Population dynamics of a mathematical model for Campylobacteriosis

T. T. Ashezua[1], M. T. Salemkaan[2] and S. A. Somma[3]
[1,2]Department of Mathematics, Joseph Sarwuan Tarka University Makurdi, Nigeria
[3]Department of Mathematics, Federal University of Technology, Minna, Nigeria

Corresponding author's email/Phone number: timothy.ashezua@uam.edu.ng, 07032230542

Abstract

The bacterium campylobacter is the cause of campylobacteriosis, a major cause of foodborne illness that goes by the most common name for diarrheal illnesses. This paper develops and analyzes a new mathematical model for campylobacteriosis. It is demonstrated that in cases where the corresponding reproduction number is smaller than unity, the model's disease-free equilibrium is both locally and globally stable. The numerical simulation results indicate that increasing the treatment rate for both symptomatic and asymptomatic disease-infected individuals resulted in a decrease in the number of asymptomatic and symptomatic individuals, respectively, and a rise in the population's number of recovered individuals.

Keywords: Reproduction number, stability, mathematical simulation, campylobacteriosis.

1.0 Introduction

The bacterium campylobacter is the source of campylobacteriosis, a major cause of foodborne illness that is thought to be the most prevalent indicator of diarrheal illnesses (WHO, 2020). The World Health Organization estimates that the burden of food-borne illnesses claims the lives of approximately 33 million healthy people and causes 1 in 10 people to become ill. The majority of illnesses resulting from eating unsafe food are diarrheal diseases, accounting for 550 million illnesses per year, of which 220 million are in children under the age of five (WHO, 2020). The majority of the time, campylobacter infections are mild (asymptomatic); however, in very young children, the elderly, and people with compromised immune systems, they can be fatal (symptomatic) (WHO, 2020, Health direct, 2024).

Common symptoms of campylobacter infection are; fever, cramping in the stomach, and diarrhea, which is frequently bloody. After diarrhea, nausea and vomiting are possible. Following infection, symptoms typically appear two to five days later and persist for approximately one week. Some people experience complications like arthritis, irritable bowel syndrome, and temporary paralysis (CDC, 2023). The majority

of people with campylobacter infections recover without the need for antibiotics, Centers for Disease Control and Prevention reported that (patients are advised to stay hydrated for the duration of their diarrhea), however severe cases may require antibiotic treatment. The groups most likely to experience severe cases of the disease are the elderly (65 years of age and older), pregnant women, and individuals with weakened immune systems (CDC, 2019).

Throughout the years, mathematical models of infectious diseases have offered helpful insights into the dynamics of infectious disease transmission, prevention, and control (Gulmel *et al*., 2018 are one example of this). However, only a small number of mathematical models have been created and applied to campylobateriosis in order to comprehend the disease's dynamics of transmission, management, and prevention; for instance, refer to (Rawson *et al.* 2019, Nyasagare et al. 2019, Osman *et al.* 2020, Chuma and Mussa, 2021). In 2019, Rawson and colleagues created and studied a mathematical model of the campylobacter in broiler flocks dynamics. In their investigations, the pathways of infection among co-housed birds were modeled using a system of stochastic differential equations. Nyasagare *et al.* (2019) developed and examined a mathematical model of campybacteriosis in animal and human populations using the S-I-R approach. Using an S-I-R model for both human and animal populations, Osman *et al.* (2020) developed and examined a mathematical model for campylobacteriosis using a modified finite difference method with optimal control. Chuma and Mussa (2021) created and examined an epidemic model that included sanitation control, treatment, and public health education to explain the dynamics of the campylobacteriosis disease. They did not divide the exposed class into asymptomatic and symptomatic classes in their work. A novel deterministic mathematical model for analyzing the dynamics of campylobacteriosis transmission in a population is presented in this work. The following presumptions form the basis of the model:

(i) The exposed class is split into asymptomatic and symptomatic classes, respectively. This is in line with the information obtained from (Osman *et al.* 2020, Health direct, 2024).

(ii) Asymptomatic individuals can recover without treatment and equally develop symptoms to progress to the symptomatic class while symptomatic patients might need antibiotics treatment (CDC, 2019).

In all the aforementioned mathematical models of Campylobacteriosis, none of them considered the assumptions (i) and (ii). Hence, in the present study, the exposed class have been split into the asymptomatic and symptomatic classes. Also, treatment of the asymptomatic and symptomatic patients are included as intervention strategies in our model to curtail the spread of the disease in the population.

2.0 Model Formulation

The total population at time t, denoted by $N(t)$, is divided into the human $(N_h(t))$ and animal $(N_v(t))$ populations. The total population of humans 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 $(I_a(t))$ humans. Also, the total animal population is sub-divided into the susceptible $(S_v(t))$, infected $(I_v(t))$ and recovered $(R_v(t))$ sub-population. Thus,

$$\begin{aligned} N(t) &= N_h(t) + N_v(t) \\ N_h(t) &= S_h(t) + E_h(t) + I_a(t) + I_s(t) + R_h(t), \\ N_v(t) &= S_v(t) + I_v(t) + R_v(t). \end{aligned} \tag{2.1}$$

The susceptible population (for both human and animals) are recruited through immigration at rates $\Lambda_h(\Lambda_v)$, respectively. Humans in E_h class progresses to class 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. Humans and other animals that are susceptible to the disease can contract campylobacteriosis by eating contaminated food or water or by coming into close contact with infected humans or animals (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

$$\beta_1(I_a + I_s + I_v), \tag{2.2}$$

and

$$\beta_2(I_a + I_s + I_v). \tag{2.3}$$

Animals recover from campylobacteriosis at a rate γ_3.

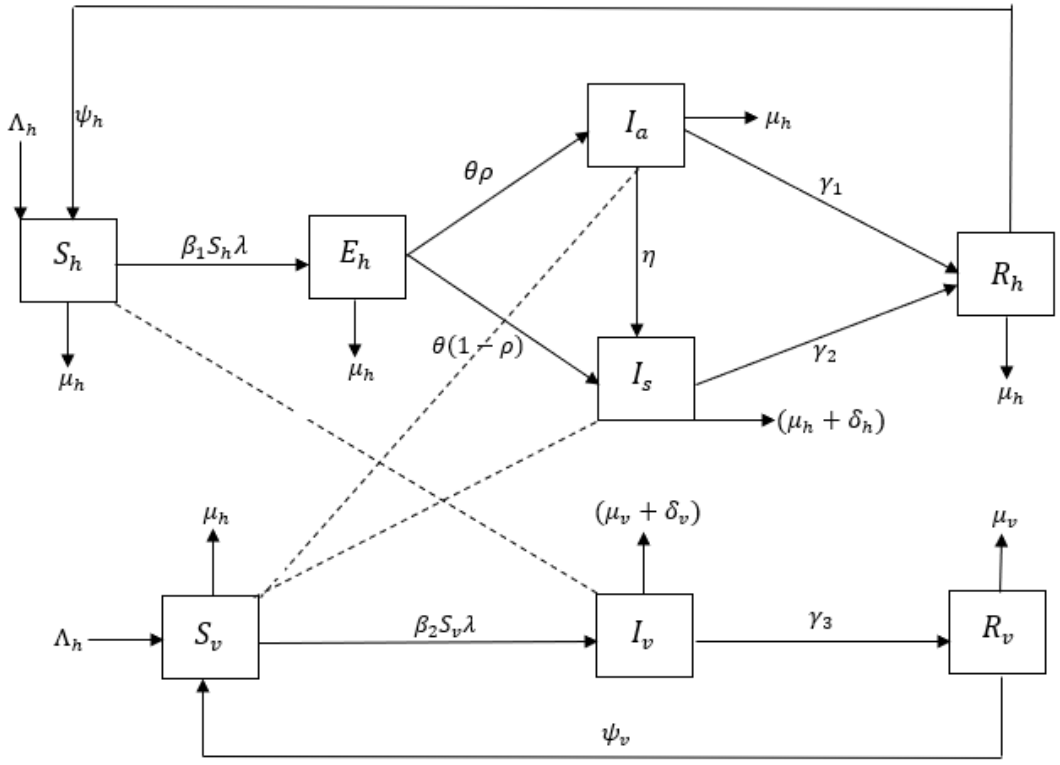

Figure 1. Schematic diagram for model (2.4).

Table 1: Description of variables and parameters in the Campylobacteriosis model (2.4).

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 rate into the susceptible human (animal) compartments
$\beta_1(\beta_2)$	Infection rates
θ	Progression rate
ρ	Proportion of exposed humans moving to class I_a

η	Progression rate from I_a to I_s
$\gamma_1(\gamma_2)$	Treatment rates for the human
γ_3	Treatment rate for the animals
$\mu_h(\mu_v)$	Human (animal) natural death rate
$\psi_h(\psi_v)$	Loss of immunity
$\delta_h(\delta_v)$	Human (animal) disease-induced death rate
λ	Force of infection

When all of these definitions and presumptions are combined, the system of differential equations in (2.4) yields the new Campylobacteriosis model. Figure 1 displays the model's flow diagram, and Table 1 lists the variables in the model.

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= \Lambda_h - \beta_1\lambda S_h - \mu_h S_h + \psi_h R_h,\\
\frac{dE_h(t)}{dt} &= \beta_1\lambda S_h - [\theta\rho + \theta(1-\rho) + \mu_h]E_h,\\
\frac{dI_a(t)}{dt} &= \theta\rho E_h - (\eta + \gamma_1 + \mu_h)I_a,\\
\frac{dI_s(t)}{dt} &= \theta(1-\rho)E_h + \eta I_a - (\gamma_2 + \mu_h + \delta_h)I_s, \qquad (2.4)\\
\frac{dR_h(t)}{dt} &= \gamma_1 I_a + \gamma_2 I_s - (\mu_h + \psi_h)R_h,\\
\frac{dS_v(t)}{dt} &= \Lambda_v - \beta_2\lambda S_v - \mu_v S_v + \psi_v R_v,\\
\frac{dI_v(t)}{dt} &= \beta_2\lambda S_v - (\gamma_3 + \mu_v + \delta_v)I_v,\\
\frac{dR_v(t)}{dt} &= \gamma_3 I_v - (\mu_v + \psi_v)R_v.
\end{aligned}$$

where the forces of infection for human and animals are as given in equations (2.2) and (2.3), respectively.

Theorem 2.1.: When starting with positive data, every solution in the model (2.4) stays positive over time. Additionally, the model is a dynamic system on the area that has, $\Omega = \Omega_1 \cup \Omega_2 \subset \Re_+^5 \times \Re_+^3$ with,

$$\Omega_1 = \left\{ \left(S_h, E_h, I_a, I_s, R_h\right) : S_h + E_h + I_a + I_s + R_h = N_h \le \frac{\Lambda_h}{\mu_h} \right\},$$
$$\Omega_2 = \left\{ \left(S_v, I_v, R_v\right) : S_v + I_v + R_v = N_v \le \frac{\Lambda_v}{\mu_v} \right\}, \tag{2.5}$$

Proof. Following similar approach as in Gumel *et al.,* (2018), it is easy to see that the following first-order inequality equations follow from the equations for humans (susceptible individuals) and animals (susceptible animals) in model (2.4):

$$\frac{dS_h}{dt} + \left(\beta_1 \lambda + \mu_h\right) S_h > 0, \text{ and } \frac{dS_v}{dt} + \left(\beta_2 \lambda + \mu_v\right) S_v > 0$$

Applying integrating factor to the inequalities

$$\alpha_{S_h}(t) = \exp^{\int [\beta_1 \lambda(\tau) + \mu_h] d\tau}, \alpha_{S_v}(t) = \exp^{\int [\beta_2 \lambda(\tau) + \mu_v] d\tau},$$

and observing that

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

then integrating from 0 to gives $S_h(t) \ge 0$ and $S_v(t) \ge 0$ at all times, respectively, with respect to time. Nevertheless, the remaining equations are not amenable to this direct method. However, the conservation law is obtained by summing the model's first five and final three equations (2.4).

$$\left.\begin{aligned} \frac{dN_h}{dt} &= \Lambda_h - \mu_h N_h - \delta_h I_s \\ \frac{dN_v}{dt} &= \Lambda_v - \mu_v N_v - \delta_a I_v \end{aligned}\right\} \tag{2.6}$$

Thus, the general a priori estimates below can be demonstrated to hold using a standard comparison theorem.

$$\left.\begin{aligned} 0 \le N_h(t) &\le N_h(0) \exp^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}\left(1 - \exp^{-\mu_h t}\right) \\ 0 \le N_v(t) &\le N_v(0) \exp^{-\mu_v t} + \frac{\Lambda_v}{\mu_v}\left(1 - \exp^{-\mu_v t}\right) \end{aligned}\right\} \tag{2.7}$$

We determine that there is only one global solution in the domain Ω. The right-hand side of the model (2.4) is locally Lipschitz (Stuart and Humphties 1998). Consequently, the dynamical system on Ω in model (2.4). Conversely, if a solution is found outside of the area Ω, that is, $N_h \le \frac{\Lambda_h}{\mu_h}$ and $N_v \le \frac{\Lambda_v}{\mu_v}$, then the conservation law mentioned above implies that $\frac{dN_h}{dt} \le 0$ and $\frac{dN_v}{dt} \le 0$. Thus, it can be seen from the estimates above that $N_h(t)$ tends to $\frac{\Lambda_h}{\mu_h}$ and $N_v(t)$ tends to $\frac{\Lambda_v}{\mu_v}$ as $t \to \infty$.. As a result, the region Ω is interesting.

3 Mathematical Analysis

3.1 Asymptotic Stability of Disease-free Equilibrium (DFE)

The DFE of the model (2.4) is given by

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

Using model (2.4) and an operator method for the next generation (van den Driessche and Watmough, 2002), the local stability of E_1 will be established. It follows that matrices F and V, the notation found in van den Driessche and Watmough (2002) is used for the new infection terms and the remaining transition terms, respectively.

$$F = \begin{pmatrix} 0 & \frac{\beta_1 \Lambda_h}{\mu_h} & \frac{\beta_1 \Lambda_h}{\mu_h} & \frac{\beta_1 \Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2 \Lambda_v}{\mu_v} & \frac{\beta_2 \Lambda_v}{\mu_v} & \frac{\beta_2 \Lambda_v}{\mu_v} \end{pmatrix} \text{ and } V = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ 0 & k_2 & 0 & 0 \\ -k_3 & -\eta & k_4 & 0 \\ 0 & 0 & 0 & k_6 \end{pmatrix},$$

where,

$$k_1 = [\theta\rho + \theta(1-\rho) + \mu_h], k_2 = (\eta + \gamma_2 + \mu_h), k_3 = \theta(1-\rho), k_4 = (\gamma_4 + \mu_h + \delta_h), \\ k_5 = (\mu_h + \psi_h), k_6 = (\gamma_3 + \mu_v + \delta_v), k_7 = (\mu_v + \psi_v). \quad (3.1).$$

Hence, the effective reproduction number of the model (2.4), denoted by R_c, is given by

$$R_c = \frac{\Lambda_h k_3 k_6 \mu_v \beta_1 + \Lambda_v k_1 k_4 \mu_h \beta_2}{\mu_h \mu_v k_1 k_4 k_6} \tag{3.2}$$

The following findings are derived from the Theorem 2 Van den Driessche and Watmough's (2002)

Lemma 3.1: The DFE (E_1) of the model (2.4) is locally asymptotically stable whenever $R_c < 1$ and unstable if otherwise.

The threshold quantity R_c measures the average number of new Campylobacteriosis infections generated by an index case in a completely susceptible population (van den Driessche and Watmough, 2002). Specifically, R_c denotes the mean quantity of newly acquired Campylobacteriosis infections within the human (animal) population, resulting from the introduction of a single infected individual into a fully susceptible human (animal) population. Lemma 3.1's epidemiological implication is that, if the initial sizes of the model's subpopulation are within the DFE's (E_1) basin of attraction, campylobacteriosis can be eradicated from the population when R_c is less than unity. As a result, a small number of humans or animals carrying the infection may enter the community; this will not cause significant outbreaks of the disease, and it will eventually go extinct. It is vital to demonstrate that the DFE is globally-asymptotically stable (GAS) if $R_c < 1$ and the initial subpopulation sizes do not affect the eradication of campylobacteriosis.

Theorem 3.2. The DFE (E_1) of the model (2.4) is globally asymptotically stable in Ω whenever $R_c \leq 1$.

Proof. The proof of Theorem 3.2 will be established using the Theorem of comparison (Lakshmikantham *et al.,* 1989). The following is the matrix-vector form for the equations on the model's (2.4) infected components:

$$\frac{dL(t)}{dt} = \left[(F - V) - \left(1 - \frac{S_h}{N_h}\right) H_1 - \left(1 - \frac{S_v}{N_v}\right) H_2 \right] L(t), \tag{3.3}$$

where $L(t) = (E_h(t), I_a(t), I_s(t), R_h(t), I_v(t), R_v(t))^T$ and the matrices F and V are given in section 3. Furthermore,

$$H_1 = \begin{pmatrix} \frac{\beta_1 \Lambda_h}{\mu_h} & \frac{\beta_1 \Lambda_h}{\mu_h} & \frac{\beta_1 \Lambda_h}{\mu_h} & \frac{\beta_1 \Lambda_h}{\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$H_2 = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2 \Lambda_v}{\mu_v} & \frac{\beta_2 \Lambda_v}{\mu_v} & \frac{\beta_2 \Lambda_v}{\mu_v} \end{pmatrix}.$$

Since H_1 and H_2 are nonnegative matrices and $S_h(t) \le N_h(t)$ and $S_v(t) \le N_v(t)$ in Ω, it follows that

$$\frac{dL(t)}{dt} \le [(F - V)]L(t) \tag{3.4}$$

The differential inequality system (3.4) is stable whenever $R_c < 1$, based on the fact that all of the matrix eigenvalues have negative real parts. Thus, according to comparison theorem of (Lakshmikantham *et al* 1989).

$$\lim_{t\to\infty}(E_h(t), I_a(t), I_s(t), R_h(t), I_v(t), R_v(t)) = (0,0,0,0,0,0). \tag{3.5}$$

It can be shown by substituting (3.5) into (2.1) that $S_h \to \frac{\Lambda_h}{\mu_h}$ and $S_v \to \frac{\Lambda_v}{\mu_v}$ as $t \to \infty$. thus,

$$\lim_{t\to\infty}(S_h(t), E_h(t), I_a(t), I_s(t), R_h(t), S_v(t), I_v(t), R_v(t) = (\tfrac{\Lambda_h}{\mu_h}, 0,0,0,0, \tfrac{\Lambda_h}{\mu_h}, 0,0) = E_1^*. \tag{3.6}$$

Hence, every solution to the equation of the model (2.4) and initial conditions in Ω, approaches the DFE (E_1) as $t \to \infty$ whenever $R_c < 1$ .The epidemiological implication of Theorem 3.2 is that irrespective of the number of infectives in the population, if the threshold quantity R_c can be kept below unity, campylobacteriosis will be effectively controlled in the community

4 Numerical Simulation

In this section, numerical simulations for the transmission dynamics of campylobacteriosis are performed on model (2.4) with the parameter values from Table 2 and the assumed initial data. The model (2.4) is solved numerically using MATLAB ODE45 solver. In Figure 2, an increase in the progression rate of the asymptomatic individuals to the symptomatic class experienced an increase in the population of the symptomatic individuals. In Figures 3 and 4 (as expected), an increase in the treatment rate of the asymptomatic and symptomatic individuals led to a decrease in the number of infected (both asymptomatic and symptomatic) individuals in the population. This suggests that if treatment of infected (both asymptomatic and symptomatic) individuals is deployed early enough, it will lead to the timely eradication of the disease in the population.

Table 2. The parameter values of the model (2.4) per year.

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

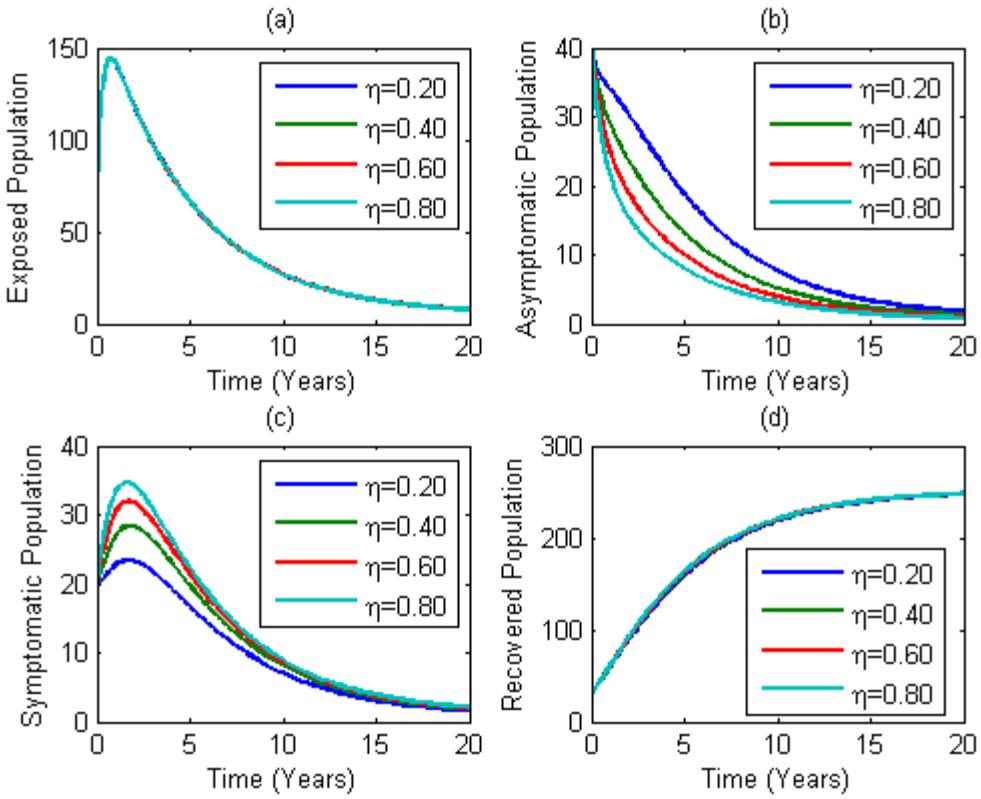

Figure 2. Simulation of model (2.4) showing the population of exposed, asymptomatic, symptomatic and recovered individuals. Here, the progression rate from the asymptomatic to symptomatic class η is varied from 0.20 to 0.80.

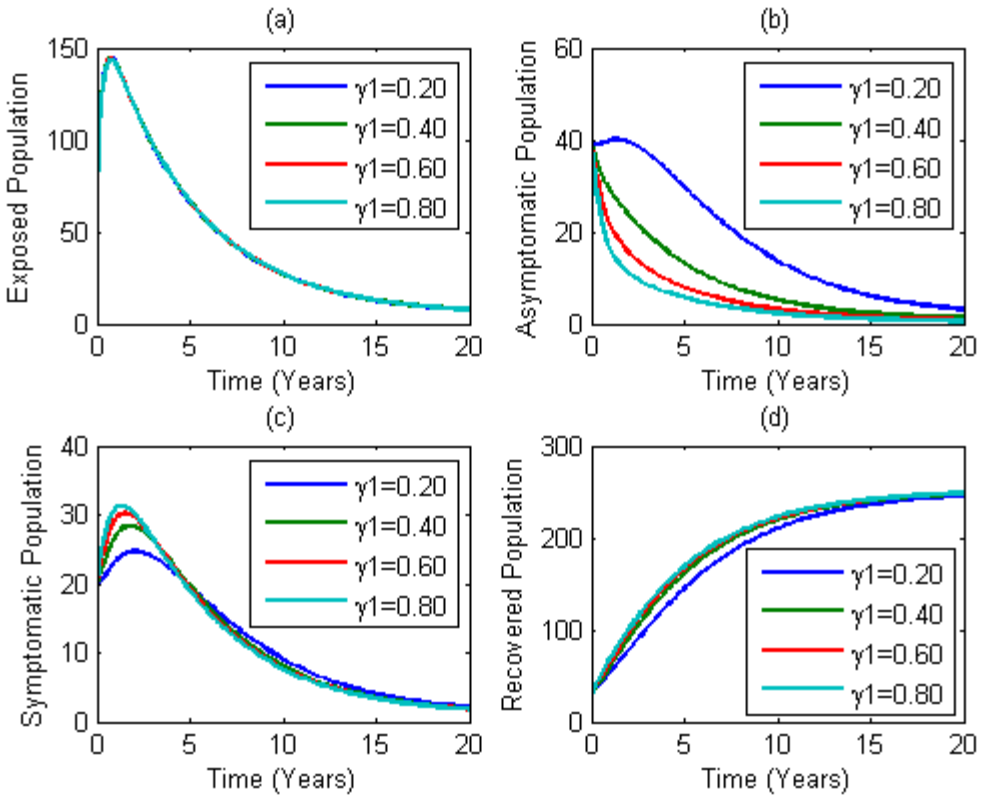

Figure 3. Simulation of model (2.4) showing the population of exposed, asymptomatic, symptomatic and recovered individuals. Here, the treatment rate of the asymptomatic individuals, γ_1 is varied from 0.20 to 0.80.

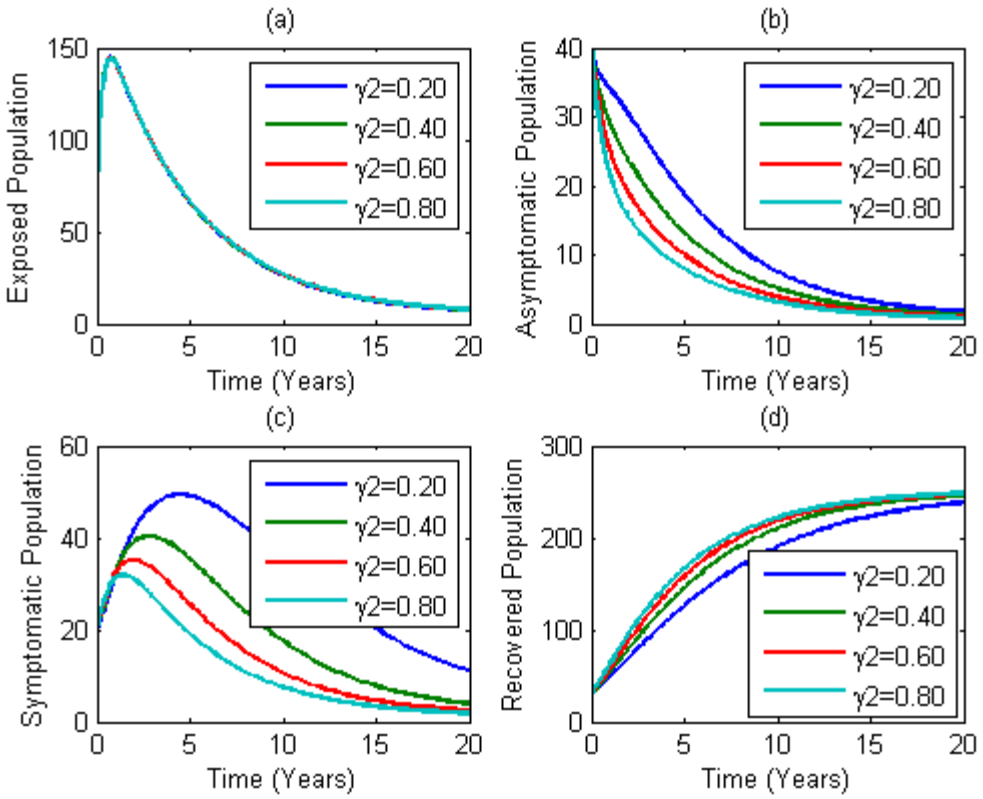

Figure 4. Simulation of model (2.4) showing the population of exposed, asymptomatic, symptomatic and recovered individuals. Here, the treatment rate of the symptomatic individuals, γ_2 is varied from 0.20 to 0.80.

5 Conclusion

In order to better understand the dynamics of Campylobacteriosis infection transmission in a population, this study offers a novel deterministic mathematical model with treatment as a control strategy. It is shown that for model (2.4), if the corresponding reproduction number is less than unity, the disease-free equilibrium (DFE) is locally asymptotically stable. The disease-free equilibrium was discovered to be globally asymptotically stable whenever the corresponding reproduction number is less than unity using the comparison theorem. The numerical simulation results show that a decrease in the population of asymptomatic individuals and an increase in the population of recovered individuals occurred when the treatment rate for both symptomatic and asymptomatic disease-infected individuals was increased.

Few mathematical models have been developed to date to investigate the dynamics of campylobacteriosis transmission as well as its prevention and control. Therefore, additional investigation of the

Campylobacteriosis model in this work is required to determine model parameters related to the reproduction number that are essential to containing the disease's spread. In light of this, we suggest further analysis our model as follows:

(i) Sensitivity analysis of the model parameters associated with the reproduction number be carried out.
(ii) Further theoretical results, such as the type of bifurcation the model (2.4) can exhibit should be explored.

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