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On the Mathematical Analysis of a Campylobacteriosis Model for Human and Animal Population

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Abstract

Campylobacteriosis, a major cause of foodborne illnesses has continued to claim millions of lives globally. Thus, concerted needs to be put in place in order to curtail further loss of lives due to the disease. In this study, an analysis is conducted on the reproduction number, sensitivity analysis is also carried out on the parameters of the model connected to the reproduction number and the possibility of backward bifurcation of the campylobacteriosis mathematical model is explored. Results from the sensitivity analysis show that the most sensitive parameters are the infection rates, progression rate and the treatment rate for humans. It is further observed that as the human recruitment rate and treatment rate for symptomatic human population increases, the reproduction number also increases. This implies that it will not be possible to eliminate campylobacteriosis in the community with at least 70% treatment rate administered to the symptomatic human population. The analysis of the campylobacteriosis model reveals that the model exhibits the phenomenon of backward bifurcation under certain conditions, where a stable disease-free equilibrium (DFE) coexists with a stable endemic equilibrium (EE) when the associated reproduction number (R_C) is less than unity. It is further shown for a special case that, a unique endemic equilibrium exists whenever the associated reproduction number is greater than unity.

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1.0 Introduction

It was the recommendation of an earlier paper by Ashezua *et al*. (2024) on the population dynamics of a mathematical model for campylobacteriosis that prompted further analysis of the model in the present study. Campylobacteriosis, a major cause of food borne, is an infection triggered by the bacterium campylobacter and it is considered as the most common signature of diarrhoeal diseases (WHO, 2020). According to the World Health Organization, it is estimated that 1 in every 10 persons fall ill and about 33 million of healthy lives are lost due to the burden of food borne diseases. Diarrhoeal diseases are the most common illnesses emanating from unsafe food, with over 550 million people falling ill yearly (including 220 million children under the age of 5 years) (WHO, 2020). Campylobacter infections are generally mild (asymptomatic), but can be fatal (symptomatic) among very young children, elderly, and immunosuppressed individuals (WHO, 2020, Health direct, 2024).

Most people with campylobacter infection often have diarrhea (usually bloody), fever, and stomach cramps. Nausea and vomiting may follow the diarrhea. Symptoms usually start as from two to five days after infection and last for about one week. Complications such as irritable bowel syndrome, temporary paralysis and arthritis are experienced by some people (CDC,

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2023). According to the Centers for Disease Control and Prevention, most people recover from campylobacter infection without antibiotic treatment (patients are advised to drink enough fluids as long as the diarrhea last) while severe cases might need antibiotics treatment. The elderly (65 years and above), pregnant women and people with compromised immune system are the category of people who are likely to contract severe cases of the disease (CDC, 2019). The disease is mostly foodborne and waterborne but can also spread through direct contact with infected humans or animals through fecal-oral path of transmission. Human-to-human spread is usually not common (Havelaar *et al.* 2009).

Many mathematical modelling studies have been conducted to help understand the transmission dynamics, prevention and control of campylobacteriosis. For example, Solow *et al*. (2003) studied the effect of temperature on viability of campylobacter jejuni and campylobacter coli on raw chicken or pork skin where campylobacter was inoculated on pieces of raw, irradiated chicken or pork skin and exposed to temperature ranging from -20 to 42°C under microaerobic or aerobic conditions. The analysis carried out in their work were not qualitative in enough nature. Osman *et al.* (2020) formulated and analyzed a deterministic mathematical model for campylobacteriosis as a zoonotic disease with optimal control. They used the nonstandard finite difference scheme for the model analysis. Further, Chuma and Mussa (2021) designed and analyzed a campylobacteriosis transmission dynamics in human: Modelling the effects of public health education, treatment and sanitation. In their work, qualitative analysis of the model was done. However, the analysis of the reproduction number and the type of bifurcation their model exhibit was not explored.

Recently, Ashezua *et al*. (2024) developed and analyzed a mathematical model for campylobacteriosis population dynamics. In their work, they computed the reproduction number of the model, obtained conditions for the local and global stability of the disease-free equilibrium and equally performed numerical simulations of their model. They however, recommended that future work should focus on conducting sensitivity analysis of the model parameters associated with the reproduction number and also theoretically determine the type of bifurcation their model is likely to exhibit. This indeed, prompted further analysis of the model developed in Ashezua *et al*. (2024).

The goal of this paper (in addition to the recommendation made in the work of Ashezua *et al.* 2024) is to analyze the reproduction number, conduct sensitivity analysis using the parameters of model (4) associated with the reproduction number, R_c and to categorize the type of bifurcation model (4) is likely to exhibit.

The rest of the paper is organized as follows: The campylobacteriosis model developed in Ashezua *et al.* (2024) and the reproduction number obtained are reproduced in Section 2. Analysis of the reproduction number, sensitivity and backward bifurcation analysis are presented in Section 3. The paper is concluded in Section 4.

2. 0 Materials and Methods

In this section, the model developed by Ashezua *et al.* (2024) is reproduced here and described for convenience of flow in the next section.

2.1 Model Description

Under this sub-heading, the model variables and parameters in Ashezua *et al.* (2024) are adopted. In their work, the total population at time t, denoted by $N(t)$, is divided into the human $(N_h(t))$ and animal $(N_v(t))$ population. The total human population is further sub-divided into the five mutually-exclusive compartments of the susceptible $(S_h(t))$, exposed $(E_h(t))$, asymptomatic $(I_a(t))$, symptomatic $(I_s(t))$ and recovered $(R_h(t))$ humans. Similarly, the total animal population is sub-divided into the susceptible $(S_v(t))$, infected $(I_v(t))$ and recovered $(R_v(t))$ sub-population as shown in equation (1). Thus,

$$\begin{aligned} N(t) &= N_h(t) + N_v(t) \\ N_h(t) &= S_h(t) + E_h(t) + I_a(t) + I_s(t) + R_h(t) \quad (\mathbf{1}) \\ N_v(t) &= S_v(t) + I_v(t) + R_v(t) \end{aligned}$$

The susceptible population (for both human and animals) are recruited through immigration at rates Λ_h and Λ_v, respectively. They are infected with campylobacteriosis through ingestion of contaminated water, foods, and direct contact with infected human and animals at a rate $\beta_1\lambda$. Humans in E_h class progresses to classes I_a and I_s at rate θ while ρ is the proportion of humans that progressed to class (I_a). The humans in classes I_a and I_s recover from campylobacteriosis at rates γ_1 and γ_2, respectively. Furthermore, natural death rate $\mu_h(\mu_v)$ occurs in all the epidemiological classes of human (animal) population while humans (animals) in classes I_s and I_v suffer an additional campylobacteriosis induced death at a rate $\delta_h(\delta_v)$, respectively. Susceptible humans (animals) acquire campylobacteriosis infection through ingestion of contaminated water, foods, and direct contact with infected animals and humans (i.e. those in the I_a, I_s and I_v classes), at a rate $\beta_1\lambda$ and $\beta_2\lambda$, respectively, given by equations (2) and (3) below

$\beta_1(I_a + I_s + I_v)$, (**2**)

and

$\beta_2(I_a + I_s + I_v)$. (**3**)

Animals recover from campylobacteriosis at a rate γ_3. The variables and parameters of the model are tabulated in Table 1.

Table 1: Description of parameters in the campylobacteriosis model (4).

Variables/parameters	Interpretation
S_h	Susceptible human population
E_h	Exposed human population
I_a	Asymptomatic human population
I_s	Symptomatic human population
R_h	Recovered human population
S_v	Susceptible animal population
I_v	Infected animal population
R_v	Recovered animal population
$\Lambda_h(\Lambda_v)$	Recruitment rates for human (animal)
$\beta_1(\beta_2)$	Infection rates for human (animal)
θ	Progression rate
ρ	Proportion of the exposed humans moving to class I_a
η	Progression rate from I_a to I_s
$\gamma_1(\gamma_2)$	Treatment rates for humans for $I_a(I_s)$ individuals
$\mu_h(\mu_v)$	Human (animal) natural death rates
$\psi_h(\psi_v)$	Loss of immunity for human (animal)
γ_3	Treatment rate for animal
$\delta_h(\delta_v)$	Human (animal) disease-induced death rate
λ	Force of infection

$$
\begin{aligned}
\frac{dS_h(t)}{dt} &= \Lambda_h - \beta_1\lambda S_h - \mu_h S_h + \psi_h R_h,\\
\frac{dE_h(t)}{dt} &= \beta_1\lambda S_h - [\theta\rho + \theta(1-\rho) + \mu_h]E_h,\\
\frac{dI_a(t)}{dt} &= \theta\rho E_h - (\eta + \gamma_1 + \mu_h)I_a,\\
\frac{dI_s(t)}{dt} &= \theta(1-\rho)E_h + \eta I_a - (\gamma_2 + \mu_h + \delta_h)I_s,\\
\frac{dR_h(t)}{dt} &= \gamma_1 I_a + \gamma_2 I_s - (\mu_h + \psi_h)R_h,\\
\frac{dS_v(t)}{dt} &= \Lambda_v - \beta_2\lambda S_v - \mu_v S_v + \psi_v R_v,\\
\frac{dI_v(t)}{dt} &= \beta_2\lambda S_v - (\gamma_3 + \mu_v + \delta_v)I_v,\\
\frac{dR_v(t)}{dt} &= \gamma_3 I_v - (\mu_v + \psi_v)R_v.
\end{aligned}
\tag{4}
$$

where the forces of infection for human and animals are as given in equations (2) and (3), respectively. The result below holds for model (4).

Theorem 2.1. All solutions of the model (4) with positive initial data remain positive for all time $t > 0$. Furthermore, the model is a dynamical system on the region $\Omega = \Omega_1 \cup \Omega_2 \subset \mathbb{R}_+^5 \times \mathbb{R}_+^3$ with,

$$\Omega_1 = \left\{(S_h, E_h, I_a, I_s, R_h): N_h \leq \frac{\Lambda_h}{\mu_h}\right\}$$

$$\Omega_2 = \left\{(S_v, I_v, R_v): N_A \leq \frac{\Lambda_v}{\mu_v}\right\}$$

with,

$$S_h + E_h + I_a + I_s + R_h = N_h$$

and

$S_v + I_v + R_v = N_A$.

and the region Ω is attracting with respect to the model (4) with initial conditions in $\mathbb{R}^8_+$.

Proof. Following similar approach as in Gumel *et al.* (2018), it is easy to see that the equations for human (susceptible individuals) and animal (susceptible animals) in model (4) leads to the following first-order inequality equations: $\frac{dS_h}{dt} + (\beta_1\lambda + \mu_h)S_h > 0$, and $\frac{dS_v}{dt} + (\beta_2\lambda + \mu_v)S_v > 0.\alpha_{S_h}(t) = \exp^{\int [\beta_1\lambda(\tau)+\mu_h]d\tau}, \alpha_{S_v}(t) = \exp^{\int [\beta_2\lambda(\tau)+\mu_v]d\tau}, (\mathbf{5})$

and observing that

$$\alpha_{S_h}(t)\left[\frac{dS_h}{dt} + (\beta_1\lambda + \mu_h)S_h\right] = \frac{dS_h\alpha_{S_h}}{dt}$$
$$\alpha_{S_v}(t)\left[\frac{dS_v}{dt} + (\beta_2\lambda + \mu_v)S_A\right] = \frac{dS_v\alpha_{S_v}}{dt}$$

Then, integrating with respect to time from 0 to t gives $S_h(t) \geq 0$ and $S_v(t) \geq 0$ at all times, respectively. However, this direct approach does not apply to the remaining equations. In any case, having the nonnegativity of S_h and S_v in mind, it can be shown that the remaining six equations of the model (4) form a monotone system. Consequently, all its solutions corresponding to positive initial data remain positive at all times $t \geq 0$. By adding the first five and the last three equations of model (4), the following conservation law is obtained.

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_s,$$
$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v - \delta_\alpha I_v$$

Thus, a standard comparison theorem can be used to show that the general a priori estimates below hold

$$0 \leq N_h(t) \leq N_h(0)\exp^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - \exp^{-\mu_h t})$$
$$0 \leq N_v(t) \leq N_v(0)\exp^{-\mu_v t} + \frac{\Lambda_v}{\mu_v}(1 - \exp^{-\mu_v t})$$

Combining these a priori estimates and the fact that, the right-hand side of model (4) is locally Lipschitz, we conclude that there exists a unique global solution in the domain Ω, see, Theorem 2.1.5 in Stuart and Humphties, (1998). Thus, the model (4) is a dynamical system on Ω. On the other hand, if a solution is outside the region Ω, that is $N_h(t) \geq \frac{\Lambda_h}{\mu_h}$ and $N_v(t) \geq \frac{\Lambda_v}{\mu_v}$, then, it follows from the above conservation law that $\frac{dN_h}{dt} \leq 0$ and $\frac{dN_v}{dt} \leq 0$. Hence, the above general a priori estimates show that $N_H(t)$ tends to $\frac{\Lambda_h}{\mu_h}$ and $N_v(t)$ tends to $\frac{\Lambda_v}{\mu_v}$ as $t \to \infty$. Thus, the region Ω is attracting.

2.2 Basic Reproduction Number (R_c)

The DFE of the model (4) is given by

$E_1 = (S_h^0, E_h^0, I_a^0, I_s^0, R_h^0, S_v^0, I_v^0, R_v^0) = \left(\frac{\Lambda_h}{\mu_h}, 0,0,0,0, \frac{\Lambda_v}{\mu_v}, 0,0\right)$.

From (4), F and V are obtained as,

$$F = \begin{pmatrix} 0 & \frac{\beta_1\Lambda_h}{\mu_h} & \frac{\beta_1\Lambda_h}{\mu_h} & \frac{\beta_1\Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2\Lambda_v}{\mu_v} & \frac{\beta_2\Lambda_v}{\mu_v} & \frac{\beta_2\Lambda_v}{\mu_v} \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ 0 & k_2 & 0 & 0 \\ -k_3 & -\eta & k_4 & 0 \\ 0 & 0 & 0 & k_6 \end{pmatrix},$$

where,

$$k_1 = [\theta\rho + \theta(1-\rho) + \mu_h], k_2 = (\eta + \gamma_1 + \mu_h),$$
$$k_3 = \theta(1-\rho), k_4 = (\gamma_2 + \mu_h + \delta_h),$$
$$k_5 = (\mu_h + \psi_h), k_6 = (\gamma_3 + \mu_v + \delta_v).$$

It follows that the reproduction number of the model (4), denoted by R_c, is given by

$$R_c = \frac{(\Lambda_h k_3 k_6 \mu_v \beta_1 + \Lambda_v k_1 k_4 \mu_h \beta_2)}{\mu_h \mu_v k_1 k_4 k_6}. \quad (\mathbf{6})$$

Table 2: The parameter values of model (4)

Parameter	Nominal value	Reference
β_1	0.03 yr $^{-1}$	Osman *et al.* (2020b)
β_2	0.004 yr $^{-1}$	Parshotama (2011)
Λ_h	0.002 yr $^{-1}$	Osman *et al.* (2020b)
Λ_v	0.005 yr $^{-1}$	Osman *et al.* (2020b)
μ_h	0.0001 yr $^{-1}$	Osman *et al.* (2020b)
μ_v	0.0002 yr $^{-1}$	Parshotama (2011)
θ	0.20 yr $^{-1}$	Assumed
ρ	0.6 yr $^{-1}$	Assumed
ϕ	0.3 yr $^{-1}$	Assumed
γ_1	0.4 yr $^{-1}$	Assumed
γ_2	0.7 yr $^{-1}$	Assumed
δ_h	0.001 yr $^{-1}$	Osman *et al.* (2020b)
δ_v	0.003 yr $^{-1}$	Osman *et al.* (2020b)
γ_3	0.05 yr $^{-1}$	Parshotama (2011)
ψ_h	0.004 yr $^{-1}$	Osman *et al.* (2020b)
ψ_v	0.007 yr $^{-1}$	Parshotama (2011)
η	0.5 yr $^{-1}$	Assumed

3.0 Results and Discussion

In this section, the results of the analysis of the reproduction number, sensitivity analysis and the type of bifurcation model (4) is likely to exhibit are presented.

3.1 Analysis of the Reproduction Number, R_c.

By using the threshold value, R_c, it is extremely important to determine the effect of treatment rate for individuals in the symptomatic stage of campylobacteriosis infection and the rate of progression from the exposed human population to the asymptomatic and symptomatic human population, respectively on the control of campylobacteriosis in the population. The approach used in obtaining equations (7), (8), (9) and (10) are same as used in the work of Iboi and Okuonghae, (2016).

It is obvious from (6) that

$$\lim_{\gamma_2 \to \infty} R_c = \frac{\Lambda_v \beta_2}{\mu_v(\mu_h + \psi_h)} > 0, \qquad (7)$$

$$\lim_{\theta \to \infty} R_c = G > 0, \qquad (8)$$

where,

$$G = \frac{\Lambda_h \beta_1 \mu_v(\mu_h + \psi_h)(1-\rho) + \Lambda_v \beta_2 \mu_h(\gamma_2 + \delta_h + \mu_h)}{\mu_h \mu_v(\gamma_2 + \delta_h + \mu_h)(\mu_h + \psi_h)}.$$

Hence, a campylobacteriosis control programme that results in high treatment rate for the symptomatic human population ($\gamma_2 \to \infty$) and a scenario where the rate of progression from the exposed human population to the asymptomatic and symptomatic human population, respectively are high can lead to the effective control of campylobacteriosis if the results on the right-hand sides of (7) and (8) are less than unity. Also, by computing the partial derivatives of R_c with respect to γ_2 and θ further show the effect of these parameters on campylobacteriosis control in the population. This gives

$$\frac{\partial R_c}{\partial \gamma_2} = -\frac{(1-\rho)\Lambda_h \beta_1 \theta}{(\gamma_2 + \delta_h + \mu_h)^2(\theta + \mu_h)\mu_h} < 0\,, \quad (9)$$

$$\frac{\partial R_c}{\partial \theta} = \frac{(1-\rho)\Lambda_h \beta_1}{(\gamma_2 + \delta_h + \mu_h)(\theta + \mu_h)^2} > 0\,. \qquad (10)$$

It is evident that the right-hand side of (9) is less than zero. This implies that, an effective treatment rate for the humans at the symptomatic stage of infection will have a positive impact in reducing the spread of campylobacteriosis in the population no matter the values of the other parameters on the right-hand side of (9). Further, it is observed that the right-hand side of (10) is greater than zero. This means that, the progression rate of individuals from the exposed human population to the asymptomatic and symptomatic human population will have a negative impact in triggering the prevalence rate of

campylobacteriosis in the population if precautionary measures are not taken.

3.2 Sensitivity Analysis

The aim of conducting sensitivity analysis is basically to determine the contribution of each model parameters associated with the reproduction number (R_c) as they affect the transmission or otherwise of campylobacteriosis in the population (Nyasagare *et al.* 2019, Osman *et al.* 2020a, Gweryina *et al.* 2021). The normalized forward sensitivity index and contour map illustration approaches are adopted.

Definition 3.1. The normalized forward sensitivity index of a variable, m, which depends differentially on the parameter, n, is defined as:

$$\Pi_n^m = \frac{\partial m}{\partial n} \times \frac{n}{m}. \qquad (11)$$

In models of epidemiology, the value of R_c determines whether or not campylobacteriosis will spread within the population. The sensitivity indices of each parameter associated with the reproduction number determines the contribution of each parameter in the dynamics of the disease. In this wise, the sensitivity of R_c to each of the parameters connected with the reproduction number is derived. The results of the sensitivity indices of R_c, with respect to each parameter of the model as contained in the reproduction number, are presented in Table 3.

Table 3: The parameters values of model (4)

Parameter	Sensitivity index
β_1	+0.6326
β_2	+0.3674
Λ_h	+0.6326
Λ_v	+0.3674
θ	+0.0003
ρ	-0.9489
γ_2	-0.6316
δ_v	-0.0022

From the results of the sensitivity analysis of R_c, it is observed that increasing (γ_2) would decrease R_c and decreasing (γ_2) would increase R_c. Also, an increase in $\beta_1, \beta_2, \Lambda_h, \Lambda_v$ and θ would cause a corresponding increase in R_c and a decrease in the values of ρ and δ_v would cause a decrease in R_c.

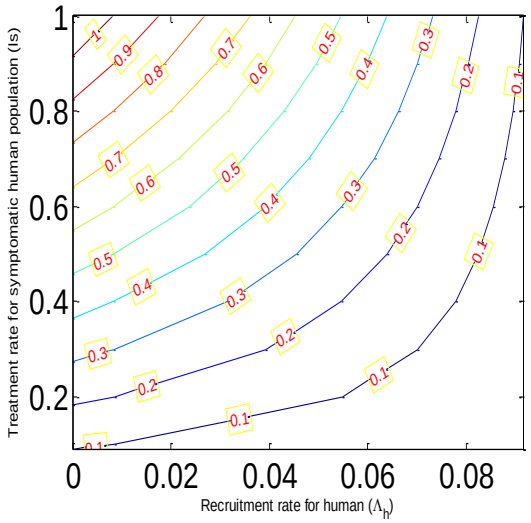

Figure 3.1. Contour plot of R_c as a function of human recruitment rate (Λ_h) and treatment rate for the symptomatic human population (γ_2).

In Figure 3.1, a contour plot of the reproduction number R_c, as a function of the human recruitment rate (Λ_h) and treatment rate for symptomatic human population (I_s) is shown using the parameter values in Table 2. It is observed here that as the human recruitment rate and treatment rate for symptomatic human population increases, the reproduction number also increases. This implies that it will not be possible to eliminate campylobacteriosis in the community with at least 70% treatment rate administered to the symptomatic human population.

3.2 Backward Bifurcation Analysis

The phenomenon of backward bifurcation is normally found in models that have multiple endemic equilibria when $R_c < 1$ (Greenhalgh *et al.* 2000, Hadeler and van den Driessche, 1997). Hence, the classical epidemiological requirement of having $R_c < 1$, is necessary but no longer sufficient for effective disease control or elimination (Anguelov *et al.* 2014). We claim the following result.

Theorem 3.1. Model (4) exhibits backward bifurcation at $R_c = 1$ whenever the bifurcation coefficients, denoted by a and b are positive.

Proof. Suppose $E_2 = (S_h^{**}, E_h^{**}, I_a^{**}, I_s^{**}, R_h^{**}, S_v^{**}, I_v^{**}, R_v^{**})$ represents any arbitrary endemic equilibrium of model (4) (that is an equilibrium in which at least one of the infected components is non-zero). The existence of backward bifurcation will be explored using the centre manifold theory (Carr, 1981, Castillo-Chavez and Song, 2004). In order to apply this theory, the following change of variables is performed. Let $S_h = x_1, E_h = x_2, I_a = x_3, I_s = x_4, R_h = x_5, S_v = x_6, I_v = x_7$ and $R_v = x_8$. Further, by using the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$, the model (4) is written in the form $\frac{dX}{dt} = F(X)$ with $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$, as shown in equation (12).

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = \Lambda_h - \beta_1\lambda x_1 - \mu_h x_1 + \psi_h x_5,\\
\frac{dx_2}{dt} &\equiv f_2 = \beta_1\lambda x_1 - k_1 x_2,\\
\frac{dx_3}{dt} &\equiv f_3 = \theta\rho x_2 - k_2 x_2,\\
\frac{dx_5}{dt} &\equiv f_5 = \gamma_1 x_3 + \gamma_2 x_4 - k_5 x_5, \qquad (\mathbf{12})\\
\frac{dx_6}{dt} &\equiv f_6 = \Lambda_v - \beta_2\lambda x_6 - \mu_v x_6 + \psi_v x_8,\\
\frac{dx_7}{dt} &\equiv f_7 = \beta_2\lambda x_6 - k_6 x_7,\\
\frac{dx_8}{dt} &\equiv f_8 = \gamma_3 x_7 - k_7 x_8.
\end{aligned}$$

where,

$$k_1 = [\theta\rho + \theta(1-\rho) + \mu_h], k_2 = (\eta + \gamma_1 + \mu_h), k_3 = \theta(1-\rho)$$

and, the force of infection is given by

$$\lambda = (x_3 + x_4 + x_7). \qquad (\mathbf{13})$$

The case where $\beta_1 = \beta_2 = \beta^*$ is choosen as the bifurcation parameter is considered. Solving for $\beta_1 = \beta_2 = \beta^*$ from $R_c = 1$, gives

$$\beta_1 = \beta_2 = \beta^* = \frac{\mu_h\mu_v k_1 k_4 k_6}{(\Lambda_h k_3 k_6 \mu_v + \Lambda_v k_1 k_4 \mu_h)}. \qquad (\mathbf{14})$$

The Jacobian of the transformed system (12), evaluated at the DFE (E_1) with $\beta_1 = \beta_2 = \beta^*$, is given by

$$J^* = \begin{pmatrix}
-\mu_h & 0 & -d_1 & -d_1 & \psi_h & 0 & -d_1 & 0\\
0 & -k_1 & -d_1 & d_1 & 0 & 0 & d_1 & 0\\
0 & \theta\rho & -k_2 & 0 & 0 & 0 & 0 & 0\\
0 & k_3 & \eta & -k_4 & 0 & 0 & 0 & 0\\
0 & 0 & \gamma_1 & \gamma_2 & -k_5 & 0 & 0 & 0\\
0 & 0 & -d_2 & -d_2 & 0 & -\mu_v & -d_2 & \psi_v\\
0 & 0 & d_2 & d_2 & 0 & 0 & (d_2 - k_6) & 0\\
0 & 0 & 0 & 0 & 0 & 0 & \gamma_3 & -k_7
\end{pmatrix}$$

with $d_1 = \frac{\beta^*\Lambda_h}{\mu_h}$ and $d_2 = \frac{\beta^*\Lambda_v}{\mu_v}$.

Matrix $J(E_0)$ has a right eigenvector given by $w = [w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8]^T$, where

$$\begin{aligned}
w_1 &= \frac{1}{\mu_h}[\psi_h w_5 - d_1(w_3 + w_4 + w_7)]\\
w_2 &= w_2 > 0,\\
w_3 &= \frac{\theta\rho}{k_3} w_2 > 0,\\
w_4 &= \frac{1}{k_4}(k_3 w_2 + \eta w_3) > 0,\\
w_5 &= \frac{1}{\mu_v}[\psi_v w_8 - d_2(w_3 + w_4 + w_7)],\\
w_6 &= \frac{1}{\mu_v}[\psi_v w_8 - d_2(w_3 + w_4 + w_7)],\\
w_7 &= -\frac{1}{(d_2 - k_6)}[d_2(w_3 + w_4)],\\
w_8 &= \frac{\gamma_3}{k_7} w_7.
\end{aligned}$$

Furthermore, J^* has a left eigenvector, $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)$, satisfying $v \cdot w = 1$, with

$$\begin{aligned}
v_1 = 0, v_2 = v_2 > 0,\\
v_3 = \frac{1}{k_2}[d_1 v_2 + \eta v_4 + d_2 v_7],\\
v_4 = \frac{1}{k_4}[d_1 v_2 + d_2 v_7], v_5 = v_6 = 0.
\end{aligned}$$

It follows from Theorem 4.1 in Castillo-Chavez and Song, (2004), if we compute the associated non-zero partial derivatives of $F(X)$ at the DFE (E_0), that the associated bifurcation coefficients, a and b, defined by

$$a = \sum_{k,i,j=1}^{8} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0),$$

and

$$b = \sum_{k,i=1}^{8} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0),$$

are computed to be

$$a = 2\beta^*(w_3 + w_4 + w_7)(v_2 w_1 + v_7 w_6),$$

and

$$b = (w_3 + w_4 + w_7)\left(v_2 \frac{\Lambda_h}{\mu_h} + v_7 \frac{\Lambda_v}{\mu_v}\right).$$

It is instructive to note that the eigenvectors $v_2, w_2, w_3, w_4, w_5, w_6, w_7$ are all positive. It follows from Theorem 4.1 in Castillo-Chavez and Song, (2004) that model (4), or the transformed model (12), will undergo backward bifurcation if the backward bifurcation coefficients a and b is positive.

3.3 Existence of Unique Endemic Equilibrium: Special Case

To establish the existence of endemic equilibria of model (4), for the special case $\psi_h = \psi_v = 0$, let $E_2^* = (S_h^{**}, E_h^{**}, I_a^{**}, I_s^{**}, R_h^{**}, S_v^{**}, I_v^{**}, R_v^{**})$ represent any arbitrary endemic equilibrium of model (4). The equations in (4), with $\psi_h = \psi_v = 0$, are solved in terms of the force of infection at steady state to get

$$S_h^{**} = \frac{\Lambda_h}{(\beta_1\lambda^{**} + \mu_h)}, E_h^{**} = \frac{\lambda^{**}\beta_1\Lambda_h}{k_1(\beta_1\lambda^{**} + \mu_h)},$$
$$I_a^{**} = \frac{\lambda^{**}\theta\rho\beta_1\Lambda_h}{k_1k_2(\beta_1\lambda^{**} + \mu_h)}, S_v^{**} = \frac{\Lambda_v}{(\beta_2\lambda^{**} + \mu_v)}, \mathbf{(15)}$$
$$I_v^{**} = \frac{\lambda^{**}\beta_2\Lambda_v}{k_6(\beta_2\lambda^{**} + \mu_v)}, R_v^{**} = \frac{\lambda^{**}\gamma_3\beta_2\Lambda_v}{\mu_v k_6(\beta_2\lambda^{**} + \mu_v)}$$

where,

$$k_1 = [\theta\rho + \theta(1-\rho) + \mu_h], k_2 = (\eta + \gamma_1 + \mu_h),$$
$$k_3 = \theta(1-\rho), k_4 = (\gamma_2 + \mu_h + \delta_h).$$

The force of infection at the steady state, λ^{**}, is expressed as

$$\lambda^{**} = (I_a^{**} + I_s^{**} + I_v^{**}). \quad \mathbf{(16)}$$

Substituting (15) into (16), simplifying and re-arranging gives the following quadratic equation in terms of λ^{**}

$$a(\lambda^{**})^2 + b\lambda^{**} + c = 0 \quad \mathbf{(17)}$$

where,

$a = \beta_1\beta_2$

$b = (\beta_1\mu_v + \beta_2\mu_h - A_1\beta_2 - A_2\beta_2 - A_3\beta_1)$

$c = \mu_h\mu_v[1 - (A_4 + A_5)R_c^1 - R_c^2]$

$A_1 = \frac{\theta\rho\beta_1\Lambda_h}{k_1k_2}, A_2 = \frac{\Lambda_h\beta_1(\eta\rho\theta + k_2k_3)}{k_1k_2k_4}, A_3 = \frac{\beta_2\Lambda_v}{k_6}, A_4 = \frac{k_4}{k_3}, A_5 = \frac{(\eta\rho\theta + k_2k_3)}{k_2k_4}$.

Theorem 3.2. The model (4), for the special case: $\psi_h = \psi_v = 0$, has.

i. A unique endemic equilibrium if $c < 0$.

ii. A unique endemic equilibrium if $b < 0$ and $c = 0$ or $\Delta = b^2 - 4ac = 0$.

iii. Two endemic equilibria if $b < 0, c > 0$ and $\Delta > 0$.

iv. No endemic equilibrium otherwise.

It is instructive to state that a is always positive and c is positive if $R_c < 1$ and negative if $R_c > 1$. Hence, it is clear from case (i) of Theorem 5.2 that model (4), for the special case $\psi_h = \psi_v = 0$, has a unique endemic equilibrium whenever $R_c > 1$.

4.0 Conclusion

In this paper, an analysis is conducted on the reproduction number, sensitivity analysis is also carried out on the model parameters connected to the reproduction number and the possibility of backward bifurcation is explored on the campylobacteriosis mathematical model developed in Ashezua *et al.* (2024). Results from the sensitivity analysis show that the most sensitive parameters are the infection rates, progression rate and the treatment rate for humans. It is further observed that as the human recruitment rate and treatment rate for symptomatic human population increases, the effective reproduction number also increases. This implies that it will not be possible to eliminate campylobacteriosis in the population with at least 70% treatment rate administered to the symptomatic human population. The analysis of the campylobacteriosis model equally reveal that the model exhibits the phenomenon of backward bifurcation under certain conditions, where a stable disease-free equilibrium (DFE) coexists with a stable endemic equilibrium (EE) when the associated reproduction number (R_C) is less than unity. It is further shown for a special case that, a unique endemic equilibrium exists whenever the associated reproduction number is greater than unity.

The study can be extended in several ways such as incorporating the effect of contaminated surfaces on the transmission dynamics of campylobacteriosis, carrying out optimal control and cost-effectiveness analysis of the treatment parameters. Further theoretical results such as the global asymptotic stability of the unique endemic equilibrium, for the special case $\psi_h = \psi_v = 0$ can be explored.

Declarations

Ethics approval and consent to participate

Not Applicable.

Consent for publication

All authors have read and consented to the submission of the manuscript.

Availability of data and material

Not Applicable.

Competing interests

All authors declare no competing interests.

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