

Research Article

Modelling the Dynamics of Campylobacteriosis Using Nonstandard Finite Difference Approach with Optimal Control

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Campylobacter genus is the bacteria responsible for campylobacteriosis infections, and it is the commonest cause of gastroenteritis in adults and infants. The disease is hyperendemic in children in most parts of developing countries. It is a zoonotic disease that can be contracted via direct contact, food, and water. In this paper, we formulated a deterministic model for Campylobacteriosis as a zoonotic disease with optimal control and to determine the best control measure. The nonstandard finite difference scheme was used for the model analysis. The disease-free equilibrium of the scheme in its explicit form was determined, and it was shown to be both locally and globally asymptotically stable. The campylobacteriosis model was extended to optimal control using prevention of susceptible humans contracting the disease and treatment of infected humans and animals. The objective function was optimised, and it was established that combining prevention of susceptible humans and treatment of infected animals was the effective control measure in combating campylobacteriosis infections. An analysis of the effects of contact between susceptible and infected animals as well as susceptible and infected humans was conducted. It showed an increase in infected animals and humans whenever the contact rate increases and decreases otherwise. Biologically, it implies that campylobacteriosis infections can be controlled by ensuring that interactions among susceptible humans, infected animals, and infected humans is reduced to the barest minimum.

1. Introduction

Campylobacter genus is the bacteria responsible for campylobacteriosis infections. This is solely the commonest cause of gastroenteritis in adults and mostly infants [1]. The Campylobacter bacteria have been confirmed as the leading cause of diarrhea in the United States of America. Campylobacteriosis is mostly hyperendemic in children in most parts of developing countries. Campylobacteriosis can be spread or contracted through the fecal-oral path. It is a zoonotic disease that can be contracted via direct contact, food, and water. The disease is zoonotic in nature and hence can be spread from animals to humans and also from humans to humans [2].

A campylobacteriosis-infected person is usually asymptomatic at the incubation period, that is, between one and three days of infection. Diarrhea, fever, and abdominal cramps

are usually the commonest symptoms of the disease. Symptoms of campylobacteriosis can last for at least five to eight days of infections. Children in developing countries mostly show symptoms of campylobacteriosis infections while adults rarely show any symptoms of infections. But on the contrary, the infection is less common in the developed world [1].

Symptomatic persons can infect others directly and contaminate water and food during the infectious period of campylobacteriosis. The mode of infection of campylobacteriosis is mostly through food, water, and milk that has been infected by contaminated feces which has been poorly treated. However, water contamination is basically via water fowl feces, sewage, and farm animal manure. On the other hand, human contamination is via leaked septic tanks into groundwater supply that is poorly disinfected or not disinfected at all. The disease is mostly foodborne and waterborne illness but can also be spread

through direct contact with infected humans or animals via the fecal-oral path of transmission. But human-to-human spread is usually uncommon [3].

Understanding the spread dynamics of campylobacteriosis at policy and implementation levels of public health is necessary to design effective optimal control and cost-effective strategies at prevention levels. Deterministic models enhance the general understanding of the disease spread by the provision of a theoretical frame which underlines factors that accounts for the spread and control of diseases [4, 5].

The concept of deterministic modelling involves the process of constructing, testing, and validating models. These models are real representations of natural phenomena of systems or hypothesis in a mathematical perspective [6, 7].

Generally, the intended use of a deterministic model is paramount in guiding the development of the model since the model structure has to adequately address its objective. Hence, understanding the mechanism and causes of patterns present in an observed data is usually an objective that initiates a deterministic modelling process [8, 9]. Moreover, epidemiological models explain dynamics of infections and determine the best optimal control strategies and the most cost effective among these strategies [10, 11]. However, authors in [12–14] proposed and formulated models that attempts to explain this hidden and existing phenomena.

2. Model Formulation and Description

The model diagram in Figure 1 shows the transmission dynamics of campylobacteriosis in humans and animals. This diagram is significant as it gives an overview of the disease transmission pattern in humans and animals.

We divided the model into two parts, the total human and animal populations. These populations at any time, t are also divided into six subcompartments with respect to their disease status in the system. The total human population, represented by $N_h(t)$, is divided into subpopulations of susceptible humans $S_h(t)$, infected humans $I_h(t)$, and recovered humans $R_h(t)$. Susceptible humans are recruited through immigration into the population at a rate Λ_h . They are infected with campylobacteriosis through ingestion of contaminated water, foods, and direct contact with infected animals and humans at a rate $(I_v + I_h)\beta$. Infected humans recover from campylobacteriosis at a rate γ . Campylobacteriosis-related death rate is given by δ_h . Recovered individuals may lose immunity and return to the susceptible group at a rate σ_h . Campylobacteriosis natural death rate for all human compartments is μ_h . Susceptible animals S_v are recruited through immigration at a rate Λ_v . Animals can be infected with campylobacteriosis through ingestion of contaminated food, water, and contact with infected animals at a rate $(I_v + I_h)\lambda$. Susceptible and infected animal natural death rate is μ_v . Infected animal death rate as a result of campylobacteriosis is δ_v , and animals may recover at a rate α . Animals may lose immunity at a rate σ_v .

Hence, total human population is

$$N_h(t) = S_h(t) + I_h(t) + R_h(t). \quad (1)$$

Total animal population, $N_v(t)$, is divided into subpopu-

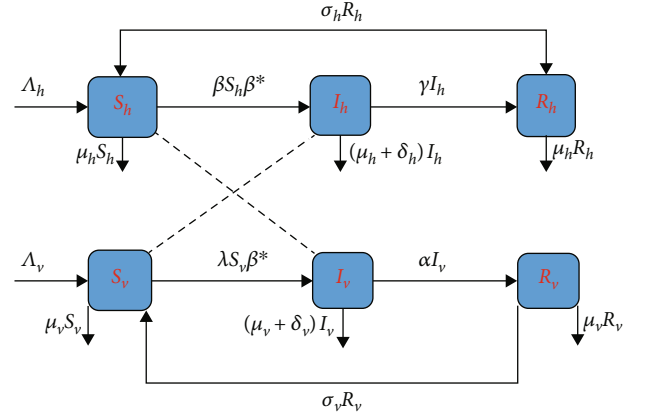


FIGURE 1: Model flow diagram showing the transmission dynamics of the disease.

lations of susceptible animals $S_v(t)$, infectious animals $I_v(t)$, and recovered animals $R_v(t)$.

Hence, total animal population is

$$N_v(t) = S_v(t) + I_v(t) + R_v(t). \quad (2)$$

System of equations obtained from the model in Figure 1 are as follows:

$$\left. \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h, \\ \frac{dI_h}{dt} &= \beta_m^* \beta S_h - \gamma I_h - (\mu_h + \delta_h) I_h, \\ \frac{dR_h}{dt} &= \gamma I_h - (\sigma_h + \mu_h) R_h, \\ \frac{dS_v}{dt} &= \Lambda_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v, \\ \frac{dI_v}{dt} &= \beta_m^* \lambda S_v - \alpha I_v - (\mu_v + \delta_v) I_v, \\ \frac{dR_v}{dt} &= \alpha I_v - (\sigma_v + \mu_v) R_v, \end{aligned} \right\} \quad (3)$$

where $\beta_m^* = I_h + I_v$.

3. Model Analysis

3.1. Positivity and Boundedness of Solutions. The solution of the system in (3) is a function of the form

$$X : t \in \mathbb{J} \subset \mathbb{R} \rightarrow X(t) = \begin{pmatrix} S_h(t) \\ I_h(t) \\ R_h(t) \\ S_v(t) \\ I_v(t) \\ R_v(t) \end{pmatrix} \in \mathbb{R}^6. \quad (4)$$

Considering,

$$F : X \in \mathbb{R}^6 \rightarrow F(X) \in \mathbb{R}^6, \quad (5)$$

where

$$F(X) = \left\{ \begin{array}{l} \frac{dS_h}{dt} = \Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h, \\ \frac{dI_h}{dt} = \beta_m^* \beta S_h - \gamma I_h - (\mu_h + \delta_h) I_h, \\ \frac{dR_h}{dt} = \gamma I_h - (\sigma_h + \mu_h) R_h, \\ \frac{dS_v}{dt} = \Lambda_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v, \\ \frac{dI_v}{dt} = \beta_m^* \lambda S_v - \alpha I_v - (\mu_v + \delta_v) I_v, \\ \frac{dR_v}{dt} = \alpha I_v - (\sigma_v + \mu_v) R_v. \end{array} \right. \quad (6)$$

Hence,

$$\frac{dX}{dt} = F(X), \quad (7)$$

$$X(0) = X_0 = \left(S_{h(0)}, I_{h(0)}, R_{h(0)}, S_{v(0)}, I_{v(0)}, R_{v(0)} \right)^T.$$

Based on the existence and uniqueness theorem, F is C^1 . Hence, \exists a unique global solution of the initial value problem of (3) and this solution should be nonnegative whenever its initial conditions are nonnegative.

4. Nonstandard Finite Difference Scheme

This is basically a numerical scheme with step size Δt that is usually used in the approximation of solution $X(t_k)$ of autonomous system of differential equations of the form

$$\frac{dX}{dt} = F(X), \quad (8)$$

subject to

$$X(0) = X_0, \quad (9)$$

where F is C^1 usually of the form

$$D_{\Delta t}(X_k) = F_{\Delta t}(X_k), \quad (10)$$

where

$$\left. \begin{array}{l} D_{\Delta t}(X_k) \approx \frac{dX(t_k)}{dt}, \\ X_k \approx X(t_k), \\ F_{\Delta t}(X_k) \approx F(X_k), \\ t_k \approx t_0 + k\Delta t. \end{array} \right\} \quad (11)$$

The scheme

$$D_{\Delta t}(X_k) = F_{\Delta t}(X_k). \quad (12)$$

Definition 1. The scheme (12) can be referred to as a nonstandard finite difference scheme when it at least satisfies the following conditions:

- (i) $D_{\Delta t}(X_k) = (X_{k+1} - \psi X_k) / \varphi(\Delta t)$, where ψ and φ are positive functions which depend on parameters of the differential equations, step size, (Δt) and satisfy

$$\left. \begin{array}{l} \psi(\Delta t) = 1 + \mathcal{O}(\Delta t), \\ \varphi(\Delta t) = \Delta t + \mathcal{O}(\Delta t^2) \end{array} \right\} \quad (13)$$

- (ii) $F_{\Delta t}(X_k) = g(X_k, X_{k+1}, \Delta t)$, where g denotes an approximation of the nonlocal right hand side of the system

Definition 2. The nonstandard finite difference scheme is called elementary stable, if, for any value of the step size, its only fixed points are those of the original differential system, the linear stability properties of each fixed points being the same for both the differential system and the discrete scheme.

Based on the definition of the nonstandard finite difference (NSFD) scheme and the rules governing its construction in [15–18], the NSFD scheme for the system of (3) is given by

$$\left. \begin{array}{l} \frac{S_h^{n+1} - S_h^n}{\varphi_1(\Delta t)} = \Lambda_h - \beta(I_h^n + I_v^n)S_h^{n+1} - \mu_h S_h^{n+1} + \sigma_h R_h^{n+1}, \\ \frac{I_h^{n+1} - I_h^n}{\varphi_2(\Delta t)} = \beta(I_h^n + I_v^n)S_h^{n+1} - \gamma I_h^{n+1} - (\mu_h + \delta_h)I_h^{n+1}, \\ \frac{R_h^{n+1} - R_h^n}{\varphi_3(\Delta t)} = \gamma I_h^{n+1} - (\sigma_h + \mu_h)R_h^{n+1}, \\ \frac{S_v^{n+1} - S_v^n}{\varphi_4(\Delta t)} = \Lambda_v - \lambda(I_h^n + I_v^n)S_v^{n+1} - \mu_v S_v^{n+1} + \sigma_v R_v^{n+1}, \\ \frac{I_v^{n+1} - I_v^n}{\varphi_5(\Delta t)} = \lambda(I_h^n + I_v^n)S_v^{n+1} - \alpha I_v^{n+1} - (\mu_v + \delta_v)I_v^{n+1}, \\ \frac{R_v^{n+1} - R_v^n}{\varphi_6(\Delta t)} = \alpha I_v^{n+1} - (\sigma_v + \mu_v)R_v^{n+1}, \end{array} \right\} \quad (14)$$

where

$$\left. \begin{aligned} \varphi_j(\Delta t, k_j^*) &= \frac{1 - e^{-k_j^* \Delta t}}{k_j^*}, \\ k_j^* &= \max \{|\gamma_i|\}, \\ j &= 1, 2, 3, \dots, 6, \\ i &= 1, 2, 3, \dots, 6, \end{aligned} \right\} \quad (15)$$

with

$$\left. \begin{aligned} \gamma_i &= \partial f / \partial x_i |_{x=\bar{x}}, \\ f(\bar{x}) &= 0, \end{aligned} \right\} \quad (16)$$

where;

$$\left. \begin{aligned} \varphi_1(\Delta t) &= \left(\frac{1 - e^{-\mu_h \Delta t}}{\mu_h}, \frac{-\beta \Lambda_h}{\mu_h}, \sigma_h, 0, \frac{-\beta \Lambda_h}{\mu_h}, 0 \right), \\ \varphi_2(\Delta t) &= \left(\frac{1 - e^{-|(\beta \Lambda_h / \mu_h) - \gamma - \mu_h - \delta_h| \Delta t}}{|(\beta \Lambda_h / \mu_h) - \gamma - \mu_h - \delta_h|}, \frac{-\beta \Lambda_h}{\mu_h}, 0, 0, \frac{-\beta \Lambda_h}{\mu_h}, 0 \right), \\ \varphi_3(\Delta t) &= \left(\frac{1 - e^{-(\sigma_h + \mu_h) \Delta t}}{(\sigma_h + \mu_h)}, \gamma, -\sigma_h - \mu_h, 0, 0, 0 \right), \\ \varphi_4(\Delta t) &= \left(\frac{1 - e^{-\mu_v \Delta t}}{\mu_v}, 0, -\frac{\lambda \Lambda_v}{\mu_v}, -\mu_v, -\frac{\lambda \Lambda_v}{\mu_v}, \sigma_v \right), \\ \varphi_5(\Delta t) &= \left(\frac{1 - e^{-|(\lambda \Lambda_v / \mu_v) - \alpha - \mu_v - \delta_v| \Delta t}}{|(\lambda \Lambda_v / \mu_v) - \alpha - \mu_v - \delta_v|}, 0, \frac{\lambda \Lambda_v}{\mu_v}, 0, 0, \frac{\lambda \Lambda_v}{\mu_v} - \alpha - \mu_v - \delta_v \right), \\ \varphi_6(\Delta t) &= \left(\frac{1 - e^{-(\sigma_v + \mu_v) \Delta t}}{(\sigma_v + \mu_v)}, 0, 0, 0, \alpha, \sigma_v - \mu_v \right), \end{aligned} \right\} \quad (17)$$

where,

The scheme in its explicit form is given by

$$\left. \begin{aligned} \varphi_1(\Delta t) &= \frac{1 - e^{-\mu_h \Delta t}}{\mu_h}, \\ \varphi_2(\Delta t) &= \frac{1 - e^{-|(\beta \Lambda_h / \mu_h) - \gamma - \mu_h - \delta_h| \Delta t}}{|(\beta \Lambda_h / \mu_h) - \gamma - \mu_h - \delta_h|}, \\ \varphi_3(\Delta t) &= \frac{1 - e^{-(\sigma_h + \mu_h) \Delta t}}{(\sigma_h + \mu_h)}, \\ \varphi_4(\Delta t) &= \frac{1 - e^{-\mu_v \Delta t}}{\mu_v}, \\ \varphi_5(\Delta t) &= \frac{1 - e^{-|(\lambda \Lambda_v / \mu_v) - \alpha - \mu_v - \delta_v| \Delta t}}{|(\lambda \Lambda_v / \mu_v) - \alpha - \mu_v - \delta_v|}, \\ \varphi_6(\Delta t) &= \frac{1 - e^{-(\sigma_v + \mu_v) \Delta t}}{(\sigma_v + \mu_v)}. \end{aligned} \right\} \quad (18)$$

$$\left. \begin{aligned} S_h^{n+1} &= \frac{(\Lambda_h + \sigma_h R_h^{n+1}) \varphi_1(\Delta t) + S_h^n}{1 + (\beta I_h^n + \beta I_h^n + \mu_h) \varphi_1(\Delta t)}, \\ I_h^{n+1} &= \frac{(\beta I_h^n S_h^{n+1} + \beta I_h^n S_h^{n+1}) \varphi_2(\Delta t) + I_h^n}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)}, \\ R_h^{n+1} &= \frac{\gamma I_h^{n+1} \varphi_3(\Delta t) + R_h^n}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)}, \\ S_v^{n+1} &= \frac{(\Lambda_v + \sigma_v R_v^{n+1}) \varphi_4(\Delta t) + S_v^n}{1 + (\lambda I_h^n + \lambda I_h^n + \mu_v) \varphi_4(\Delta t)}, \\ I_v^{n+1} &= \frac{(\lambda I_h^n S_v^{n+1} + \lambda I_v^n S_v^{n+1}) \varphi_5(\Delta t) + I_v^n}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)}, \\ R_v^{n+1} &= \frac{\alpha I_v^{n+1} \varphi_6(\Delta t) + R_v^n}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)}. \end{aligned} \right\} \quad (19)$$

5. Disease-Free Equilibrium

Given initial conditions,

$$S_h(t) = 0, I_h(t) = 0, R_h(t) = 0, S(t) = 0, I_v(t) = 0, R_v(t) = 0. \quad (20)$$

The disease-free equilibrium of the system of equations in its explicit form can be established by linearising the system in its explicit form. The Jacobian matrix of the system of equations is given by

$$\begin{pmatrix} \frac{1}{1 + \mu_h \varphi_1(\Delta t)} & P_1 & \frac{\sigma_h \varphi_1(\Delta t)}{1 + \mu_h \varphi_1(\Delta t)} & 0 & P_6 & 0 \\ 0 & P_2 & 0 & 0 & P_7 & 0 \\ 0 & P_3 & \frac{1}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)} & 0 & 0 & 0 \\ 0 & P_4 & 0 & \frac{1}{1 + \mu_v \varphi_4(\Delta t)} & P_8 & \frac{\sigma_v \varphi(\Delta t)}{1 + \mu_v \varphi_4(\Delta t)} \\ 0 & P_5 & 0 & 0 & P_9 & 0 \\ 0 & 0 & 0 & 0 & P_{10} & \frac{1}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)} \end{pmatrix}, \quad (21)$$

where

$$\left. \begin{aligned} P_1 &= \frac{-(\beta \Lambda_h / \mu_h) \varphi_1(\Delta t)}{1 + \mu_h \varphi_1(\Delta t)}, P_6 = \frac{-(\beta \Lambda_h / \mu_h) \varphi_1(\Delta t)}{1 + \mu_h \varphi_1(\Delta t)}, \\ P_2 &= \frac{1 + (\beta \Lambda_h / \mu_h) \varphi_2(\Delta t)}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)}, P_7 = \frac{(\beta \Lambda_h / \mu_h) \varphi_2(\Delta t)}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)}, \\ P_3 &= \frac{\gamma \varphi_3(\Delta t)}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)}, P_8 = \frac{-(\lambda \Lambda_v / \mu_v) \varphi_4(\Delta t)}{1 + \mu_v \varphi_4(\Delta t)}, \\ P_4 &= \frac{-(\lambda \Lambda_v / \mu_v) \varphi_4(\Delta t)}{1 + \mu_v \varphi_4(\Delta t)}, P_9 = \frac{(\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)}, \\ P_5 &= \frac{(\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)}, P_{10} = \frac{\alpha \varphi_6(\Delta t)}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)} \end{aligned} \right\} \quad (22)$$

The corresponding eigenvalues of the Jacobian matrix are obtained as

$$\left. \begin{aligned} \lambda_1 &= \frac{1}{1 + \mu_h \varphi_1(\Delta t)}, \lambda_2 = \frac{1 + (\beta \Lambda_h / \mu_h) \varphi_2(\Delta t)}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)}, \\ \lambda_3 &= \frac{1}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)}, \lambda_4 = \frac{1}{1 + \mu_v \varphi_4(\Delta t)}, \\ \lambda_5 &= \frac{1 + (\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)}, \lambda_6 = \frac{1}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)}. \end{aligned} \right\} \quad (23)$$

5.1. Local Stability of the Disease-Free Equilibrium

Theorem 3. *The DFE is locally asymptotically stable for every value of (Δt) if the following conditions are satisfied:*

- (i) $\beta \Lambda_h / \mu_h (\gamma + \mu_h + \delta_h) < 1$
- (ii) $\lambda \Lambda_v / \mu_v (\alpha + \mu_v + \delta_v) < 1$

Proof. The sequence

$$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n), \quad (24)$$

should converge to the disease-free equilibrium

$$\text{DFE} = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right), \quad (25)$$

for any positive initial conditions when conditions (i) and (ii) are satisfied for every value of (Δt) .

Linearising system (3) at the DFE, the eigenvalues of the corresponding Jacobian matrix are given by

$$\left. \begin{aligned} \lambda_1 &= \frac{1}{1 + \mu_h \varphi_1(\Delta t)}, \lambda_2 = \frac{1 + (\beta \Lambda_h / \mu_h) \varphi_2(\Delta t)}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)}, \\ \lambda_3 &= \frac{1}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)}, \lambda_4 = \frac{1}{1 + \mu_v \varphi_4(\Delta t)}, \\ \lambda_5 &= \frac{1 + (\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)}, \lambda_6 = \frac{1}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)}. \end{aligned} \right\} \quad (26)$$

It shows that the DFE is locally asymptotically stable for every value of (Δt) if the conditions (i) and (ii) of Definition 1 are satisfied.

For λ_1 ,

$$|\lambda_1| = \left| \frac{1}{1 + \mu_h \varphi_1(\Delta t)} \right|, \quad (27)$$

$$|\lambda_1| < 1 \text{ since } 1 + \mu_h \varphi_1(\Delta t) > 1.$$

For λ_2 ,

$$|\lambda_2| = \left| \frac{1 + (\beta \Lambda_h / \mu_h) \varphi_2(\Delta t)}{1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t)} \right|, \quad (28)$$

$$|\lambda_2| < 1 \text{ if and only if } 1 + (\beta \Lambda_h / \mu_h) \varphi_2(\Delta t) < 1 + (\gamma + \mu_h + \delta_h) \varphi_2(\Delta t).$$

For λ_3 ,

$$|\lambda_3| = \left| \frac{1}{1 + (\sigma_h + \mu_h) \varphi_3(\Delta t)} \right|, \quad (29)$$

$$|\lambda_3| < 1 \text{ since } 1 + (\sigma_h + \mu_h) \varphi_3(\Delta t) > 1$$

For λ_4 ,

$$|\lambda_4| = \left| \frac{1}{1 + \mu_v \varphi_4(\Delta t)} \right|, \quad (30)$$

$$|\lambda_4| < 1 \text{ since } 1 + \mu_v \varphi_4(\Delta t) > 1.$$

For λ_5 ,

$$|\lambda_5| = \left| \frac{1 + (\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t)} \right|, \quad (31)$$

$$|\lambda_5| \leq 1 \text{ on condition that } 1 + (\lambda \Lambda_v / \mu_v) \varphi_5(\Delta t) < 1 + (\alpha + \mu_v + \delta_v) \varphi_5(\Delta t).$$

For λ_6 ,

$$|\lambda_6| = \left| \frac{1}{1 + (\sigma_v + \mu_v) \varphi_6(\Delta t)} \right|, \quad (32)$$

$$|\lambda_6| < 1 \text{ since } 1 + (\sigma_v + \mu_v) \varphi_6(\Delta t) > 1.$$

5.2. Global Stability of the Disease-Free Equilibrium

Theorem 4. *The disease-free equilibrium is globally asymptotically stable if the conditions stated in Theorem 3 are satisfied.*

Proof. The sequence;

$$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n), \quad (33)$$

should converge to the disease-free equilibrium

$$\left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right), \quad (34)$$

for any positive initial condition whenever conditions (i) and (ii) of Definition 1 are satisfied for every value of Δt .

From Definition 1, the DFE is LAS for every value of Δt whenever conditions (i) and (ii) of Definition 1 hold.

Suppose that for $n > 0$,

$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n)$ converges to $(\Lambda_h/\mu_h, 0, 0, \Lambda_v/\mu_v, 0, 0)$.

Then, it can be shown that

$$(S_h^{n+1}, I_h^{n+1}, R_h^{n+1}, S_v^{n+1}, I_v^{n+1}, R_v^{n+1}), \quad (35)$$

converges to

$$\left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right). \quad (36)$$

Considering the system of equation in (19), we prove that the disease-free equilibrium is globally asymptotically stable using the conditions in Definition 1.

For I_h^{n+1} ,

$$I_h^{n+1} = \frac{(\beta I_h^n S_h^{n+1} + \beta I_v^n S_h^{n+1})\varphi_2(\Delta t) + I_h^n}{1 + (\gamma + \mu_h + \delta_h)\varphi_2(\Delta t)}, \quad (37)$$

then, $I_h^{n+1} \rightarrow 0$, as $n \rightarrow \infty$.

For R_h^{n+1} ,

$$R_h^{n+1} = \frac{\gamma I_h^{n+1} \varphi_3(\Delta t) + R_h^n}{1 + (\sigma_h + \mu_h)\varphi_3(\Delta t)}, \quad (38)$$

then, $R_h^{n+1} \rightarrow 0$ as $n \rightarrow \infty$.

For S_h^{n+1} ,

$$S_h^{n+1} = \frac{(\Lambda_h + \sigma_h R_h^{n+1})\varphi_1(\Delta t) + S_h^n}{1 + (\beta I_h^n + \beta I_v^n + \mu_h)\varphi_1(\Delta t)}, \quad (39)$$

then, $S_h^{n+1} \rightarrow \Lambda_h/\mu_h$ as $n \rightarrow \infty$.

For I_v^{n+1} ,

$$I_v^{n+1} = \frac{(\lambda I_h^n S_v^{n+1} + \lambda I_v^n S_v^{n+1})\varphi_5(\Delta t) + I_v^n}{1 + (\alpha + \mu_v + \delta_v)\varphi_5(\Delta t)}, \quad (40)$$

then, $I_v^{n+1} \rightarrow 0$ as $n \rightarrow \infty$.

For R_v^{n+1} ,

$$R_v^{n+1} = \frac{\alpha I_v^{n+1} \varphi_6(\Delta t) + R_v^n}{1 + (\sigma_v + \mu_v)\varphi_6(\Delta t)}, \quad (41)$$

then, $R_v^{n+1} \rightarrow 0$ as $n \rightarrow \infty$.

For S_v^{n+1} ,

$$S_v^{n+1} = \frac{(\Lambda_v + \sigma_v R_v^{n+1})\varphi_4(\Delta t) + S_v^n}{1 + (\lambda I_h^n + \lambda I_v^n + \mu_v)\varphi_4(\Delta t)}, \quad (42)$$

then, $S_v^{n+1} \rightarrow \Lambda_v/\mu_v$ as $n \rightarrow \infty$.

Hence, the DFE is GAS since conditions (i) and (ii) are satisfied for every value of (Δt) .

6. Optimal Control Analysis of the Model

In this section, we carried out an analysis of optimal control to determine the impact of all intervention of the control schemes. This is derived by incorporating the following controls into the model in Figure 1 and the introduction of an objective functional that seeks to minimise: (u_1, u_2, u_3) , where u_1 denotes prevention of S_h , u_2 denotes treatment of I_h , and u_3 denotes treatment of I_v .

By introducing all controls, the system in equation (3) becomes

$$\left. \begin{aligned} \frac{dS_h}{dt} &= \Lambda_h + \sigma_h - (1 - u_1)\beta(I_v + I_h)S_h - \mu_h S_h, \\ \frac{dI_h}{dt} &= (1 - u_1)\beta(I_v + I_h)S_h - (u_2 + \gamma)I_h - (\delta_h + \mu_h)I_h, \\ \frac{dR_h}{dt} &= (u_2 + \gamma)I_h - (\sigma_h + \mu_h)R_h, \\ \frac{dS_v}{dt} &= \Lambda_v - (1 - u_1)\lambda(I_v + I_h)S_v - (\mu_v S_v + \sigma_v R_v), \\ \frac{dI_v}{dt} &= (1 - u_1)\lambda(I_v + I_h)S_v - \alpha I_v - (\delta_v + \mu_v)I_v, \\ \frac{dR_v}{dt} &= \alpha I_v - (\sigma_v + \mu_v)R_v. \end{aligned} \right\} \quad (43)$$

In epidemiological models, the essence of optimal control analysis is to minimise the spread or number of infections and cost associated with treatment and prevention controls. The objective functional required to achieve this is formulated by

$$J = \min_{(u_1, u_2, u_3)} \int_0^{t_f} (B_1 I_v + B_2 I_h + B_3 u_1^2 + B_4 u_2^2 + B_5 u_3^2) dt, \quad (44)$$

subject to the system of equations in (3).

Control efforts of model in (3) is by linear combination of $u_i^2(t)$, ($i = 1, 2$). It is assumed to be a quadratic in nature by the assumption that cost is generally nonlinear in nature. Thus, the aim is to minimise the number of infection and reduce the cost of treatment.

In (44), B_1, B_2, B_3, B_4 , and B_5 denote weight constants to facilitate balance terms in the integral to avoid dominance of one another. $B_1 I_h, B_2 I_v$ are costs associated with I_h and I_v , respectively. $B_3 u_1^2$, is cost associated with prevention of S_h . $B_4 u_2^2$, is cost of treatment of I_h , and $B_5 u_3^2$ is cost associated with treatment of I_v . t_f , is the period of intervention. Hence, $(B_1 I_h, B_2 I_v)$ denotes a linear function for cost associated with infections and $(B_3 u_1^2, B_4 u_2^2,$

$B_5 u_3^2$), denotes a quadratic function for the cost associated with controls [19, 20].

The objective is to find the optimal functions $(u_1^*(t), u_2^*(t), u_3^*(t))$ such that

$$J(u_1^*(t), u_2^*(t), u_3^*(t)) = \min_{(u_1, u_2, u_3) \in \cup} J(u_1, u_2, u_3), \quad (45)$$

where

$\{u : u, 0 \leq u_i(t) \leq 1, t \in [0, t_f], i = 1, 2, 3\}$ denotes the control set.

6.1. Pontryagin's Maximum Principle. This principle provides the necessary conditions that an optimal must satisfy. It changes the system in equations (3) and (44) into minimisation problem

pointwise Hamiltonian (H), with respect to (u_1, u_2, u_3) .

$$\begin{aligned} H = & B_1 I_v + B_2 I_h + B_3 u_1^2 + B_4 u_2^2 + B_5 u_3^2 + M_{I_h} \\ & \cdot \{(1 - u_1)\beta(I_v + I_h)S_h - (u_2 + \gamma)I_h - (\delta_h + \mu_h)I_h\} \\ & + M_{R_h} \{(u_2 + \gamma)I_h - (\sigma_h + \mu_h)R_h\} + M_{S_v} \\ & \cdot \{\Lambda_v - (1 - u_1)\lambda(I_v + I_h)S_v - (\mu_v S_v + \sigma_v R_v)\} \\ & + M_{I_v} \{(1 - u_1)\lambda(I_v + I_h)S_v - (u_3 + \alpha)I_v \\ & - (\delta_v + \mu_v)I_v\} M_{R_v} \{(u_3 + \alpha)I_v - (\sigma_v + \mu_v)R_v\}, \end{aligned} \quad (46)$$

where

$M_{S_h}, M_{I_h}, M_{R_h}, M_{S_v}, M_{I_v}$, and M_{R_v} are referred to as the adjoint variables.

The adjoint (costate) variables are solutions of adjoint systems below:

$$\left. \begin{aligned} \frac{dM_{S_h}}{dt} &= ((1 - u_1)(I_v + I_h)\beta(M_{S_h} - M_{I_h}) + \mu_h M_{S_h}), \\ \frac{dM_{I_h}}{dt} &= -B_2 + (1 - u_1)\beta S_h(M_{S_h} - M_{I_h}) + (u_2 + \gamma)(M_{I_h} - M_{R_h}) \\ &\quad + (\mu_h + \delta_h)M_{I_h} + (1 - u_1)\lambda S_v(M_{S_v} - M_{I_v}) + b\lambda V_v(M_{V_v} - M_{I_v}), \\ \frac{dM_{R_h}}{dt} &= -\sigma_h M_{S_h} + (\sigma_h + \mu_h)M_{R_h}, \\ \frac{dM_{S_v}}{dt} &= (1 - u_1)\lambda(I_v + I_h)(M_{S_v} - M_{I_v}) + \mu_v M_{S_v}, \\ \frac{dM_{I_v}}{dt} &= -B_1 + (1 - u_1)\beta S_h(M_{S_h} - M_{I_h}) + (1 - u_1)\lambda S_v(M_{S_v} - M_{I_v}) \\ &\quad + (\mu_v + \delta_v)M_{I_v} + (u_4 + \alpha)(M_{I_v} - M_{R_v}), \\ \frac{dM_{R_v}}{dt} &= -\sigma_h M_{S_v} + (\sigma_v + \mu_v)M_{R_v}. \end{aligned} \right\} \quad (47)$$

This satisfies the transversality condition

$$M_{S_h}(t_f) = M_{I_h}(t_f) = M_{R_h}(t_f) = M_{S_v}(t_f) = M_{I_v}(t_f) = M_{R_v}(t_f) = 0, \quad (48)$$

by combining the Pontryagin's maximum principle and the existence of the optimal control.

Theorem 5. The optimal control vector $(u_1^*(t), u_2^*(t), u_3^*(t))$ that maximises the objective function (J) over \cup is given by

$$\left. \begin{aligned} u_1^*(t) &= \max \left\{ 0, \min \left(1, \frac{\beta(M_{I_h} - M_{S_h})(I_v + I_h)S_h^*}{2B_3} + \frac{\lambda(M_{I_v} - M_{S_v})(I_v + I_h)S_v^*}{2B_3} \right) \right\}, \\ u_2^*(t) &= \max \left\{ 0, \min \left(1, \frac{(M_{I_h} - M_{R_h})I_h^*}{2B_4} \right) \right\}, \\ u_3^*(t) &= \max \left\{ 0, \min \left(1, \frac{(M_{I_v} - M_{R_v})I_v^*}{2B_5} \right) \right\}, \end{aligned} \right\} \quad (49)$$

where

M_{S_h} , M_{I_h} , M_{R_h} , M_{S_v} , M_{I_v} , and M_{R_v} are the solutions of equation (47) and (48).

Proof. The existence of an optimal control is as a result the convexity of the integral of J with respect to u_1 , u_2 , and u_3 , the Lipschitz property of the state system with respect to the state variables, and a priori boundedness of the state solutions [21, 22]. The system in (47) was obtained by differentiating the Hamiltonian function and evaluated at optimal control. However, by equating the derivatives of the Hamiltonian with respect to the controls to zero, the following are obtained:

$$\left. \begin{aligned} u_1 = \tilde{u}_1 &:= \left\{ \left(\frac{\beta(M_{I_h} - M_{S_h})(I_v + I_h)S_h^*}{2B_3} + \frac{\lambda(M_{I_v} - M_{S_v})(I_v + I_h)S_v^*}{2B_3} \right) \right\} \\ u_2 = \tilde{u}_2 &:= \left\{ \left(\frac{(M_{I_h} - M_{R_h})I_h^*}{2B_4} \right) \right\}, \\ u_3 = \tilde{u}_3 &:= \left\{ \left(\frac{(M_{I_v} - M_{R_v})I_v^*}{2B_5} \right) \right\}. \end{aligned} \right\} \quad (50)$$

In conclusion, by standard control arguments involving bounds on controls,

$$\left. \begin{aligned} u_1^* &= \begin{cases} 0, & \text{if } \tilde{u}_1 \leq 0, \\ \tilde{u}_1 & \text{if } 0 < \tilde{u}_1 < 1, \\ 1 & \text{if } \tilde{u}_1 \geq 1, \end{cases} & u_2^* &= \begin{cases} 0, & \text{if } \tilde{u}_2 \leq 0, \\ \tilde{u}_2 & \text{if } 0 < \tilde{u}_2 < 1, \\ 1 & \text{if } \tilde{u}_2 \geq 1, \end{cases} \\ u_3^* &= \begin{cases} 0, & \text{if } \tilde{u}_3 \leq 0, \\ \tilde{u}_3 & \text{if } 0 < \tilde{u}_3 < 1, \\ 1 & \text{if } \tilde{u}_3 \geq 1. \end{cases} \end{aligned} \right\} \quad (51)$$

The system in (49) leads to system in (48). The optimal control uniqueness for small t_f was obtained as a result of the Lipschitz structure of system of equations and the priori boundedness of the state solutions and adjoint functions. Existence of optimal control uniqueness is in line with uniqueness of optimal system that comprises equations (3), (47), (48), and (49) [22–24].

7. Numerical Analysis

In this section, we solved the optimal system by employing the Range-Kutta fourth-order scheme. We solved the state systems, adjoint equations, and the transversality conditions by considering it as a two-point boundary value problem with boundary conditions at $t = 0$ and $t = t_f$. Our goal is to solve for $t_f = 90$ days or three months. At this value, it is assumed that campylobacteriosis can easily spread. System of equations of the model in Figure 1 is solved numerically using the Range-Kutta fourth-order scheme with a guess on controls over a period of time. Moreover, we used current iter-

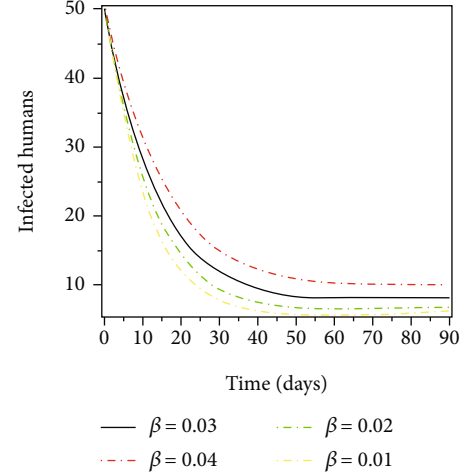


FIGURE 2: Effects of contact rate on infected humans.

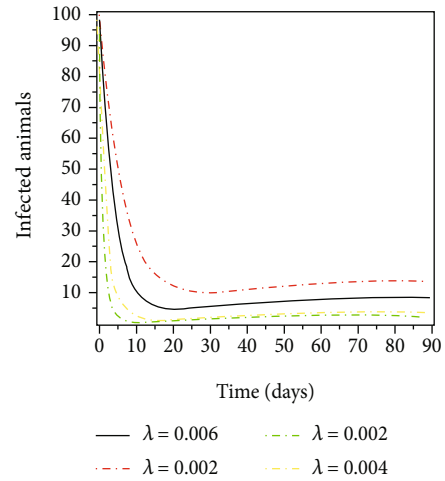


FIGURE 3: Effects of contact rate on infected animals.

ations of the model equations in Figure 1, the costate equations, and transversality conditions by backward approach. Convex combinations of controls in previous iterations and characterisations of values from the system are then updated. The process is repeated continuously, and iteration stops if values of unknowns at previous iteration is as close as those at present iteration [25, 26]. A number of combination of controls were considered and the best and most effective selected.

7.1. Analysis of Contact Rate (β) on Infected Humans. Figure 2 shows the analysis of contact rate (β) on infected humans. As the contact rate (β) increases, there seem to be an increase in the number of infections. As the contact rate (β) decreases, there is a corresponding decrease in the number of infected humans. This confirms the effects of contact rate (β) on infected humans. Hence, infections can be curbed by ensuring that the value of contact rate (β) reduces to the bearest minimum.

7.2. Analysis of Contact Rate (λ) on Infected Animals. Figure 3 shows the analysis of contact rate (λ) on infected

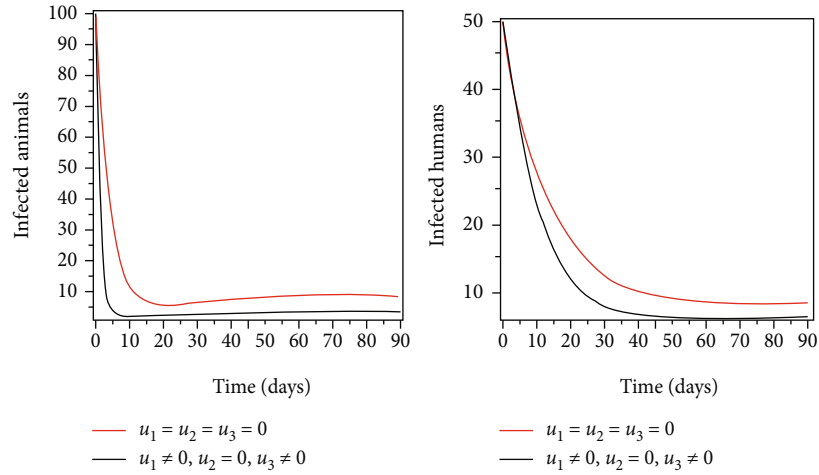


FIGURE 4: Population dynamics of infected animals and humans with and without optimal control.

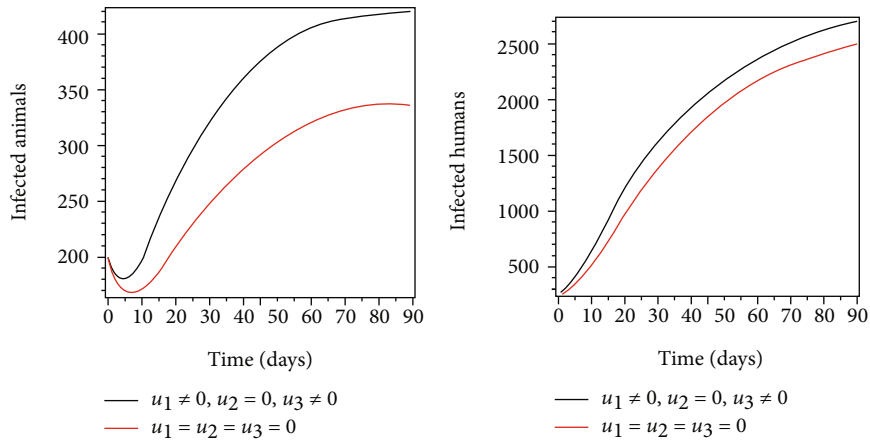


FIGURE 5: Population dynamics of recovered animals and humans with and without optimal control.

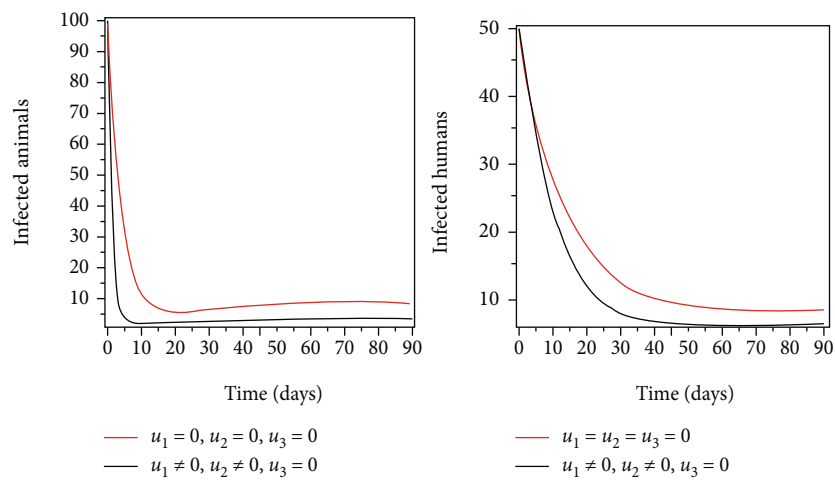


FIGURE 6: Population dynamics of infected animals and humans with and without optimal control.

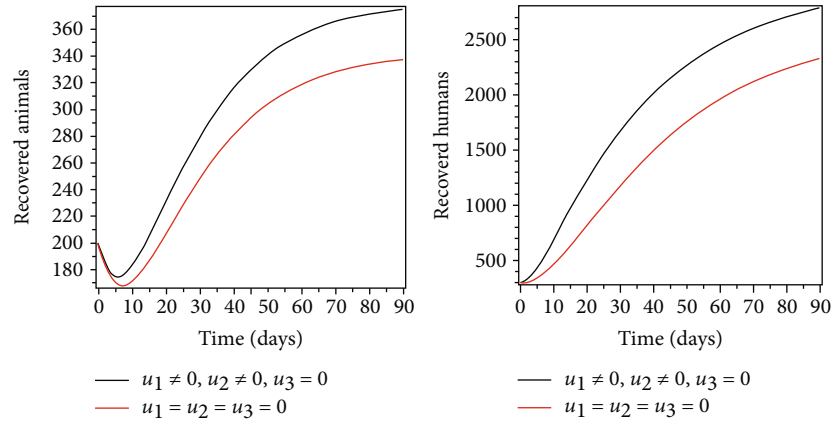


FIGURE 7: Population dynamics of recovered animals and humans with and without optimal control.

TABLE 1: Shows the parameter values used in the model simulation.

Parameter	Value	References
β	0.03	Assumed
λ	0.004	[4]
Λ_v	0.005	Assumed
Λ_h	0.002	Assumed
μ_v	0.0002	[4]
δ_v	0.003	Assumed
α	0.05	[4]
μ_h	0.0001	Assumed
δ_h	0.001	Assumed
σ_h	0.004	Assumed
σ_v	0.007	[4]....

animals. As the contact rate increases, there seem to be an increase in the number of infections. As the contact rate decreases, there is a corresponding decrease in the number of infected animals. This confirms the effects of contact rate (λ) on infected animals. Hence, infections can be curbed by ensuring that the value of contact rate (λ) reduces to the bearest minimum.

7.3. Strategy 1: Optimal Prevention of S_h and Treatment of I_v . We optimised the objective function using prevention of S_h and treatment of I_v as control measures. This was done by setting the treatment of infected humans, u_2 , to zero. Figure 4 indicates a reduction in number of campylobacteriosis-infected animals and humans. Figure 5 indicates an increase in campylobacteriosis recovery in both animal and human populations.

Biologically, the implication is that campylobacteriosis infections can be controlled effectively by prevention of humans and treatment of infected animals.

7.4. Strategy 2: Optimal Prevention of S_h and Treatment of I_h . We optimised the objective function using prevention of S_h and treatment of I_h as control measures. This was done by

setting the treatment of infected humans, u_3 , to zero. Figure 6 indicates a reduction in number of campylobacteriosis-infected animals and humans. Figure 7 indicates an increase in campylobacteriosis recovery in both animal and human populations.

Biologically, the implication is that campylobacteriosis infections can be curbed effectively by prevention of humans and treatment of infected animals.

8. Conclusion

A deterministic model that explains the spread dynamics of campylobacteriosis infection was formulated and analysed for its qualitative and quantitative solutions. The qualitative analysis of the model was carried out using the nonstandard finite difference scheme for boundedness of solution, disease-free equilibrium, and its local and global stability. Campylobacteriosis disease-free equilibrium of the scheme in its explicit form was established. Analysis of the scheme established that the disease-free equilibrium was both locally and globally asymptotically stable.

The campylobacteriosis model was extended to optimal control using prevention of susceptible humans, treatment of infected humans, and treatment of infected animals. The objective functional was optimised, and it was established that combining prevention of susceptible humans and treatment of infected animals was the effective control measure in combating campylobacteriosis infections.

An analysis of the effects of contact rate between susceptible and infected animals as well as susceptible and infected humans was conducted. This showed an increase in infected animals and humans whenever the contact rate increases and decreases otherwise. Biologically, campylobacteriosis infections can be controlled by ensuring that interactions between susceptible humans, infected animals, and infected humans is reduced to the bearest minimum.

Data Availability

The parameter values supporting this deterministic model simulations were assumed and others taken from some published articles. All these were dully acknowledged and cited in

this paper. These published articles are also cited at relevant places within the text as references.

Conflicts of Interest

We declare that there are no conflict of interest regarding the publication of this article.

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