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# Campylobacteriosis Transmission Dynamics in Humans: Modeling the Effects of Public Health Education, Treatment, and Sanitation

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**Abstract**
An epidemic model for the dynamics of campylobacteriosis disease with public health education, treatment, and sanitation control strategies was formulated and analyzed. The stabilities of the equilibrium points were analyzed by using the Routh-Hurwitz criterion. The effective reproduction number was computed by using the Next Generation Matrix method. Numerical simulations were carried out and the results showed that, public health education has a substantial influence on the reduction of the effective reproduction number. Moreover, treatment and sanitation control strategies have also shown significant reduction of infected individuals from the respective population.

**Keywords**: Campylobacteriosis; public health education; treatment; sanitation.

**2000 MR subject classification:** 92B05, 92D30, 93A30.

## Introduction

Campylobacteriosis is the infection caused by the bacterium *Campylobacter*, and it is the most common cause of human gastroenteritis (Acheson and Allos 2001, Nyasagare et al. 2019, Rawson et al. 2019). There are about 13 species of *Campylobacter*, of which *Campylobacter jejuni* and *Campylobacter coli* are the most frequent causes of acute bacterial gastroenteritis in humans (Cody et al. 2012, Kaakoush et al. 2015).

Humans can be affected by the disease through contaminated food and water or contact with infected individuals (Katsma et al. 2007, Cousins et al. 2019, Neves et al. 2019). Different cases have been reported in recent years across the World. About 240,379 cases were reported in 2014 in Europe and it was noted that the notification rate was 59.8 cases per 100000 population of people (ECDC 2016). In addition, reports show that 1.3 million individuals are affected every year in the USA (Kaakoush et al. 2015). However, campylobacteriosis cases were not well documented in Africa, Asia, and the Middle East due to lack of data, difficulties in diagnosis, and differences in reporting systems and surveillance though the disease is endemic in all regions (Kaakoush et al. 2015). In all regions with high cases of the campylobacteriosis disease, it is noted that the disease is zoonotic and thus poultry, pigs, cows, and their products are found to be the most common sources of campylobacteriosis disease (Kaakoush et al. 2015, Neves et al. 2019, Nyasagare et al. 2019). The major infection problems caused by *Campylobacter* are severe diarrhea, fever, and abdominal pain,

and the disease mostly affects children under 5 and young adults (20–29 years) (Acheson and Allos 2001, Gölz et al. 2014, Schielke et al. 2014, Nyasagare et al. 2019). Moreover, Louis et al. (2005) suggested that males are much more affected by campylobacteriosis disease than females; however, the reason for the gender differences in *Campylobacter* infections is unclear. Currently, prevention and control of the campylobacteriosis disease to humans and animals is done using anti-*Campylobacter* compounds, probiotics, bacteriophage, and vaccines. Additionally, some anti-*Campylobacter* bacteriocins are often used (Johnson et al. 2017).

Interventions of public health education in any epidemic aim at reducing the transmission and control of the disease. The effects of public health education on the transmission of a disease dynamics have been studied in the cases of typhoid fever (Edward and Nyerere 2016), HIV/AIDS (Abraham and Sheeran 1994, Ogoye-Ndegwa 2005, Mukandavire et al. 2009, Hussaini 2010), Ebola (Levy et al. 2017), etc. Studies have shown that public health education campaigns improve general understanding of the disease and in turn, it helps to reduce disease transmissions (Del Valle et al. 2005, Joshi et al. 2008, Mukandavire et al. 2009, Levy et al. 2017). Moreover, Kwasi-Do et al. (2019), Edward and Nyerere (2015), and Rai et al. (2019) described the roles of treatment and sanitation control strategies in the reduction of transmission of infectious diseases. All the studies have shown effectiveness of treatment and sanitation control strategies in the control of the spread of cholera and other infectious diseases.

Several mathematical models have been developed to describe the transmission dynamics of campylobacteriosis disease (Katsma et al. 2007, van Gerwe et al. 2009, Cousins et al. 2019, Neves et al. 2019, Nyasagare et al. 2019). Despite the use of all these mathematical models to study the transmission dynamics of campylobacteriosis disease, still there is lack of knowledge in reducing the transmission of campylobacteriosis among the human population.

In this article, we propose a mathematical model to study the dynamics of campylobacteriosis disease among the human population in the presence of public health education, treatment, and sanitation as control strategies. Our model is extended from the *SIR* model of Adimy et al. (2020) to include the exposed population of humans and the bacterial population which was considered as the polluted environment such as food and water.

## Materials and Methods

### Model descriptions and formulation

This study aimed at developing a structured $SEIRC$ mathematical model that incorporates the effects of the public health education, treatment, and sanitation control parameters on the transmission of campylobacteriosis in humans and the incidence rate as proposed in Sahu and Dhar (2012), Jana et al. (2016), and Khan et al. (2018). In the formulation of this model, it was assumed that there is a direct transmission of the disease among humans if hygiene measures are not well maintained. The formulated model is made up of two populations, the human population and the concentration of *Campylobacter* in the environment. Here we assume that the *Campylobacter* in the environment is the result of polluted food and water from the infected humans and animals (Connerton and Connerton 2017). The human population is divided into four classes, namely $S$, a healthy individual who is in danger of getting the disease at any time $t$ when conditions allow, $E$, the exposed population of humans who are assumed to have acquired the disease but not showing any clinical signs of the disease and not capable of infecting other humans. Again, $I$ is the infected population with all clinical signs of the disease and capable of transmitting and shedding the *Campylobacter* into the environment. The last class is the recovered population denoted by $R$ which is usually temporal.

Moreover, $C$ is the concentration of *Campylobacter* in the environment which gets polluted by the infected human $I$. The dotted line between $C$ and $S$ indicates the interaction of susceptible humans with the polluted environment that contributes to the force of infections $A(t)$for the disease to develop at the rate $\tau C$. The dotted line between $C$ and $I$shows the interactions between the polluted environment and the infected human population which increase the rate of bacteria into the environment $C$ at the rate $\psi I$. The susceptible population is recruited at a constant birth rate $\Lambda$ and get infections when swallows the polluted food or water from the environment at the non-linear incident rate

$$A(t) = \xi IS + (1-\omega)\frac{\tau CS}{1+\alpha C} \qquad (1)$$

where $\tau > 0$ is the transmission rate of bacteria between humans and the environment, $\xi$ is the person-to-person transmission rate, and

$\alpha > 0$ is the half-saturation constant. The parameter $\omega$ in a range of $0 \leq \omega \leq 1$ is the public health education which is introduced to the susceptible population to increase the control awareness of the campylobacteriosis disease among humans. The ranges of omega indicate the time when there are no public health awareness programs to the society and when the full public health awareness programs are introduced to the whole society. Two to seven days after new infections, a proportion $\xi IS + (1-\omega)\frac{\tau CS}{1+\alpha C}$ of susceptible individuals progress to the exposed class $E$. The exposed class is reduced by the natural death rate parameter $\mu$ and the incubation period parameter $\varphi$.The infected humans are reduced by the natural death at rate $\mu$, the disease-induced death rate $\delta$, the recovery rate $\rho$, as well as the treatment control parameter $b$. The recovery of humans from campylobacteriosis is temporal and hence, a person who recovers from the disease reverts to the susceptible population at the rate $\sigma R$. The recovered population of humans is reduced by the natural death rate $\mu$ and the bacteria are cleared out from the environment at the rate $\phi C$. The removal rate of bacteria from the environment is through the sanitation practices such as practicing good hygiene in both food and water through washing hands before eating, cooking thoroughly poultry products especially eggs and meat, and avoiding touching uncooked meat by bare hands. The epidemiological representation of campylobacteriosis disease is as shown in Figure 1.

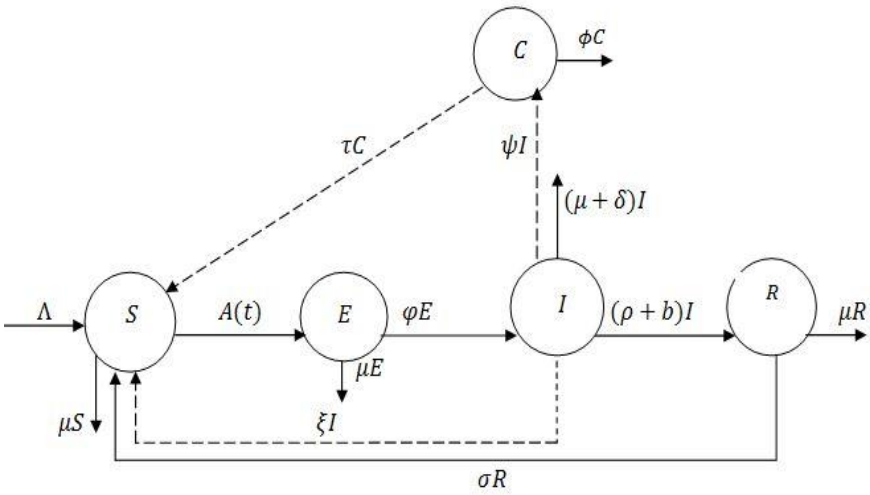


**Figure 1:** Compartmental model for campylobacteriosis disease transmission.

Based on the model descriptions and schematic diagram in Figure 1, the model equations with saturated incidence rate and public health education, treatment, and sanitation control strategies are represented by a system of the following non-linear differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda + \sigma R - \xi IS - (1-\omega)\frac{\tau CS}{1+\alpha C} - \mu S \\
\frac{dE}{dt} &= \xi IS + (1-\omega)\frac{\tau CS}{1+\alpha C} - (\mu+\varphi)E \\
\frac{dI}{dt} &= \varphi E - (\mu+\delta+\rho+b)I \\
\frac{dR}{dt} &= (\rho+b)I - (\mu+\sigma)R \\
\frac{dC}{dt} &= \psi I - \phi C
\end{aligned} \qquad (2)$$

with non-negative initial conditions, $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, $C(0) > 0$.

**Positivity and boundedness of the solution**

**Theorem**

*Given* $S(0) > 0,\ E(0) > 0,\ I(0) > 0,\ R(0) > 0,\ C(0) > 0$; *the solution* $S(t), E(t), I(t), R(t),$ *and* $C(t)$ *of the model (2) are positively invariant for all* $t \in \mathbb{R}^+$.

**Proof**

For the model to be biologically meaningful, it is required to prove that all of the state variables are non-negative $\forall t > 0$. Now let $T = \sup\{S, E, I, R, C\}$,

then, $S(0) > 0;\ E(0) > 0;\ I(0) > 0;\ R(0) > 0;\ C(0) > 0$ on time $t$.

Thus, the first equation of the model (2) becomes

$$\frac{dS}{dt} \geq \Lambda - (A_0 + \mu)S \qquad (3)$$

where $A_0(t) = (1-\omega)\frac{\tau C}{1+\alpha C}$. Integrating Equation (3) with respect to $t$ the result is

$$S \geq \frac{\Lambda}{A_0+\mu} + \left(S(0) - \frac{\Lambda}{A_0+\mu}\right)e^{-(A_0+\mu)t} \qquad (4)$$

as $t \to \infty$, $S(t) \geq \frac{\Lambda}{A_0+\mu} > 0$.

Using the same procedures, all other state variables could be shown that $E > 0$, $I > 0$, $R > 0$ and $C > 0, \forall t > 0$.

Furthermore, let us consider the sum of the first three equations of the model (2), at any time $t$ we have the total human population;

$$N(t) = S(t) + E(t) + I(t) + R(t) \qquad (5)$$

$$\frac{d(S(t) + E(t) + I(t) + R(t))}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \qquad (6)$$

$$\frac{dN}{dt} = \Lambda - \delta I - \mu N \leq \Lambda - \mu N \qquad (7)$$

Solving the inequality (7) results into

$$N \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}.$$

Applying limits we get the solution as $\lim_{t\to\infty} \sup N(t) \leq \frac{\Lambda}{\mu}$

which follows that $0 \leq N(t) \leq \frac{\Lambda}{\mu}$.

For the bacteria class, we have

$$\frac{dC}{dt} = \psi I - \phi C \leq \psi N - \phi C \qquad (8)$$

It follows that,

$$\psi I - \phi C \leq \frac{dC}{dt} \leq \psi N - \phi C \leq \frac{\psi\Lambda}{\mu} - \phi C \qquad (9)$$

and hence the solution

$$0 \leq \lim_{t\to\infty} \inf\ C(t) \leq \lim_{t\to\infty} \sup\,(C(t)) \leq \frac{\psi\Lambda}{\mu}.$$

This shows that all solutions $(S(t), E(t), I(t), R(t), \mathrm{C(t)})$ of the model (2) are bounded and

well-posed in the invariant set $\Omega = \Omega_1 \cup \Omega_2 = \mathbb{R}_+^4 \times \mathbb{R}_+^1$

where $\Omega = \left\{(S,E,I,R) \in \mathbb{R}_+^4 : 0 \le N(t) \le \frac{\Lambda}{\mu}; C(t) \in \mathbb{R}_+^1 : 0 \le C(t) \le \frac{\psi\Lambda}{\mu}\right\}$

**Model analysis**

**Existence of equilibria of the model**

Model (2) has one disease-free equilibrium point $D_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ obtained after solving $\frac{dX}{dt} = 0$ where $X$ refers to the state variable of the model when $E(t) = I(t) = R(t) = C(t) = 0$.

The model has also an endemic equilibrium point $D^*(S^*, E^*, I^*, R^*, C^*)$, where

$$
\begin{aligned}
S^* &= \frac{\left(\phi+\alpha\ \psi\ I^*\right)\left(\Lambda\ (\mu+\sigma)+\sigma(\rho+b)I^*\right)}{(\mu+\sigma)\left[\xi\ I^*\left(\phi+\alpha\ \psi\ I^*\right)+(1-\varpi)\ \tau\ \psi\ I^*\right]}\\
E^* &= \frac{\Lambda\ (\mu+\sigma)+\sigma(\rho+b)\ I^*}{(\mu+\sigma)(\mu+\varphi)}\\
I^* &= \frac{\varphi\ \Lambda\ (\mu+\sigma)}{(\mu+\sigma)(\mu+\varphi)(\mu+\rho+\delta+b)-\sigma\ \varphi\ (\rho+b)}\\
R^* &= \frac{\varphi\ \Lambda(\rho+b)}{(\mu+\sigma)(\mu+\varphi)(\mu+\rho+\delta+b)-\sigma\ \varphi\ (\rho+b)}\\
C^* &= \frac{\psi\ \varphi\ \Lambda\ (\mu+\sigma)}{\phi(\mu+\sigma)(\mu+\varphi)(\mu+\rho+\delta+b)-\sigma\ \varphi\ (\rho+b)}
\end{aligned}
\qquad (10)
$$

**The effective reproduction number**

The effective reproduction number denoted by $R_e$ is an average number of secondary cases caused by one infected individual introduced in a purely susceptible population (Jana et al. 2016, Kelatlhegile and Kgosimore 2016, Mbuthia and Chepkwony 2019). Here we find the effective reproduction number using the Next Generation matrix as in Van den Driessche and Watmough (2002). Using this method, $R_e$ is defined as the spectral radius of $FV^{-1}$. Now, let us consider the infected classes of the model (2),

$$
\begin{aligned}
\frac{dE}{dt} &= \xi IS + (1-\omega)\frac{\tau CS}{1+\alpha C} - (\mu+\varphi)E\\
\frac{dI}{dt} &= \varphi E - (\mu+\delta+\rho+b)I\\
\frac{dR}{dt} &= (\rho+b)I - (\mu+\sigma)R\\
\frac{dC}{dt} &= \psi I - \phi C
\end{aligned}
\qquad (11)
$$

which can be written as

$$\frac{dZ}{dt} = B(Z) - G(Z) \qquad (12)$$

where $B(Z)$ is the rate of new infection in the population and $G(Z)$ is the rate of transfer in

and out of the population. Therefore

$$B(z)=\begin{pmatrix} \xi IS+(1-\omega)\dfrac{\tau CS}{1+\alpha C} \\ 0 \\ 0 \end{pmatrix}$$

and $G(z)=\begin{pmatrix} (\mu+\varphi)E \\ -\varphi E+(\mu+\rho+\delta+b)I \\ -\psi I+\phi C \end{pmatrix}$.

Then

$$F=\frac{\partial B(S,I,C)}{dt}\Big|D_0=\begin{pmatrix} 0 & \dfrac{\xi\Lambda}{\mu} & \dfrac{\tau\Lambda(1-\omega)}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$ and

$$V=\frac{\partial G(E,I,C)}{dt}\Big|D_0=\begin{pmatrix} \mu+\varphi & 0 & 0 \\ -\varphi & \rho+\mu+\delta+b & 0 \\ 0 & -\psi & \phi \end{pmatrix}$$

The effective reproduction number is the spectral radius of $FV^{-1}$ as given in Equation (13).

$$R_e=\frac{\Lambda\varphi\left(\xi\phi+\tau\psi(1-\omega)\right)}{\mu\phi(\mu+\varphi)(\rho+\mu+\delta+b)} \qquad (13)$$

This dimensionless quantity is made of the survival rate of human beings $\frac{1}{\mu}$, the infectious period $\frac{1}{\phi(\mu+\varphi)(\rho+\mu+\delta+b)}$, contact rate of a human being with the contaminated environment $\tau$, the human recruitment rate $\Lambda$, the shading rate of bacteria by the infected human $\psi$, and interaction rate of infected and susceptible humans $\xi$.

**Local stability of the disease-free equilibrium point**

**Theorem 2.** *From the model (2), the disease-free equilibrium point* $(D_0)$ *is locally asymptotically stable if* $R_e<1$, *and unstable if* $R_e>1$, *where,* $R_e$ *is defined by equation (13).*

**Proof**: The next generation matrix at the disease-free equilibrium point is

$$J(D_0)=\begin{bmatrix} -\mu & 0 & -\dfrac{\xi\Lambda}{\mu} & \sigma & \dfrac{-(1-\omega)\tau\Lambda}{\mu} \\ 0 & -(\mu+\varphi) & \dfrac{\xi\Lambda}{\mu} & 0 & \dfrac{(1-\omega)\tau\Lambda}{\mu} \\ 0 & \varphi & -(\mu+\delta+\rho+b) & 0 & 0 \\ 0 & 0 & (\rho+b) & -(\mu+\sigma) & 0 \\ 0 & 0 & \psi & 0 & -\phi \end{bmatrix} \qquad (14)$$

After the computation of the trace, $Tr\,(J(D_0))$, and the determinant, $\det(J(D_0))$ of the variational matrix (14), the results are

$Tr\left(J(D_0)\right)=-\left(4\mu+\varphi+\sigma+\phi+\delta+\rho+b\right)$

and

$\det(J(D_0))=\mu\phi(\mu+\sigma)(\mu+\varphi)(\rho+\mu+\delta+b)(1-R_e)$.

Following these results, it is seen that $Tr\,(J(D_0))<0$ , $\det(J(D_0))>0$ for $R_e<1,$ . Hence, by the Routh-Hurwitz criterion, the disease-free equilibrium point is asymptotically stable whenever $R_e<1$.

**Local stability of the endemic equilibrium point**

The endemic equilibrium point exists if the disease persists in a population.

**Theorem 3**. *The endemic equilibrium point of model (2) is locally asymptotically stable if* $R_e > 1$.

**Proof**: We use the Routh-Hurwitz criterion in proving the existence of the endemic equilibrium point.

Now, let us consider the variational matrix of model (2) at $D^*(S^*,\ E^*, I^*, R^*,\ C^*)$

$$J^* - \lambda I^* = \begin{bmatrix} -K_1 - \lambda & 0 & -K_2 & \sigma & -K_3 \\ K_4 & -(\mu+\varphi) - \lambda & K_2 & 0 & K_3 \\ 0 & \varphi & -(\mu+\delta+\rho+b) - \lambda & 0 & 0 \\ 0 & 0 & (\rho+b) & -(\mu+\sigma) - \lambda & 0 \\ 0 & 0 & \psi & 0 & -\phi - \lambda \end{bmatrix} \tag{15}$$

Where:

$$K_1 = \frac{\xi\ I^*\left(\phi+\alpha\ \psi\ I^*\right)+(1-\omega)\,\alpha\ \psi\ I^*}{\phi+\alpha\ \psi\ I^*} + \mu$$

$$K_2 = \frac{\xi\ \left(\Lambda\ (\mu+\sigma)+\sigma(\rho+b)I^*\right)}{(\mu+\sigma)\left[\xi\ I^*\left(\phi+\alpha\ \psi\ I^*\right)+(1-\omega)\,\tau\ \psi\ I^*\right]}$$

$$K_3 = \frac{\phi^2\ (1-\omega)\,\tau\left(\phi+\alpha\ \psi\ I^*\right)\left(\Lambda\ (\mu+\sigma)+\sigma(\rho+b)I^*\right)}{(\mu+\sigma)\left(\phi+\alpha\ \psi\ I^*\right)^2\left[\xi\ I^*\left(\phi+\alpha\ \psi\ I^*\right)+(1-\omega)\,\tau\ \psi\ I^*\right]}$$

$$K_4 = \frac{\xi\ I^*\left(\phi+\alpha\ \psi\ I^*\right)+(1-\omega)\,\tau\ \psi\ I^*}{\phi+\alpha\ \psi\ I^*}$$

Its characteristics polynomial is given by

$$a_0\lambda^5 + a_1\lambda^4 + a_2\lambda^3 + a_3\lambda^2 + a_4\lambda + a_5 = 0 \tag{16}$$

where as

$$a_0 = 32$$

$$a_1 = 16\left(\varphi + b + \delta + 3\mu + \phi + \rho + \sigma + K_1\right)$$

$$a_2 = 24\mu^2 + 8\mu\left(b+\delta+\sigma+\varphi+3\phi+\rho+3K_1\right) + 8\varphi\left(b+\delta+\rho+\sigma+\phi+K_1-K_2\right)$$
$$+ 8\phi\left(b+\delta+\sigma+\rho+K_1\right) + 8K_1\left(b+\delta+\sigma+\rho\right) + 8\,\sigma\left(b+\delta+\rho\right)$$

$$a_3 = 4\mu^3 + 4\mu^2(b+\delta+\sigma+\varphi+3\phi+\rho+3K_1)$$
$$+\mu\begin{pmatrix}4\varphi(b+\delta+\sigma+\rho+2\phi+2K_1-K_2)+4\phi(2b+2\delta+2\sigma+2\rho+3K_1)\\+8K_1(b+\delta+\sigma+\rho)+4\sigma(b+\delta+\rho)\end{pmatrix}$$
$$+\varphi(4\phi(b+\delta+\sigma+\rho+K_1-K_2)+4K_1(b+\delta+\sigma+\rho-K_2)+4\sigma(b+\delta+\rho-K_2)+4K_4K_2-4\psi K_3)$$
$$+\phi(4K_1(b+\delta+\sigma+\rho)+4\sigma(b+\delta+\rho))+4K_1\sigma(b+\delta+\rho)$$

$$a_4 = 2\mu^3(\phi+K_1)+2\mu^2(\varphi(\phi+K_1)+\phi(b+\delta+\sigma+\rho+3K_1)+K_1(b+\delta+\sigma+\rho))$$
$$+2\mu\begin{pmatrix}((b+\delta+\sigma+\rho+2K_1-K_2)\phi+(b+\delta+\sigma+\rho-K_2)K_1+K_4K_2-\psi K_3)\varphi\\+\phi(K_1(2b+2\delta+2\sigma+2\rho)+\sigma(b+\delta+\rho))+\sigma K_1(b+\delta+\rho)\end{pmatrix}$$
$$+2\varphi\begin{pmatrix}(b+\delta+\sigma+\rho-K_2)K_1+(\sigma(b+\delta+\rho-K_2)+K_4K_2)\phi+K_1((b+\delta+\rho-K_2)\sigma-\psi K_3)\\+\sigma(K_2K_4-K_3\psi-K_4b-K_4\rho)\sigma+2K_4\psi K_3\end{pmatrix}$$
$$+2K_1\phi\sigma(b+\delta+\rho)$$

$$a_5 = K_1\phi\,\mu^3 + K_1\phi(b+\delta+\sigma+\rho+\varphi)\mu^2$$
$$+((((b+\delta+\sigma+\rho-K_2)K_1+K_4K_2)\phi+\psi K_3(K_4-K_1))\varphi+K_1\phi\sigma(b+\delta+\rho))\mu$$
$$-\varphi\sigma(((-b-\delta-\rho+K_2)K_1+K_4(b+\rho-K_2))\phi-\psi K_3(K_4-K_1))$$

Using the Routh-Hurwitz criterion on the polynomial equation of degree five, $D^*$ is asymptotically stable if and only if $H_1 = a_1 > 0$, $H_2 = a_1a_2 - a_3 > 0$,
$H_3 = a_1a_2a_3 + a_1a_5 - a_1^2a_4 - a_3^2 > 0$,
$H_4 = (a_3a_4 - a_2a_5)(a_1a_2 - a_3) - (a_1a_4 - a_5)^2 > 0$,
and $H_5 = a_4H_4 > 0$.

**Global stability of the disease-free**

**Theorem 4**. *The disease-free equilibrium point* $(D_0)$ *of system 2 is globally asymptotically stable provided that* $R_e < 1$.

**Proof**: The global stability of the disease-free equilibrium point is proved using Castillo-Chavez et al. (2002). We write the system as

$$\frac{dX}{dt} = M(X, I)$$
$$\frac{dY}{dt} = N(X, I); \quad N(X,0) = 0 \qquad (17)$$

where $X$ and $I$ defines the susceptible and infected population, respectively. In this case, the disease-free equilibrium point $D_0$ is defined as $D_0 = \{X^*, 0\}$ where $D_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$. It is enough to prove the two conditions that;

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

(ii) $N(X, I) = TI - \hat{N}(X, I)$; $\hat{N}(X, I) \geq 0$, for $(X, I) \in \Omega$, where $\Omega$ is the invariant region and $T = D_1N(X^*, 0)$ is an M-matrix with non-negative off-diagonal elements.

Now consider the matrix

$$\frac{dX}{dt} = M(X,0) = \begin{pmatrix}\Lambda - \mu S\\0\\0\\0\end{pmatrix} \qquad (18)$$

with $X = \{S\} \in \mathbb{R}^1_+$, it is obvious that the solution of equation (18) is

$S(t) = \frac{\Lambda}{\mu} + \left( S(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$. It suffices to say that $S(t) \to \frac{\Lambda}{\mu}$ as $t \to \infty$, and thus condition (i) holds.

For condition (ii), we need to show that, $S(X,I) = TI - \hat{N}(X,I); \quad \hat{N}(X,I) \geq 0$ for $(X,I) \in \Omega$.

Now the Jacobian matrix of infected individuals in the model system (2) at $D_0$ gives an M–matrix $T$ as

$$T = \begin{bmatrix} -\mu-\varphi & \frac{\xi\Lambda}{\mu} & 0 & \frac{(1-\omega)\tau\Lambda}{\mu} \\ \varphi & -\rho-\delta-\mu-b & 0 & 0 \\ 0 & \rho+b & -\mu-\sigma & 0 \\ 0 & \psi & 0 & -\phi \end{bmatrix} \quad (19)$$

with all negative entries in its main diagonal and non-negative entries in off-diagonal. Further we have

$$\hat{N}(X,I) = \begin{bmatrix} \xi\left(\frac{\Lambda}{\mu} - S\right) I + (1-\omega)\tau C\left(\frac{\Lambda}{\mu} - \frac{S}{1+\alpha C}\right) \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

with $\hat{N}_1(X,I) \geq 0$ and $\hat{N}_2(X,I) = \hat{N}_3(X,I) = \hat{N}_4(X,I) = 0$.

Therefore condition (ii) holds, and thus $D_0$ is globally asymptotically stable.

**Global stability of the endemic equilibrium point**

**Theorem 5**. *The system (2) is said to have a stable endemic equilibrium point which is globally asymptotically stable if and only if* $R_e > 1$.

*Proof*: We consider the Lyapunov function

$$L(t) = \sum_{i=1}^{n} \Phi_i(t)(X_i - X_i^* \ln X_i), \Phi_i(X_i) \geq 0 \quad (20)$$

where $\Phi$ is a positive factor and $X_i$ is a population variable for $i = 1, 2, 3, 4, 5$ and $X_i$ is the endemic equilibrium point. Now the time derivative of $L(t)$ with respect to $X_i$ is

$$\frac{dL}{dt} = \Phi_1\left(1 - \frac{X_1^*}{X_1}\right)\frac{dX_1}{dt} + \Phi_2\left(1 - \frac{X_2^*}{X_2}\right)\frac{dX_2}{dt} + \Phi_3\left(1 - \frac{X_3^*}{X_3}\right)\frac{dX_3}{dt} + \Phi_4\left(1 - \frac{X_4^*}{X_4}\right)\frac{dX_4}{dt} + \Phi_5\left(1 - \frac{X_5^*}{X_5}\right)\frac{dX_5}{dt} \quad (21)$$

Introducing constants of the model system (2) into Equation (21) leads to

$$r = -\lambda_1\Phi_1\left(1 - \frac{X_1^*}{X_1}\right)^2 X_1$$

$$\frac{dL}{dt} = r + s \quad (22)$$

$$s = -\lambda_2\Phi_2\left(1 - \frac{X_1^*}{X_1}\right)\left(1 - \frac{1}{X_2}\right)\lambda_2 X_1 - \Phi_2\left(\rho+\mu+\delta+b\right)\left(1 - \frac{X_3^*}{X_3}\right)\left(1 - \frac{X_3^*}{X_2 X_3}\right)X_3$$

$$-\Phi_4\left(\mu+\sigma\right)\left(1 - \frac{X_4^*}{X_4}\right)\left(1 - \frac{X_4^*}{X_3 X_4}\right)X_4 - \Phi_5\,\phi\left(1 - \frac{X_5^*}{X_5}\right)\left(1 - \frac{X_5^*}{X_3 X_5}\right)X_5$$

From equation (22), $\frac{dL}{dt} = 0$ if and only if $X_i^* = X_i$ hence, the largest invariant set $\left\{ X_1 ... X_4 \in \Omega : \frac{dL}{dt} = 0 \right\}$ is a singleton in $X^*$.

According to LaSalle (1976), Mafuta et al. (2013), and Chuma and Mwanga (2019), it suffices to say that $D^*$ is globally asymptotically stable in the invariant set as $t \to +\infty$.

**Results**

In this section, we perform numerical simulations of the model system (2) by using MATLAB software. The parameter values to run the model are obtained from the related literature and others are assumed depending on the epidemiology of the campylobacteriosis disease among the human population and the polluted environment. In this article, it is assumed that the lifespan of a human is 70 years which gives $\mu = 3.9 \times 10^{-5}$ per day (Patanarapelert and Tang 2007). Further, we have used the public health education parameter $(\omega)$ in the range between 0 and 1 inclusively. However, the effect of $\omega$ is done on $0\%, 50\%, 75\%$, and $90\%$ of susceptible individuals, respectively. On the other hand, the recovery period of the disease is assumed to be $2-7$ days and hence $\rho$ values range between 0.143 and 0.5 per day. We assume the initial conditions of the model to be $S(0) = 1000, E(0) = 500, I(0) = 100, R(0) = 150,$ and $C(0) = 1000$. Other parameter values are as seen in Table 1.

**Table 1:** Parameter description of the campylobacteriosis model

| Parameter | Descriptions | Value | Reference |
|---|---|---|---|
| $\Lambda$ | Human recruitment rate | 0.0042 | Coutinhoa et al. (2006) |
| $\sigma$ | Loss of immunity | 0.5 | Assumed |
| $\omega$ | Rate of education campaign | $0-1$ | Assumed |
| $\tau$ | A transmission rate of bacteria from the environment to human | 0.1 | Assumed |
| $\alpha$ | Half saturated constant | 0.01 | Assumed |
| $\mu$ | Human mortality rate | $3.9\times10^{-5}/day$ | Patanarapelert and Tang (2007) |
| $\varphi$ | The incubation period of campylobacter | 0.5-1 | Horn and Lake (2013), Awofisayo-Okuyelu et al. (2017) |
| $\rho$ | Natural human recovery rate | $0.143-0.5$ | Blaser (1997), Cousins et al. (2019) |
| $\delta$ | Disease induced rate for human | 0-1 | Assumed |
| $\psi$ | Shading rate of bacteria by the infected individuals | 0.1 | Assumed |
| $\phi$ | The rate of practicing good hygiene on the environment | 0-1 | Assumed |
| $b$ | The recovery rate of human due to treatment | 0.0025 | Assumed |
| $\xi$ | Human to a human transmission rate | 0.0005 | Assumed |

**Discussion**

In this section, we discuss the numerical results of the model system (2) and parameter values as described in Table 1. Figure 2 shows the total population of both humans and *Campylobacter* in the environment. The trend shows that, the susceptible humans decrease exponentially since the individuals who initially acquire infection develop new status and thus move to the exposed class. Similarly, the exposed population increases exponentially due to new individuals from the susceptible population and then decreases due to the mortality rate while others change status and move to the infected population. Consequently, the exponential increase in the infected human population is because some exposed individuals join this group. However, the exponential decrease of the infected population is due to natural mortality rate, disease-induced death rate, recovered individuals due to treatment, and those who recover naturally and join the recovered population. Conversely, the exponential decrease of the recovered population is caused by the removal of some individuals from the compartment due to the natural mortality rate and loss of immunity. The same trend is observed in the bacterial population where the increase is due to the shading rate of *Campylobacter* by the infected human population to the environment, while the decrease is due to good hygiene practices (sanitation) that are applied in controlling the disease.

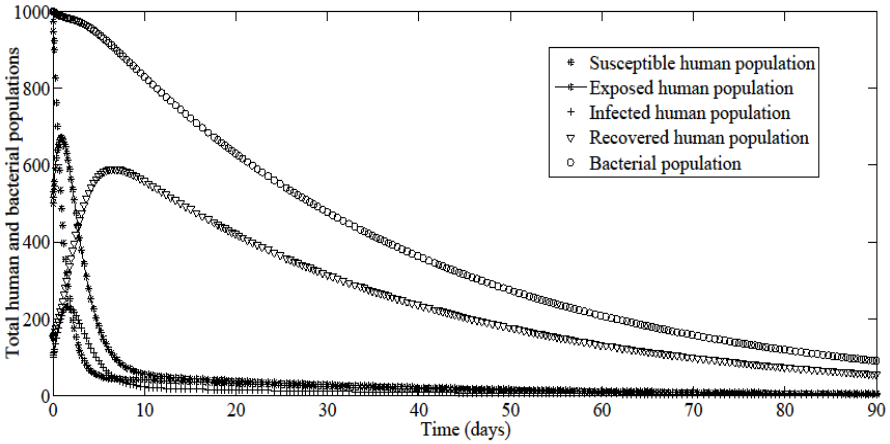


**Figure 2:** Proportions of total human and bacterial populations.

Figure 3 shows the disease-free equilibrium point of the susceptible human population. It shows that different initial values of the population converge to the same point which is referred to as the disease-free equilibrium point of the susceptible population. This demonstrates that the disease-free equilibrium is locally asymptotically stable.

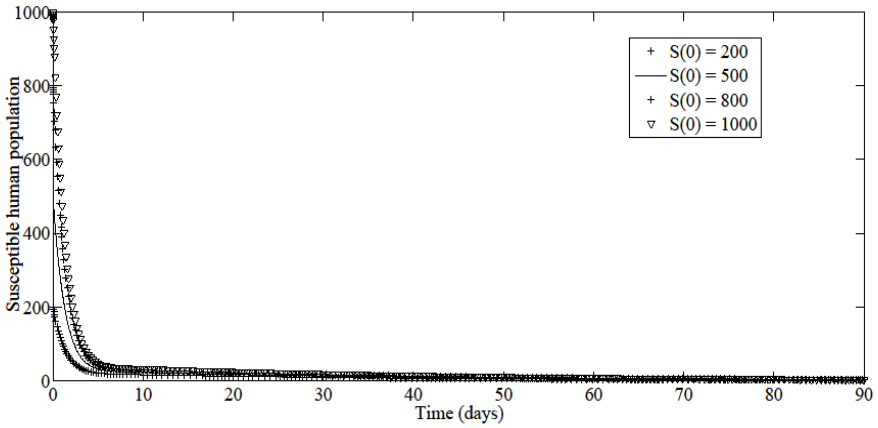


**Figure 3:** Equilibrium point of susceptible human at disease-free.

Moreover, Figures 4, 5 and 6 show the disease-free equilibrium point for the exposed, infected, and recovered populations, respectively. All of the initial conditions used in the figures converge to the same point which demonstrates that $D^*$ is globally asymptotically stable.

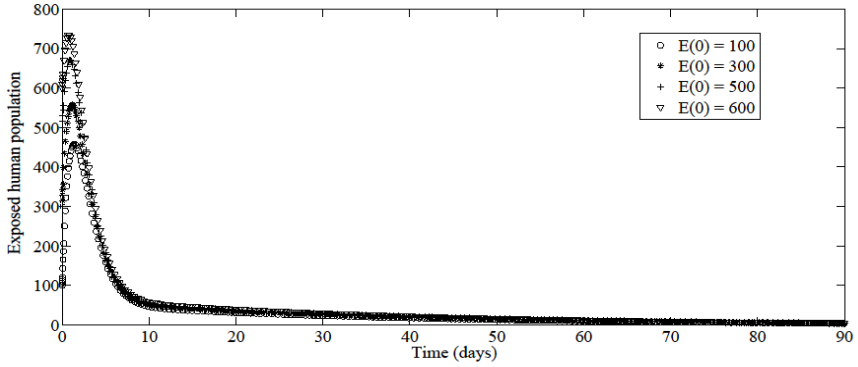


**Figure 4:** Disease-free equilibrium point for exposed human population.

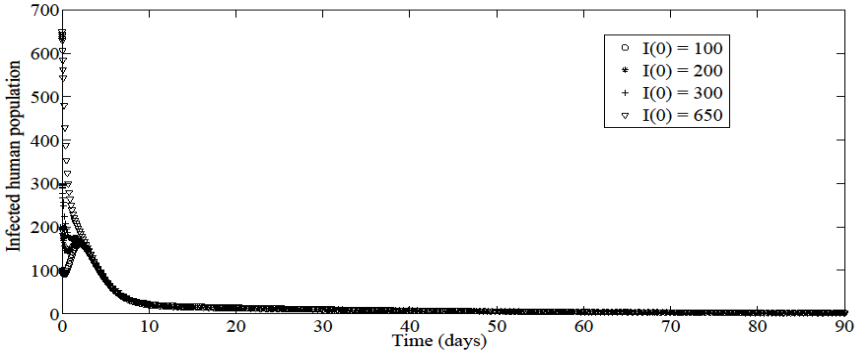


**Figure 5:** Equilibrium point of infected human at disease-free.

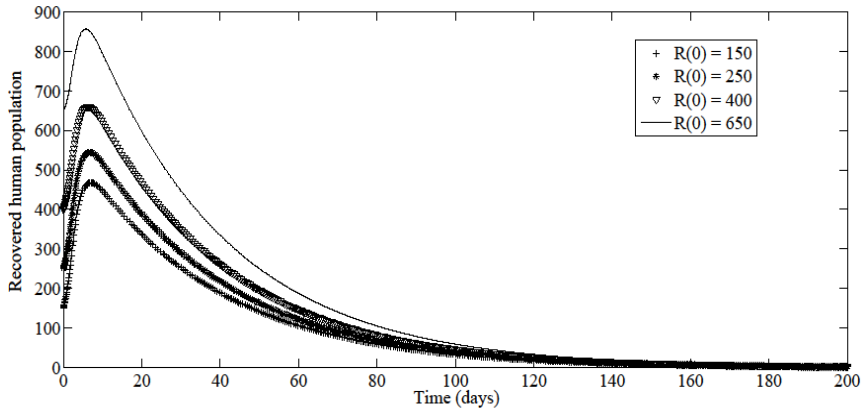


**Figure 6:** Disease-free equilibrium point for recovered human population.

Figure 7 shows the disease-free equilibrium point for the bacterial population. The exponential decrease of the bacterial population is because the inactive bacteria are removed from the population.

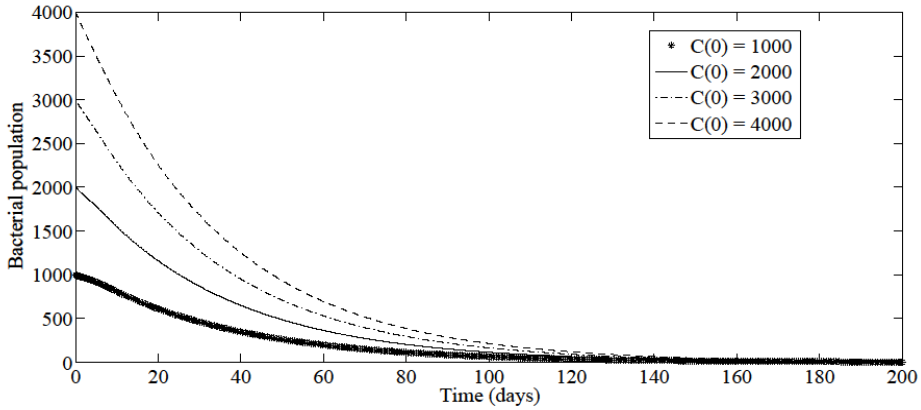


**Figure 7:** Disease-free equilibrium point for bacterial population.

Furthermore, Figures 8 and 9 show the effects of treatment on the infected and recovered human populations, respectively. The results indicate that the increase of treatment to the infected and recovered human populations has shown the great influence on reducing the number of infected individuals from the human population. Figure 10 shows the effect of practicing good hygiene on the environment. According to the results, the more the good hygiene reduces bacterial population from the environment. The results show that with no hygiene ($\phi = 0$), the population of bacteria becomes very high. Conversely, when $\phi = 1$, it means large population is practicing good hygiene and hence lowers the population of bacteria from the environment. This means that good environment hygiene on both food and water leads to safeguard the human population from the campylobacteriosis dynamics. Figure 11 shows the effect of public health education on the effective reproductive number. The more the public health education campaign to human population shows the significant reduction of the effective reproduction numbers.

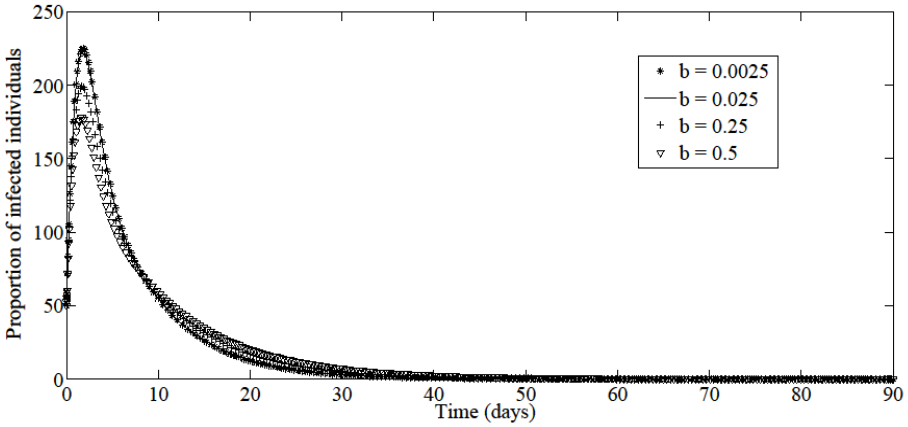


**Figure 8:** Effects of treatment on the infected class of human population.

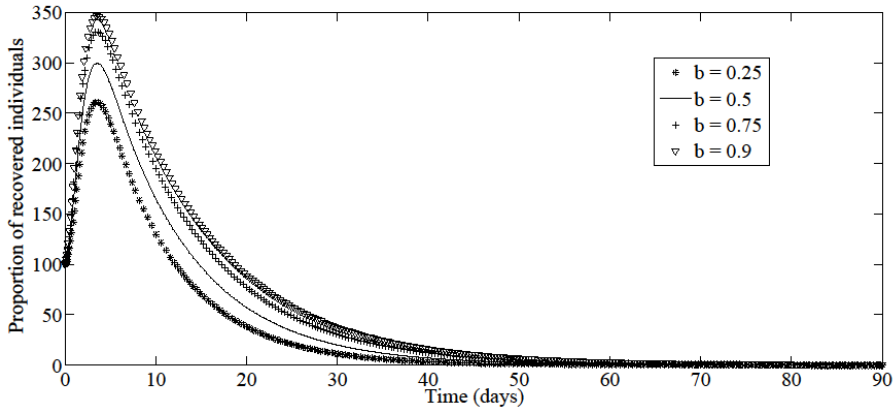


**Figure 9:** Effects of treatment on recovered class of human population.

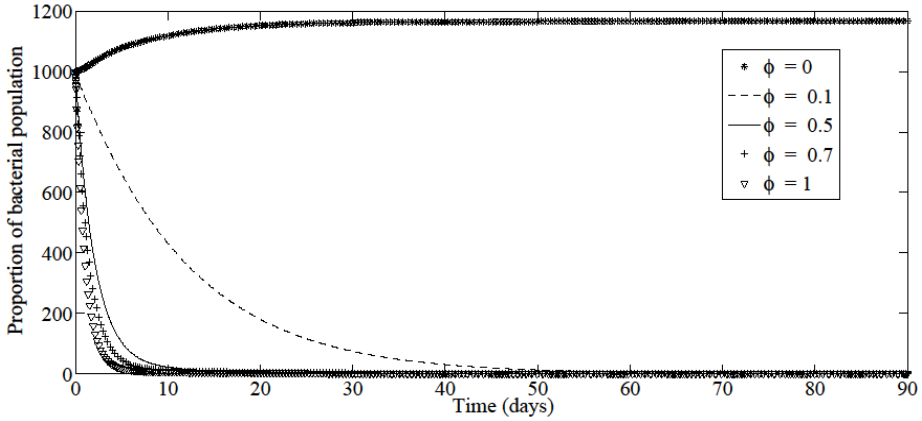


**Figure 10:** Effects of sanitation on bacterial population.

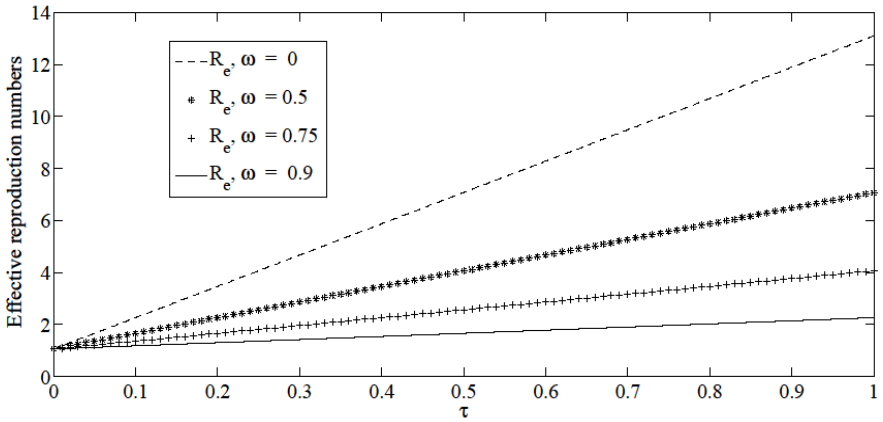


**Figure 11:** The effect of the rate of education campaign on effective reproduction number.

## Conclusion

In this article, we presented a deterministic model to study campylobacteriosis disease among the human population. The basic properties of the model show that the model is biologically meaningful and well-posed in the invariant region. Besides this, the effective reproduction number $R_e$ is computed and discussed. In the analysis of the model, both disease-free and endemic equilibrium points show that they are respectively locally and globally asymptotically stable. Also, public health education is numerically investigated to see its effects on the effective reproduction number. The results show that the public health education has a substantial influence on the control of campylobacteriosis disease among the human population. Hence, it is more important to deploy public health education strategies in a large population via different ways for reducing the spread of the disease among humans. Generally, the study concludes that public health awareness on the campylobacteriosis disease, treatment of the infected individuals, and practicing good hygiene reduce the chances of the disease to spread among the human population.

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## Conflict of Interests

Authors declare that there is no conflict of interest regarding this work.

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