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Modelling and Analysis of Campylobacteriosis in Human and Animal Populations

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Abstract

Campylobacter infection is a common cause of travelers diarrhea and gastroenteritis globally. This can be manifested through acute diarrhea. Campylobacter jejuni and campylobacter coli are the most common causes of acute enteritis while campylobacter fetus is the most common cause of extra-intestinal illness. We proposed and analysed a campylobacter model describing the dynamics of the disease in human and animal population. Ordinary differential equations and the stability theory was used in the model's qualitative analysis. It was revealed that the model exhibited multiple endemic equilibrium. Sensitivity analysis was carried out to determine the contribution of each parameter on the basic reproduction number. Numerical simulation of the model was conducted and the results displayed graphically.

Keywords: Campylobacteriosis, equilibrium point, local stability, global stability, reproductive number.

1. INTRODUCTION

Campylobacter infection is one of the leading causes of travelers diarrhea and gastroenteritis across the globe which is mainly manifested through acute diarrhea. Eleven species of campylobacter are known to cause human illness out of the many species of campylobacter [16]. Campylobacter jejuni and campylobacter coli are the most common causes of acute enteritis while campylobacter fetus is the most common cause of extra-intestinal illness. C. jejuni is the most common cause of acute enteritis accounting for over 80 percent of all reported cases followed by C. coli.

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The diarrhea is always self-limiting with its peak at 24 to 48 hours after exposure and resolves on average after 6 days. C. jejuni and C. coli infect people of all ages but it is more prevalent in young children aged between 1 and 4 years old and young adults aged between 15 and 24. The disease is more endemic in developing countries particularly among young children. Research shows that the infection ratio decreases with age which can be attributed to the fact that individuals develop acquired immunity once they infected at a tender age thus boosting population-level immunity [17]. As a consequence, asymptomatic campylobacter is more prevalent in developing countries thus impacting the transmission rates through contamination of the environment by asymptomatic excretions. The campylobacter bacteria are carried by various wild and domestic animals, however, in most cases it is traceable to poultry products or water. Geographical distribution of disease varies greatly due to variance of standards of handling food and availability of campylobacter reserves in those regions. The infection manifests itself in the form of acute watery diarrhea, fever, cramps, chills, vomiting prodromal headache, myalgia, and weight loss that persists for 6 days. Campylobacter infection is self-limiting and is not known to cause bacteraemia. However, it is associated with complications such as Guillain-Barre syndrome, inflammatory bowel disease, colorectal cancer, Miller Fischer syndrome, and Barrett's Oesophagus. However, there is limited knowledge about the role of campylobacter bacteria in those complications [19].

Campylobacteriosis is a common class of infections caused by Gram-negative bacteria that belong to the genus of campylobacter. It is one of the leading causes of diarrhea diseases across the world with at least 550 million cases of campylobacter enteritis annually, of which 200 million are children under the age of 5 years [16]. However, surveillance in middle and low income countries is minimal leading to under reporting of the cases thus understating the disease burden is the global south. For instance, in sub-Saharan Africa, there were 3.8 million reported deaths of children under the age of 5 years of which 25 percent were due to diarrhea disease. In a multi site study undertaken in African countries, Asia, and Latin America between 2009 and 2012, campylobacter species was the most prevalent pathogen accounting for 84.9 percent of the 1892 children examined. Furthermore, the study found out that it found out that it contributed to the highest burden of diarrhea diseases borne by children in their first year. As such, it is directly linked to the high mortality rate of children under the age of 5 in sub-Saharan Africa [18].

Schielke, Rosner, and Stark (2014) conducted an analysis on the campylobacteriosis surveillance data collected in Germany in the last ten years starting from 2001 through to 2010. They found out that there was an increase of in campylobacteriosis in the said period with an annual increment of 72 infections per 100000 [16]. The trend is common across European countries in the said period with most infections acquired domestically. The study also affirmed that the increment can be attributed to an increase in poultry consumption which increased from 8.4 kg per capita to 9.9 kg per capita. It further found out from routine monitoring of zoonotic pathogens that there was a campylobacter prevalence in poultry meat ranging from 14 percent to 64 percent annually. In particular, the surveillance found out high prevalence among children under the age of four years with one-year boys more adversely affected. It concluded that there was need for comprehensive programmes to mitigate the colonization of campylobacter in chicken to control the spread of campylobacteriosis in human beings [16].

Infectious disease models, generally are capable of predicting the future dynamics of diseases. Both the qualitative and quantitative solutions of an infectious disease model explain the behaviour of the infection with time. However, most infectious disease models employ ordinary differential equations in explaining the transmission mechanism of the infection [3, 1, 2, 10, 7, 4].

2. MODEL DESCRIPTION AND FORMULATION

We divide the model into two parts; human and animal population. These populations are subdivided into six compartments at any time, (t) which reflect their diseases status in the dynamics system. The susceptible humans (S_h), the infected humans (I_h), and the recovered humans (R_h) constitute the total human population, (N_h) given by the equation:

$$N_h(t) = S_h(t) + I_h(t) + R_h(t). \tag{2.1}$$

Moreover, the total population of the vector, (N_v) is given by the equation:

$$N_v(t) = S_v(t) + I_v(t) + R_v(t) \tag{2.2}$$

The susceptible, (S_v) , the infected (I_v) and recovered, (R_v). Where, Λ_h denotes rate at which the susceptible are recruited into the class. Susceptible humans are infected with campylobacteriosis at a rate of $(I_h + I_v)\beta$. Humans recover from campylobacteriosis at a rate γ and recovered individuals move to the susceptible class at a rate σ_h. Humans die naturally at a rate of μ_h. Vectors are recruited at a rate Λ_v. Vectors are infected with campylobacter through contact with infected humans or other infected vectors at a rate of $(I_v + I_h)\lambda$. They recover at a rate α. They have a natural death rate of μ_v. We define $B_m^* = (I_v + I_h)$ for convenience of calculation.

We derived the following system of differential equations from our model:

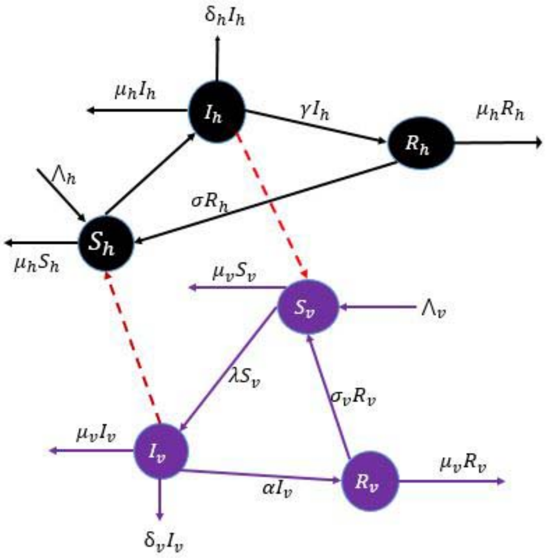

Figure 1: Flow diagram for Campylobacteriosis model.

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h+\sigma_h R_h-\beta_m^*\beta S_h-\mu_h S_h\\
\frac{dI_h}{dt} &= \beta_m^*\beta S_h-\gamma I_h-(\mu_h+\delta_h)I_h\\
\frac{dR_h}{dt} &= \gamma I_h-(\sigma_h+\mu_h)R_h\\
\frac{dS_v}{dt} &= \Lambda_v-\beta_m^*\lambda S_v-\mu_v S_v+\sigma_v R_v\\
\frac{dI_v}{dt} &= \beta_m^*\lambda S_v-\alpha I_v-(\mu_v+\delta_v)I_v\\
\frac{dR_v}{dt} &= \alpha I_v-(\sigma_v+\mu_v)R_v
\end{aligned}\right\} \tag{2.3}$$

3. POSITIVITY AND BOUNDEDNESS OF SOLUTIONS

Since the population under consideration is human and animal, the solution must be non-negative. This condition of non-negativity is of great importance. The campylobacteriosis model will be biologically meaningful if solution with non-negative initial values remains non-negative with time.

Theorem 1. *Let* $\Pi=\{(S_h(t),I_h(t),R_h(t),S_v(t),I_v(t),R_v(t))\in\mathbb{R}_+^6 :,$ $(S_h(0),I_h(0),R_h(0),S_v(0),I_v(0),R_v(0))>0\}$*then solution of* $\{(S_h(t),I_h(t),R_h(t),S_v(t),I_v(t),R_v(t))\}$*are non-negative for all time*$t\geq 0.$ *Hence, if*$S_h(0),I_h(0),R_h(0),S_v(0),I_v(0),R_v(0)$*are non-negative, then* $S_h(t),I_h(t),R_h(t),S_v(t),I_v(t),R_v(t)$*are non-negative for all time* $t>0.$

At any point in time,(t):

$$N_h(t) \;=\; S_h(t) \;+\; I_h(t) \;+\; R_h(t). \tag{3.1}$$

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \tag{3.2}$$

$\frac{dN_h}{dt} = \Lambda_h - \mu_h S_h - (\mu_h + \delta_h) I_h - (\sigma_h + \mu_h) R_h.$
When there are no campylobacteriosis infections;
$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h.$
Solving the equation;

$\Lambda_h - \mu_h N_h \geq Ae^{-\mu_h t}$, A is constant.

Setting initial condition, $N_h(0) = N_{h(0)}$,

$\Lambda_h - \mu_h N_{h(0)} = A$

Hence, $\Lambda_h - \mu_h N_h \geq \left(\Lambda_h - \mu_h N_{h(0)}\right) e^{-\mu_h t}$.

$N_h \leq \frac{\Lambda_h}{\mu_h} - \left(\frac{\Lambda_h - \mu_h N_{h(0)}}{\mu_h}\right) e^{-\mu_h t}.$

As $t \to \infty$, $N_h \to \frac{\Lambda_h}{\mu_h}$.

Hence, $0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}$ and $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$.

If $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$.

$$\Pi_h = \{(S_h, I_h, R_h) \in \mathbb{R}^3_3 : S_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h} \tag{3.3}$$

For total vector population at any time(t):

$$N_v(t) \;=\; S_v(t) \;+\; I_v(t) \;+\; R_v(t). \tag{3.4}$$

Hence;

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dI_v}{dt} + \frac{dR_v}{dt} . \tag{3.5}$$

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v - \delta_v I_v. \tag{3.6}$$

In the absence of campylobacteriosis infections;

$$\frac{dN_v}{dt} \leq \Lambda_v - \mu_v N_v. \tag{3.7}$$

$\Lambda_v - \mu_v N_v \geq Ae^{-\mu_v t}$, A is constant.
Setting initial condition, $N_v(0) = N_{v(0)}$,
$\Lambda_v - \mu_v N_{v(0)} = A$
Hence, $\Lambda_v - \mu_v N_v \geq \left(\Lambda_v - \mu_v N_{v(0)}\right) e^{-\mu_v t}$.
$N_v \leq \frac{\Lambda_v}{\mu_v} - \left(\frac{\Lambda_v - \mu_v N_{v(0)}}{\mu_v}\right) e^{-\mu_v t}$.
As $t \to \infty$, $N_v \to \frac{\Lambda_v}{\mu_v}$.
Hence, $0 \leq N_v \leq \frac{\Lambda_v}{\mu_v}$ and $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$.
When, $N_v(0) \leq \frac{\Lambda_v}{\mu_v}$, then $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$.
Hence,

$$\Pi_v = \left\{ (S_v, I_v, R_v, V_v) \in \mathbb{R}^3_+ : S_v + I_v + R_v \leq \frac{\Lambda_v}{\mu_v} \right\}. \tag{3.8}$$

Feasible region for the system in (2.3) is given by:

$$\Pi = \Pi_h \times \Pi_v \subset \mathbb{R}^3_+ \times \mathbb{R}^3_+ . \tag{3.9}$$

Where Π is positively invariant.

4. DISEASE FREE EQUILIBRIUM

By setting the system of differential equations in (2.3) to zero, the disease free equilibrium can be obtained. The diseases free equilibrium is given by:
$\xi_0 = \left(S_h^*, I_h^*, R_h^*, S_v^*, I_v^*, R_v^* \right)$.

$$\frac{dS_h}{dt} = \Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h = 0 \tag{4.1}$$

$S_h^* = \frac{\Lambda_h}{\mu_h}$.
At DFE, there are no campylobacter infections.

$$\left.\begin{aligned} I_h^* &= 0 \\ R_h^* &= 0 \end{aligned}\right\} \text{and} \left.\begin{aligned} I_v^* &= 0 \\ R_v^* &= 0 \end{aligned}\right\}.$$

$$\frac{dS_v}{dt} = \Lambda_v \quad -\beta_m^*\lambda S_v \quad -\mu_v S_v + \sigma_v R_v \quad = 0 \tag{4.2}$$

$$\Lambda_v \quad -\mu_v S_v \quad = 0$$

$$S_v^* \; = \; \frac{\Lambda_v}{\mu_v}.$$

$$S_v^* \; = \; \frac{\Lambda_v}{\mu_v}.$$

$$\xi_0 = \left(\frac{\Lambda_h}{\mu_h}, \;\; 0, \;\; 0, \;\; \frac{\Lambda_v}{\mu_v}, \;\; 0, \;\; 0, \right). \tag{4.3}$$

4.1 Stability of the disease free equilibrium

Proposition. *Disease free equilibrium point is locally asymptotically stable if the basic reproduction*

number (R_0) *is less than one* $(R_0 < 1)$ *and unstable if the basic reproduction number is greater*

than one $(R_0 > 1)$.

5. BASIC REPRODUCTION NUMBER

In this section, we employ the concept of “Next Generation Matrix” in [12, 15, 8, 11] to obtain our reproductive rate, (R_{hv}).

Consider the infectious compartment of the campylobacteriosis model in 1:

$$\left.\begin{aligned} \frac{dI_h}{dt} &= \beta_m^*\beta S_h - \gamma I_h - (\mu_h + \delta_h) I_h \\ \frac{dI_v}{dt} &= \beta_m^*\lambda S_v - \alpha I_v - (\mu_v + \delta_v) I_v \end{aligned}\right\} \tag{5.1}$$

Let f and v be total number of new infection coming into and out of the system respectively;

$$f = \begin{bmatrix} \beta_m^*\beta S_h \\ \beta_m^*\lambda S_v \end{bmatrix}, v = \begin{bmatrix} \gamma I_h + (\mu_h + \delta_h) I_h \\ \alpha I_v + (\mu_v + \delta_v) I_v \end{bmatrix}.$$

Then the Jacobian matrix of f and v are as follows:

$$F = \begin{bmatrix} \beta S_h & \beta S_h \\ \lambda S_v & \lambda S_v \end{bmatrix} \tag{5.2}$$

$$V = \begin{bmatrix} \gamma + (\mu_h + \delta_h) & 0 \\ 0 & \alpha + (\mu_v + \delta_v) \end{bmatrix} \tag{5.3}$$

Jacobian matrix at DFE:

$$F = \begin{bmatrix} \beta S_h^* & \beta S_h^* \\ \lambda S_v^* & \lambda S_v^* \end{bmatrix}, \tag{5.4}$$

$$V = \begin{bmatrix} \gamma + (\mu_h + \delta_h) & 0 \\ 0 & \alpha + (\mu_v + \delta_v) \end{bmatrix}. \tag{5.5}$$

Hence;

$$V^{-1} = \begin{bmatrix} \dfrac{1}{\gamma + (\mu_h + \delta_h)} & 0 \\ 0 & \dfrac{1}{\alpha + (\mu_v + \delta_v)} \end{bmatrix}$$

However,

$$FV^{-1} = \begin{bmatrix} \dfrac{\beta S_h^*}{(u_2 + \gamma) + (\mu_h + \delta_h)} & \dfrac{\beta S_h^*}{(u_4 + \alpha) + (\mu_v + \delta_v)} \\ \dfrac{\lambda S_v^*}{\gamma + (\mu_h + \delta_h)} & \dfrac{\lambda S_v^*}{\alpha + (\mu_v + \delta_v)} \end{bmatrix} \tag{5.6}$$

$$FV^{-1} = \begin{bmatrix} \dfrac{\beta S_h^*}{\gamma + (\mu_h + \delta_h)} & \dfrac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} \\ \dfrac{\lambda S_v^*}{\gamma + (\mu_h + \delta_h)} & \dfrac{\lambda S_v^*}{\alpha + (\mu_v + \delta_v)} \end{bmatrix} \tag{5.7}$$

Obtaining the eigenvalues of FV^{-1} and picking the dominant one.

$$\begin{vmatrix} \dfrac{\beta S_h^*}{\gamma + (\mu_h + \delta_h)} - A & \dfrac{\beta S_h^*}{\alpha + (\mu_v + \delta_v)} \\ \dfrac{\lambda S_v^*}{\gamma + (\mu_h + \delta_h)} & \dfrac{\lambda S_v^*}{\alpha + (\mu_v + \delta_v)} - A \end{vmatrix} = 0$$

Where Adenotes eigenvalues.

$$A^2 - \left[\left(\frac{\lambda S_v^*}{\alpha + (\mu_v + \delta_v)} \right) + \left(\frac{\beta S_h^*}{\gamma + (\mu_h + \delta_h)} \right) \right] A = 0 \tag{5.8}$$

$A_1 = 0$ and $A_2 = \left[\left(\dfrac{\lambda S_v^*}{\alpha + (\mu_v + \delta_v)} \right) + \left(\dfrac{\beta S_h^*}{\gamma + (\mu_h + \delta_h)} \right) \right].$

Dominant eigenvalue is A_2. This implies that;

$$R_{hv} = \left[\left(\frac{\beta S_h^*}{\gamma + (\mu_h + \delta_h)}\right) + \left(\frac{\lambda S_v^*}{[\alpha + (\mu_v + \delta_v)]}\right)\right]. \tag{5.9}$$

At DFE,

$$R_{hv} = \left(\frac{\beta \Lambda_h}{\mu_h(\gamma) + (\mu_h + \delta_h)}\right) + \left(\frac{\lambda \Lambda_v}{\mu_v[(\alpha) + (\mu_v + \delta_v)]}\right). \tag{5.10}$$

Where;

$R_h = \left(\frac{\beta \Lambda_h}{\mu_h(\gamma) + (\mu_h + \delta_h)}\right)$ and $R_v = \frac{\lambda \Lambda_v}{\mu_v[\alpha + (\mu_v + \delta_v)]}$.

5.1 Local stability of the disease free equilibrium

Theorem 2. *The disease free equilibrium is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Since DFE was obtained as; $\left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0\right)$.
Jacobian matrix of the dynamical system :

$$\begin{pmatrix} -(\beta_m^*\beta + \mu_h) & -\beta S_h^* I_v^* & \sigma_h & 0 & \beta S_h I_h & 0 \\ \beta_m^*\beta & r_1 & 0 & 0 & \beta S_h I_h & 0 \\ 0 & \gamma & -(\sigma_h + \mu_h) & 0 & 0 & 0 \\ 0 & -\lambda S_v^* I_h^* & 0 & -(\beta_m^*\lambda + \mu_v) & -\lambda S_v^* I_h^* & \sigma_v \\ 0 & \lambda S_v^* I_h^* & 0 & \beta_m^*\lambda & r_2 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -(\sigma_v + \mu_v) \end{pmatrix}$$

We denote $r_1$and $r_2$as;

$r_1 = \left(\beta S_h^* I_v^* - \mu_h - \delta_h - \gamma\right)$

$r_2 = \lambda S_v^* I_h^* - (\alpha + \mu_v + \delta_v)$

$DFE = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0\right)$

$$\begin{pmatrix} -\mu_h & 0 & \sigma_h & 0 & 0 & 0 \\ 0 & -(\mu_h + \delta_h + \gamma) & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\sigma_h + \mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \mu_v & 0 & \sigma_v \\ 0 & 0 & 0 & 0 & -(\alpha + \mu_v + \delta_v) & 0 \\ 0 & 0 & 0 & 0 & \alpha & -(\sigma_v + \mu_v) \end{pmatrix}$$

Finding the eigenvalues;

$$\begin{pmatrix} -\mu_h & 0 & \sigma_h & 0 & 0 & 0 \\ 0 & -(\mu_h+\delta_h+\gamma) & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\sigma_h+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & 0 & \mu_v & 0 & \sigma_v \\ 0 & 0 & 0 & 0 & -(\alpha+\mu_v+\delta_v) & 0 \\ 0 & 0 & 0 & 0 & \alpha & -(\sigma_v+\mu_v) \end{pmatrix}$$

The eigenvalues are as follows; $\left.\begin{array}{c} -\mu_v \\ -\mu_h \\ -(\mu_v+\delta_v) \end{array}\right\}, \left.\begin{array}{c} -(\mu_h+\delta_h) \\ -(\alpha+\mu_v+\delta_v) \\ -(\gamma+\mu_h+\delta_h) \end{array}\right\}$

All eigenvalues are negative, implying DFE is locally asymptotically stable.

5.2 Global stability of the disease-free equilibrium.

Theorem 3. *If $R_{hv} \leq 1$, the disease-free equilibrium is globally asymptotically stable in the interior of Φ.*

Proof: Consider the Lyapunov function;

$$P(t) = (\alpha+\mu_v+\delta_v)I_h + (\gamma+\mu_h+\delta_h)I_v \tag{5.11}$$

The derivative of $P(t)$ along the solutions of the system in(2.3);

$$\left| \begin{array}{rc} \dfrac{dP(t)}{dt} = & (\alpha+\mu_v+\delta_v)\dfrac{dI_h}{dt} + (\gamma+\mu_h+\delta_h)\dfrac{dI_v}{dt} \\ = & (\alpha+\mu_v+\delta_v)(\beta S_h(I_h+I_v) - (\gamma+\mu_h+\delta_h)I_v) \\ & +(\gamma+\mu_h+\delta_h)[\lambda S_v(I_h+I_v) \\ & -(\alpha+\mu_v+\delta_v)I_v] \\ \leq & (\alpha+\mu_v+\delta_v)\dfrac{\beta\Lambda_h I_h}{\mu_h} + (\alpha+\mu_v+\delta_v)\dfrac{\beta\Lambda_h I_v}{\mu_h} \\ & -(\alpha+\mu_v+\delta_v)(\gamma+\mu_h+\delta_h)I_h \\ & +I_h(\gamma+\mu_h+\delta_h)\left(\dfrac{\lambda\Lambda_v}{\mu_v(\tau+\mu_v)}\right) \\ & +I_v(\gamma+\mu_h+\delta)\left(\dfrac{\lambda\Lambda_v}{\mu_v(\tau+\mu_v)}\right) \\ & +I_h(\gamma+\mu_h+\delta_h) + I_v(\gamma+\mu_h+\delta) \\ & -I_v(\gamma+\mu_h+\delta)(\alpha+\mu_v+\delta_v) \\ \leq & -I_h(\gamma+\mu_h+\delta_h)(\alpha+\mu_v+\delta_v)(1-R_{hv}) \\ & -I_v(\gamma+\mu_h+\delta_h)(\alpha+\mu_v+\delta_v)(1-R_{hv}) \\ = & -(I_h+I_v)(\gamma+\mu_h+\delta_h)(\alpha+\mu_v+\delta_v)(1-R_{hv}) \end{array} \right\} \tag{5.12}$$

Time derivative of $P(t)$ along the system gives:

$\left(\frac{dP(t)}{dt}\right) \leq 0$, if and only if $R_{hv} < 0$

$\left(\frac{dP(t)}{dt}\right) = 1$, if and only if $I_h + I_v = 0$ or $R_{hv} = 1$.

Hence, highest invariant set in $\left\{S_h, I_h, I_v, \in \Phi, \frac{dP(t)}{dt} = 0\right\}$,if $R_{hv} \leq 1$, is singleton ξ_0.

Hence, ξ_0 GAS in Φ. By LaSalle's invariant principle [14, 5, 13, 7].

6. ENDEMIC EQUILIBRIUM

6.1 Global stability of endemic equilibrium

This section considers the analysis of the global behaviour of dynamical system in (2.3).

Theorem 4. *The system of differential equations in equation is said to have a unique endemic equilibrium if $R_{hv} > 1$, and it is globally asymptotically stable.*

The EE point of the system exists if and only if $R_{hv} > 1$. Assuming $R_{hv} > 1$, then clearly EE point exists.

Consider a Lyapunov function defined as:

$$\left.\begin{array}{c} L\left(S_h^*, I_h^*, R_h^* S_v^*, I_v^* R_v^*\right) = \left(S_h - S_h^* - S_h^* \ln \frac{S_h^*}{S_h}\right) \\ + \left(I_h - I_h^* - I_h^* \ln \frac{I_h^*}{I_h}\right) + \left(R_h - R_h^* - R_h^* \ln \frac{R_h^*}{R_h}\right) + \\ \left(S_v - S_v^* - S_v^* \ln \frac{S_v^*}{S_v}\right) + \left(I_v - I_v^* - I_v^* \ln \frac{I_v^*}{I_v}\right) + \left(R_v - R_v^* - R_v^* \ln \frac{R_v^*}{R_v}\right) \end{array}\right\} \tag{6.1}$$

Taking the time derivative along the system;

$$\left.\begin{array}{c} \frac{dL}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\frac{dS_h}{dt} + \left(\frac{I_h - I_h^*}{I_h}\right)\frac{dI_h}{dt} + \left(\frac{R_h - R_h^*}{R_h}\right)\frac{dR_h}{dt} \\ + \left(\frac{S_v - S_v^*}{S_v}\right)\frac{dS_v}{dt} + \left(\frac{I_v - I_v^*}{I_v}\right)\frac{dI_v}{dt} + \left(\frac{R_v - R_v^*}{R_v}\right)\frac{dR_v}{dt} \end{array}\right\} \tag{6.2}$$

Therefore;

$$\left\{\begin{array}{l} \dfrac{dL}{dt} = \left(\dfrac{S_h - S_h^*}{S_h}\right)\left[\Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h\right] \\ \quad + \left(\dfrac{I_h - I_h^*}{I_h}\right)\left[\beta_m^* \beta S_h - \gamma I_h - (\mu_h + \delta_h) I_h\right] \\ \quad + \left(\dfrac{R_h - R_h^*}{R_h}\right)\left[\gamma I_h - (\sigma_h + \mu_h) R_h\right] + \\ \left(\dfrac{S_v - S_v^*}{S_v}\right)\left[\Lambda_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v\right] \\ \quad + \left(\dfrac{I_v - I_v^*}{I_v}\right)\left[\beta_m^* \lambda S_v - \alpha I_v - (\mu_v + \delta_v) I_v\right] \\ \quad + \left(\dfrac{R_v - R_v^*}{R_v}\right)\left[\alpha I_v - (\sigma_v + \mu_v) R_v\right] \end{array}\right\}$$

Alternatively written as;

$$\left\{\begin{array}{c} \dfrac{dL}{dt} = \Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h - \dfrac{\Lambda_h S_h^*}{S_h} + \beta_m^* \beta S_h^* - \dfrac{\sigma_h R_h S_h^*}{S_h} + \mu_h S_h^* \\ + \beta_m^* \beta S_h - \gamma I_h - (\mu_h + \delta_h) I_h - \dfrac{\beta_m^* S_h I_h^*}{I_h} + \gamma I_h^* + \mu I_h^* + \delta I_h^* + \gamma I_h - \\ (\sigma_h + \mu_h) R_h - \dfrac{\gamma_h I_h R_h^*}{R_h} + \sigma_h R_h^* + \mu_h R_h^* + \Lambda_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v - \\ \dfrac{\Lambda_v S_v^*}{S_v} + \beta_m^* \lambda S_v^* - \dfrac{\sigma_v R_v S_v^*}{S_v} + \mu_v S_v^* + \beta_m^* \lambda S_v - \alpha I_v - \mu_v I_v - \delta_v I_v - \dfrac{\beta_m^* S_v I_v^*}{I_v} \\ + \alpha I_v^* + \mu_v I_v^* + \delta_v I_v^* + \alpha I_v - \sigma_v R_v - \mu_v R_v - \dfrac{\alpha I_v R_v^*}{R_v} + \sigma_v R_v^* + \mu_v R_v^* \end{array}\right\}$$

Let;

$$\frac{dL}{dt} = M - N \tag{6.3}$$

Denoting M and N as positive and negative quantities respectively.

$$\left.\begin{array}{c} M = \Lambda_h + \sigma_h R_h + \mu_h S_h^* + \beta_m^* \beta S_h + \gamma I_h^* + \mu I_h^* + \delta I_h^* + \gamma I_h + \sigma_h R_h^* + \mu_h R_h^* + \Lambda_v \\ + \sigma_v R_v + \beta_m^* \lambda S_v^* + \mu_v S_v^* + \beta_m^* \lambda S_v + \alpha I_v^* + \mu_v I_v^* + \delta_v I_v^* + \alpha I_v + \sigma_v R_v^* + \mu_v R_v^* \end{array}\right\}$$

and

$$\left.\begin{array}{l} N = \mu_h S_h + \dfrac{\Lambda_h S_h^*}{S_h} + \dfrac{\sigma_h R_h S_h^*}{S_h} + \gamma I_h + (\mu_h + \delta_h) I_h + \dfrac{\beta_m^* S_h I_h^*}{I_h} + (\sigma_h + \mu_h) R_h + \dfrac{\gamma_h I_h R_h^*}{R_h} + \\ \beta_m^* \lambda S_v + \mu_v S_v + \dfrac{\Lambda_v S_v^*}{S_v} + \dfrac{\sigma_v R_v S_v^*}{S_v} + \alpha I_v + \mu_v I_v + \delta_v I_v + \dfrac{\beta_m^* S_v I_v^*}{I_v} + \sigma_v R_v + \mu_v R_v + \dfrac{\alpha I_v R_v^*}{R_v} \end{array}\right\}$$

Imposing a condition; if $M < N$, then $\dfrac{dL}{dt} \leqslant 0$.

But $\dfrac{dL}{dt} = 0$, if and only if $S_h = S_h^*, I_h = I_h^*, R_h = R_h*, S_v = S_v^*, I_v = I_v^*, R_v = R_v^*$

Hence, highest or largest invariant set in;

$$\left\{(S_h^*, I_h^*, R_h^* S_v^*, I_v^* R_v^*) \in \Phi : \frac{dL}{dt} = 0\right\} \tag{6.4}$$

is E^*, where $E^*$is EE point.

By [5, 6, 9], as $t \to \infty$, every solution of the system approaches the EE point if $R_{hv}> 1$. Hence, EE point is GAS in the invariant set whenever $M < N$.

7. NUMERICAL SOLUTIONS

In this section, we perform quantitative analysis of the system of differential equations of the model using Range-Kutta fourth order scheme. The duration of the spread of the disease through interaction between people at risk of infection was taken to be three months.

7.1 Human and vector population dynamics

2 shows the population dynamics of susceptible, infectious and recovered human and vector populations. An increase in the population at risk of contracting campylobacter corresponds to an increase in the population of infectious. As the population of the susceptible increases through the recruitment rate, so as the population people infected with campylobacter increases with time in the system.

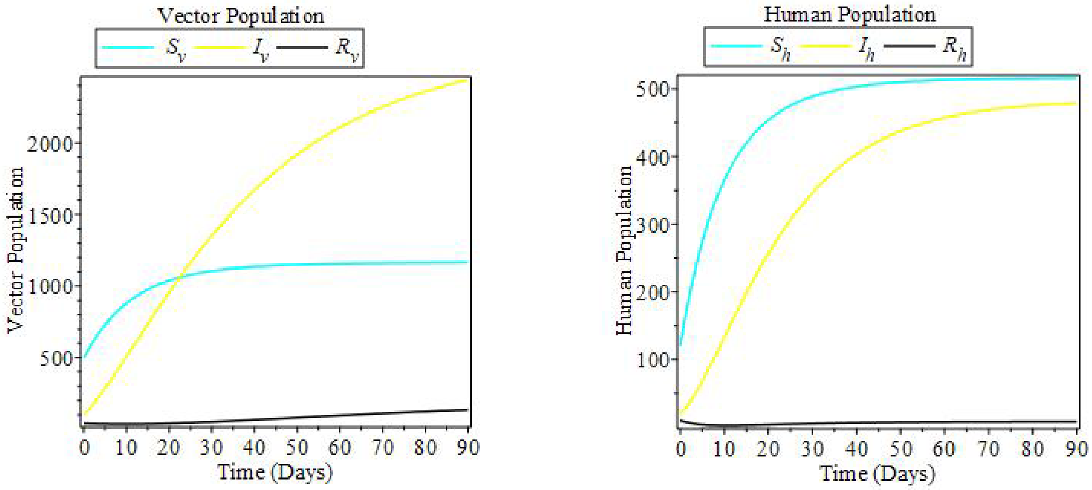

Figure 2: Human and vector population dynamics.

7.2 Infectious and recovered human and Vector population

The diagram in 3 shows the population distribution of people infected with campylobacter and people who have recovered from campylobacter infections. It can be observed that the rate at which people are infected with campylobacter far outweighs the rate at which people recover from campylobacter infections in the system.

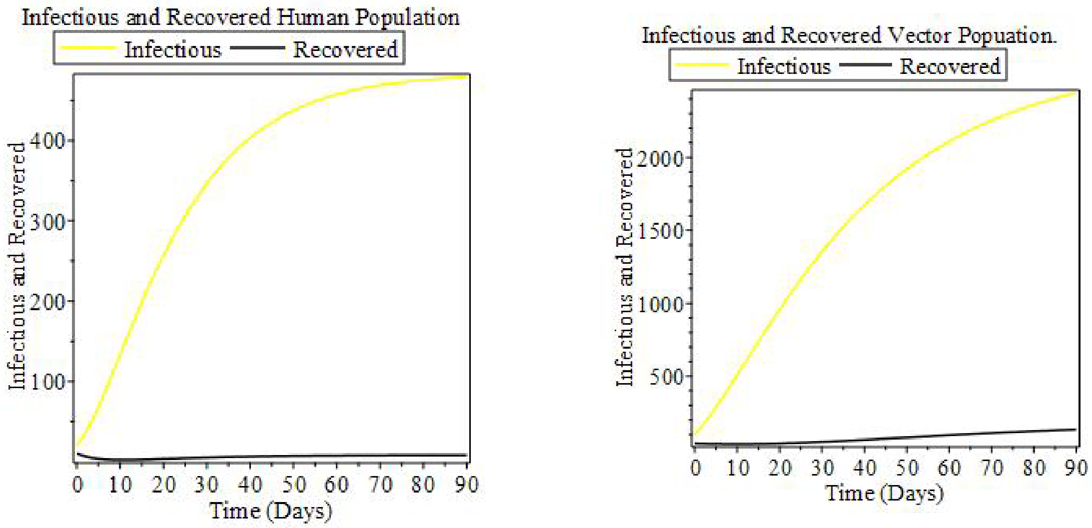

Figure 3: Infectious and recovered population of human and vector.

7.3 Effects of contact rate on vector population

4 shows the analysis of contact rate of the vector population with regards to campylobacter transmission in the system. The contact rate is varied to see its effects on the disease dynamics. It can be observed that an increase in the contact rate increases in the number of the infectious population. Hence, the contact rate of the vector population has an effect in the disease transmission. Campylobacter can be reduced by ensuring that the rate of contact between the population of vectors is controlled to the barest minimum.

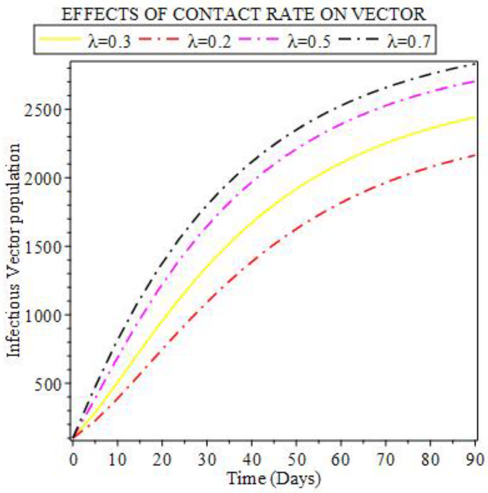

Figure 4: Effects of contact rate on vector population.

7.4 Effects of contact rate on human population

The diagram in 5 shows the analysis of contact rate of the human population with regards to campylobacter transmission in the system. Contact rate is varied to see its effects on the campylobacter dynamics. An increase in the contact rate increases in the number of the infectious population as shown in 5. Hence, human contact rate has an effect in the disease transmission. Campylobacter can be reduced by ensuring that human contact rate is reduced or controlled to the barest minimum.

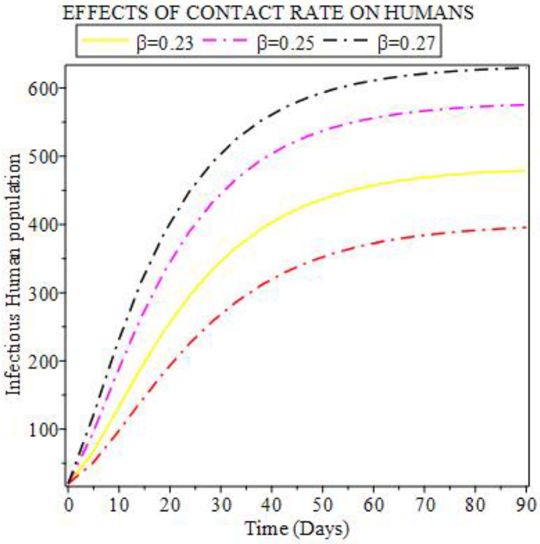

Figure 5: Effects of contact rate on human population.

8. CONCLUSION

A campylobacteriosis model for the dynamics of campylobacter infection was developed. The qualitative solution of of the model was determined by computing the basic reproductive number, equilibrium points and existence of the equilibrium points. This showed an existence of multiple endemic equilibrium points. The biological implication is that control of campylobacteriosis can be achieved whenever the basic reproductive number is less than a critical value.

We carried out sensitivity analysis of the reproduction number and it indicated that increasing contact rate would increase the reproduction number. However, decreasing contact rate would increase the reproductive number.

Numerical simulation revealed that human contact rate has an effect in the campylobacteriosis transmission. Campylobacter can be reduced by ensuring that human contact rate is reduced or controlled to the barest minimum. Contact rate of the vector population has effects in the campylobacteriosis transmission. Campylobacter can be reduced by ensuring that the rate of contact between the population of vectors is controlled to the barest minimum.

9. DATA AVAILABILITY

The data used in the analysis of the campylobacteriosis model were from previously published articles and reported studies which have been cited accordingly. Some of the parameter values are assumed and others are taken from published articles. These published articles are also cited at relevant places within the text as references.

10. CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this manuscript.

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