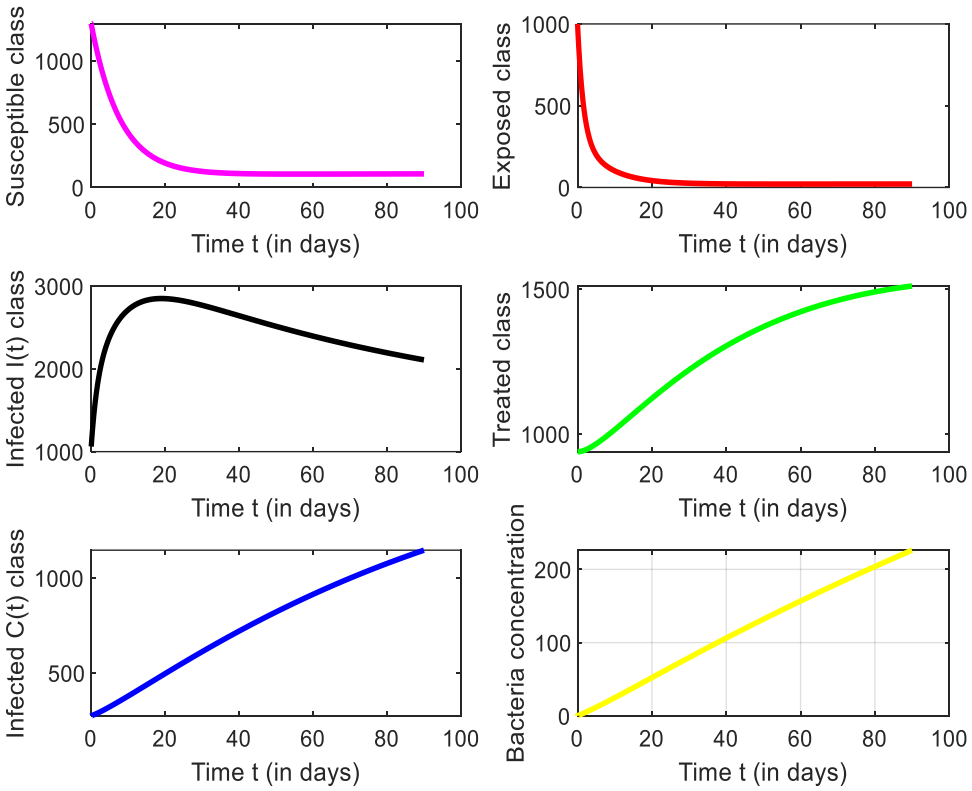

Figure 5. Shows subplots for the six sub classes from 0 to 90 days

6.2 Effect of exposed class on the susceptible class

From Figure 6, initially the number of exposed populations increases slowly with increase in the susceptible population due to low infection rate and then rapidly increases to the maximum point due to higher infection rate.

6.3 Effect of infected class with *H. pylori* on the exposed class

Initially in Figure 7, the population of infected individuals with *H. pylori* increases rapidly up to a certain point and then increases slowly before reaching a maximum value and finally decreases rapidly with decrease in exposed population.

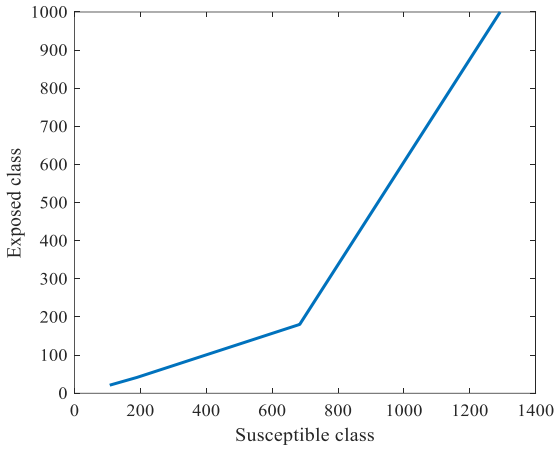

Figure 6. Exposed and Susceptible Classes

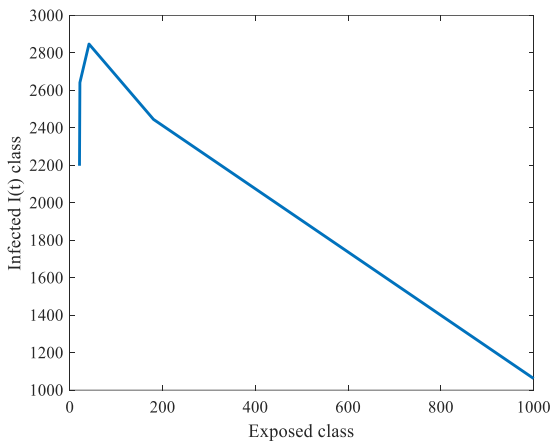

Figure 7. Infected I(t) and Exposed Classes

6.4 Effect of treatment rate on *Helicobacter pylori* infected population

Figure 8, indicate the impact of treatment rate (ω) on the infected populations with *H. pylori* which was investigated using different values of ω while all other parameters remained constant. The result shows that increase in the treatment rate can result to large number of infected individuals with *H. pylori*. From the graph also reveals that, increase in treatment rate results to decrease in *H. pylori* infectious population. Thus, we can conclude that high treatment rate contributes positively to the community by reducing the number of *H. pylori* infections.

6.5 Effect of treatment rate on treated population

The treatment rate was investigated in figure 9, on the number of treated populations by varying treatment rate from ω=0 to ω=0.115 and the other parameters were kept constant. We can see from the graph that; the number of treated humans increases if the treatment rate is increased. However, we can conclude that the population of the treated widens by increasing the treatment rate.

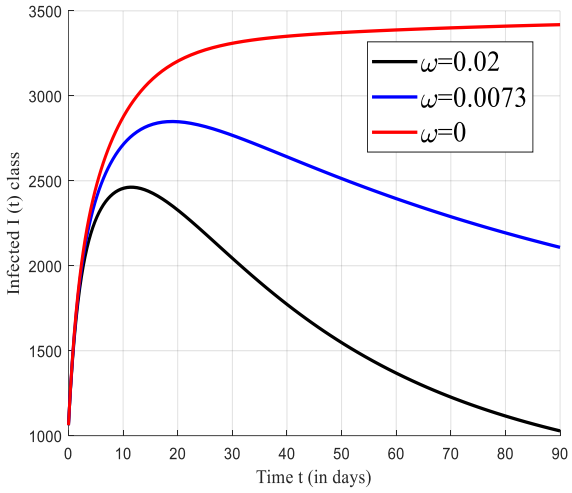

Figure 8. Variation of treatment rate on infected humans with *H. pylori*

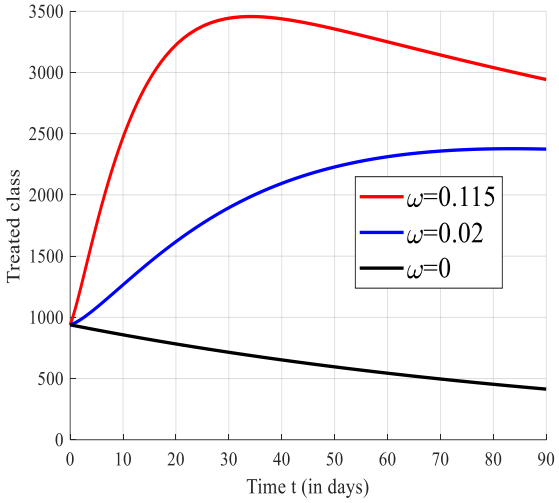

Figure 9. Variation of treatment rate on individuals who are treated

6.5 Effect of contact rate (γ) on the Stomach cancer population.

The contact rate γ was varied from γ =0 to γ =0.02 and all other parameters remain fixed. From Figure 10, we obtained that the contact rate increases as number of stomach cancer increases in the community, which indicate that if the infected individuals with *H. pylori* fails to get treatment this would increase the chances of acquiring stomach cancer. We can conclude that, when the contact rate (γ) increases the disease will persists in the community even if all other parameters remain constant.

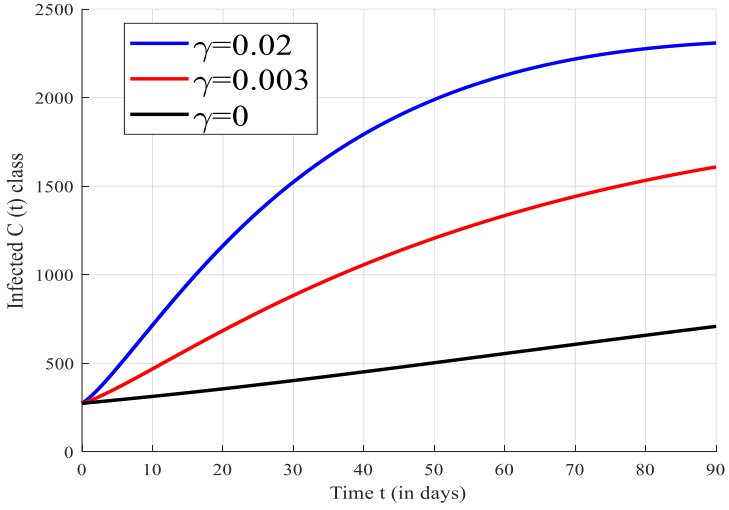

Figure 10. Variation of contact rate (γ) on infected individuals with stomach cancer

7. CONCLUSION

In this study, the qualitative behaviour of the deterministic model was analyzed by obtaining the positivity and boundness of the solution, equilibrium points, control reproduction number was studied by next generation matrix method, local stability of DFE was obtained by Routh-Hurwitz criteria for stability and global stability was also obtained by use of Castillo-Chavez method, global stability of EEP was carried out by a Lyapunov function and bifurcation analysis were also analyzed. From the analysis we investigated that, the model is bounded and lies on the positive region; local stability of DFE is LAS if $R_C < 1$ and global stability of EEP is asymptomatic stable when $R_C > 1$. On the other hand, some of the analyses were carried out using reduced system that is system (1) – (5). From this, we observed that global stability of DFE is asymptotically stable given that $R_C^* < 1$ and possibility of forward bifurcation was noted.

Moreover, sensitivity analysis of system (1) – (5) was also performed based on control reproduction number. It was observed that, increasing the value of β_1, η_1, η_2 and η_3 have a vital role in expanding the infection in the community while increasing parameters $(\delta, \mu, \omega, \rho, \gamma, \alpha, \theta)$ have a high impact in decreasing the burden of disease.

To identify the highly sensitive parameter in the model, the sensitivity analysis was performed based on PRCC method and LHS. From the PRCC analysis, we can conclude that out of the fifteen parameters which were considered in the PRCC process, parameters $\pi, \mu, \lambda_2, \rho$ and ω are highly sensitive in the model. Therefore, the five parameters will be a vital contributor to uncertainity in the model. Identifying these parameters as most sensitive can assist us to incorporate new treatment measures in this study area.

Finally, we have performed the numerical simulation which outlines that the population of the treated individuals widens by increasing the treatment rate. Therefore, this paper shows the best alternative way of curbing the stomach cancer is by reducing the contact rate, effective hygiene and improving the treatment rate of *Helicobacter pylori* infection.

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CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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