Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2022, 2022:33
https://doi.org/10.28919/cmbn/7183
ISSN: 2052-2541

NONSTANDARD FINITE DIFFERENCE METHOD OF MODELLING ZOONOTIC DISEASES

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Abstract. Zoonotic diseases are mostly the leading causes of illness and deaths in Sub-Saharan Africa but efforts to combat the spread of these diseases has always been a challenge. Incidence of zoonotic diseases has reduced substantially in most parts of Africa as a result of rigorous vaccination campaigns. However, zoonotic diseases still remain a threat to developing nations. Zoonotic diseases can be contracted either by direct contact, food and water. In this paper, we developed and analysed a general model that explains the dynamics of zoonotic diseases and analysed it using nonstantard finite difference approach. This scheme was used for the model analysis. The disease free equilibrium of the scheme in its explicit form was determined and it was both locally and globally asymptotically stable. Bifurcation and multiple equilibria as well as the threshold value for disease transmission was determined. An analysis of the effects of contact between susceptible and infected animals as well susceptible and infected humans was conducted. It showed an increase in infected animals and humans whenever the contact rate increases and decreases otherwise. The epidemiological implication is that zoonotic disease can be controlled

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Received January 16, 2022

by ensuring that interactions between susceptible humans, infected animals and infected humans is reduced to the bearest minimum.

Keywords: zoonotic disease; reproductive rate; nonstandard finite difference; stability; bifurcation.

2010 AMS Subject Classification: 92D30, 37M05.

1. INTRODUCTION

Zoonosis are among the leading causes of illness and deaths in Sub-Saharan Africa but efforts to combat the spread of these diseases has always been a challenge. Incidence of these diseases has reduced significantly in many developed nations as a result of proper vaccination campaigns. Unfortunately, zoonosis still a threat to many developing nations especially Africa. Zoonotic diseases can be contracted either by direct contact, food and water [1, 2]. Campylobacter genus is the bacteria responsible for zoonotic Campylobacteriosis infections.

Campylobacteriosis can be spread or contracted through the fecal-oral path. It is a zoonotic disease 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].

Campylobacteriosis infected person is usually asymptomatic at the incubation period, that is between one to 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 [3]. Symptomatic persons can infect others directly, contaminate water and food during the infectious period of Campylobacteriosis. The disease is mostly food and waterborne illness but can also be spread through direct contact with infected humans or animals via fecal-oral path of transmission. But human to human spread is usually uncommon [4].

Deterministic models enhance the general understanding of disease spread by the provision of a theoretical frame which underlines factors that accounts for spread and control of diseases [5, 6, 7, 8, 9, 10]. 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 [11].

Authors in [12, 13, 14] employed the concept of Non standard finte difference method in modelling waterborne disease and pharmacokinetic model respectively. This same approach has been employed in this paper to model zoonotic diseases and to explain the dynamics of disease spread among opulation.

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 mechanism and causes of patterns present in an observed data is usually an objective that initiates a deterministic modelling process [15]. Moreover, epidemiological models explain dynamics of infections, determine best optimal control strategies and the most cost effective amongst these strategies [16, 17, 18, 19, 20]. However, authors in [21, 22, 23, 24, 25, 26] proposed and formulated models that attempts to explain this hidden and existing phenomena.

2. Model Description and Formulation

We divide the zoonotic model into two parts, human and animal populations. Populations at any time, t are divided into six sub-groups with respect to their disease status in the system. The total human population also represented by $N_h(t)$, is divided into sub-populations of Susceptible humans $S_h(t)$, Infected humans $I_v(t)$, and Recovered humans $R_v(t)$. Susceptible humans are recruited 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 δ_h and may looses immunity at a rate σ_h. Campylobacteriosis natural death rate for all human compartments is μ_h. Susceptible animalsS_v, are recruited 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 animals natural death rate is μ_v. Infected animals death rate as a result of campylobacteriosis is δ_v and animals may recover at a rate α. Animals may looses immunity at a rate σ_v.

Hence, total human population:

$$N_h(t) = S_h(t) + I_h(t) + R_h(t). \tag{1}$$

Total animal population, $N_v(t)$, is divided into sub-populations of Susceptible animals $S_v(t)$, Infectious animals $I_v(t)$, and Recovered animals $R_v(t)$.

Hence, total animal population:

$$N_v(t) = S_v(t) + I_v(t) + R_v(t). \tag{2}$$

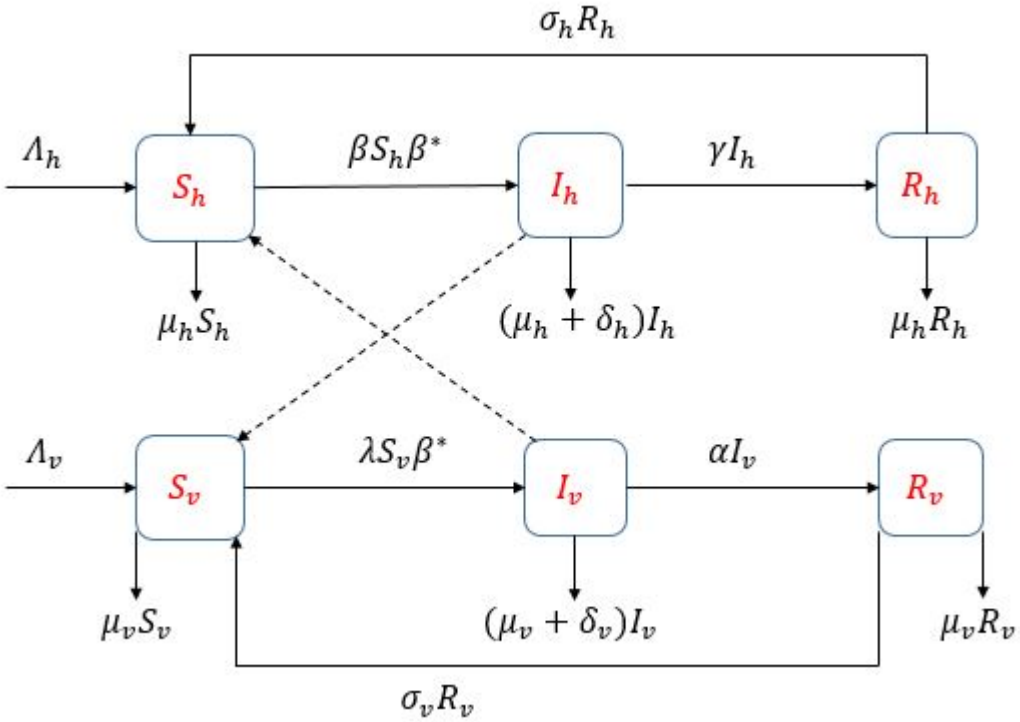


FIGURE 1. Model flow diagram.

System of equations obtained from the model in Figure 1:

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

Where, $\beta_m^* = I_h + I_v$.

3. Model Analysis

Positivity and boundedness of solutions: The solution of the system in (3) is a function of the form;

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

Considering;

$$F : X \in \mathbb{R}^6 \longrightarrow F(X) \in \mathbb{R}^6 \tag{5}$$

Where,

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

Hence;
$\frac{dX}{dt} = F(X)$
$X(0) = X_0 = \left(S_{h(0)}; I_{h(0)}; R_{h(0)}; S_{v(0)}; I_{v(0)}; R_{v(0)}\right)^T$

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 is usually non negative whenever its initial conditions are non negative.

4. NONSTANDARD FINITE DIFFERENCE SCHEME

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

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

$X(0) = X_0$

Where, F is C^1 usually of the form;

$$D_{\triangle t}(X_k) = F_{\triangle t}(X_k) \tag{8}$$

Where;

$$\left.\begin{array}{l} D_{\triangle t}(X_k) \approx \dfrac{dX(t_k)}{dt} \\ \\ X_k \approx X(t_k) \end{array}\right\} \quad , \quad \left.\begin{array}{l} F_{\triangle t}(X_k) \approx F(X_k) \\ \\ t_k \approx t_0 + k\triangle_t \end{array}\right\} \tag{9}$$

The scheme;

$$D_{\triangle t}(X_k) = F_{\triangle t}(X_k) \tag{10}$$

Theorem 4.1. *The scheme* (10) *can be referred to as nonstandard finite difference scheme when it at least satisfy the following conditions;*

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

$$\left.\begin{array}{l} \psi(\triangle t) = 1 + \mathscr{O}(\triangle t) \\ \\ \varphi(\triangle t) = \triangle t + \mathscr{O}(\triangle t^2) \end{array}\right\} \tag{11}$$

- $F_{\triangle t}(X_k) = g\,(X_k, X_{k+1}, \triangle t)$ where g denotes an approximation of the non local right hand side of the system.

Theorem 4.2. *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 [22, 23, 13], the NSFD scheme for the system of (3)

is given by:

$$
\left.
\begin{array}{rcl}
\frac{S_h^{n+1}-S_h^n}{\varphi_1(\triangle t)} = & \Lambda_h-\beta(I_h^n+I_v^n)S_h^{n+1}-\mu_h S_h^{n+1}+\sigma_h R_h^{n+1} \\
\frac{I_h^{n+1}-I_h^n}{\varphi_2(\triangle t)} = & \beta(I_h^n+I_v^n)S_h^{n+1}-\gamma I_h^{n+1}-(\mu_h+\delta_h)I_h^{n+1} \\
\frac{R_h^{n+1}-R_h^n}{\varphi_3(\triangle t)} = & \gamma I_h^{n+1}-(\sigma_h+\mu_h)R_h^{n+1} \\
\frac{S_v^{n+1}-S_v^n}{\varphi_4(\triangle t)} = & \Lambda_v-\lambda(I_h^n+I_v^n)S_v^{n+1}-\mu_v S_v^{n+1}+\sigma_v R_v^{n+1} \\
\frac{I_v^{n+1}-I_v^n}{\varphi_5(\triangle t)} = & \lambda(I_h^n+I_v^n)S_v^{n+1}-\alpha I_v^{n+1}-(\mu_v+\delta_v)I_v^{n+1} \\
\frac{R_v^{n+1}-R_v^n}{\varphi_6(\triangle t)} = & \alpha I_v^{n+1}-(\sigma_v+\mu_v)R_v^{n+1}
\end{array}
\right\} \tag{12}
$$

where,

$$
\varphi_j(\triangle t,k_j^*)=\frac{1-e^{-k_j^*\Delta t}}{k_j^*}, \tag{13}
$$

$k_j^*=\max\{|\gamma_i|\}$

$$
\left.
\begin{array}{rl}
j= & 1,2,3,\ldots 6 \\
 & \\
i= & 1,2,3,\ldots 6
\end{array}
\right\} \text{ with; } \gamma_i=\left.\frac{\partial f}{\partial x_i}\right|_{x=\bar{x}} \text{ and } f(\bar{x})=0.
$$

Where;

$$\left.\begin{aligned}
\varphi_1(\triangle t) &= \left(\tfrac{1-e^{-\mu_h\Delta t}}{\mu_h}, \tfrac{-\beta\Lambda_h}{\mu_h}, \sigma_h, 0, \tfrac{-\beta\Lambda_h}{\mu_h}, 0\right)\\
\varphi_2(\triangle t) &= \left(\tfrac{1-e^{-|\frac{\beta\Lambda_h}{\mu_h}-\gamma-\mu_h-\delta_h|\Delta t}}{|\frac{\beta\Lambda_h}{\mu_h}-\gamma-\mu_h-\delta_h|}, \tfrac{-\beta\Lambda_h}{\mu_h}, 0, 0, \tfrac{-\beta\Lambda_h}{\mu_h}, 0\right)\\
\varphi_3(\triangle t) &= \left(\tfrac{1-e^{-(\sigma_h+\mu_h)\Delta t}}{(\sigma_h+\mu_h)}, \gamma, -\sigma_h-\mu_h, 0, 0, 0\right)\\
\varphi_4(\triangle t) &= \left(\tfrac{1-e^{-\mu_v\Delta t}}{\mu_v}, 0, -\tfrac{\lambda\Lambda_v}{\mu_v}, -\mu_v, -\tfrac{\lambda\Lambda_v}{\mu_v}, \sigma_v\right)\\
\varphi_5(\triangle t) &= \left(\tfrac{1-e^{-|\frac{\lambda\Lambda_v}{\mu_v}-\alpha-\mu_v-\delta_v|\Delta t}}{|\frac{\lambda\Lambda_v}{\mu_v}-\alpha-\mu_v-\delta_v|}, 0, \tfrac{\lambda\Lambda_v}{\mu_v}, 0, 0, \tfrac{\lambda\Lambda_v}{\mu_v}-\alpha-\mu_v-\delta_v\right)\\
\varphi_6(\triangle t) &= \left(\tfrac{1-e^{-(\sigma_v+\mu_v)\Delta t}}{(\sigma_v+\mu_v)}, 0, 0, 0, \alpha, \sigma_v-\mu_v\right)
\end{aligned}\right\} \tag{14}$$

Where;

$$\left.\begin{aligned}
\varphi_1(\triangle t) &= \tfrac{1-e^{-\mu_h\Delta t}}{\mu_h}\\
\varphi_2(\triangle t) &= \tfrac{1-e^{-|\frac{\beta\Lambda_h}{\mu_h}-\gamma-\mu_h-\delta_h|\Delta t}}{|\frac{\beta\Lambda_h}{\mu_h}-\gamma-\mu_h-\delta_h|}\\
\varphi_3(\triangle t) &= \tfrac{1-e^{-(\sigma_h+\mu_h)\Delta t}}{(\sigma_h+\mu_h)}
\end{aligned}\right\}, \quad \left.\begin{aligned}
\varphi_4(\triangle t) &= \tfrac{1-e^{-\mu_v\Delta t}}{\mu_v}\\
\varphi_5(\triangle t) &= \tfrac{1-e^{-|\frac{\lambda\Lambda_v}{\mu_v}-\alpha-\mu_v-\delta_v|\Delta t}}{|\frac{\lambda\Lambda_v}{\mu_v}-\alpha-\mu_v-\delta_v|}\\
\varphi_6(\triangle t) &= \tfrac{1-e^{-(\sigma_v+\mu_v)\Delta t}}{(\sigma_v+\mu_v)}
\end{aligned}\right\} \tag{15}$$

The scheme in its explicit form is given by:

$$\left.\begin{aligned}
S_h^{n+1} &= \tfrac{(\Lambda_h+\sigma_h R_h^{n+1})\varphi_1(\triangle t)+S_h^n}{1+(\beta I_h^n+\beta I_v^n+\mu_h)\varphi_1(\triangle t)}\\
I_h^{n+1} &= \tfrac{(\beta I_h^n S_h^{n+1}+\beta I_v^n S_h^{n+1})\varphi_2(\triangle t)+I_h^n}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\triangle t)}\\
R_h^{n+1} &= \tfrac{\gamma I_h^{n+1}\varphi_3(\triangle t)+R_h^n}{1+(\sigma_h+\mu_h)\varphi_3(\triangle t)}
\end{aligned}\right\}, \quad \left.\begin{aligned}
S_v^{n+1} &= \tfrac{(\Lambda_v+\sigma_v R_v^{n+1})\varphi_4(\triangle t)+S_v^n}{1+(\lambda I_h^n+\lambda I_v^n+\mu_v)\varphi_4(\triangle t)}\\
I_v^{n+1} &= \tfrac{(\lambda I_h^n S_v^{n+1}+\lambda I_v^n S_v^{n+1})\varphi_5(\triangle t)+I_v^n}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\triangle t)}\\
R_v^{n+1} &= \tfrac{\alpha I_v^{n+1}\varphi_6(\triangle t)+R_v^n}{1+(\sigma_v+\mu_v)\varphi_6(\triangle t)}
\end{aligned}\right\} \tag{16}$$

5. DISEASE FREE EQUILIBRIUM

Given initial conditions;

$S_h(0) = 0, I_h(0) = 0, R_h(0) = 0, S(0) = 0, I_v(0) = 0, R_v(0) = 0.$

The disease free equilibrium of the scheme in its explicit form can established by linearising the system in its explicit form. The jacobian matrix of the scheme is given by;

$$
\begin{pmatrix}
\frac{1}{1+\mu_h\varphi_1(\Delta t)} & P_1 & \frac{\sigma_h\varphi_1(\Delta t)}{1+\mu_h\varphi_1(\Delta t)} & 0 & P_6 & 0 \\
0 & P_2 & 0 & 0 & P_7 & 0 \\
0 & P_3 & \frac{1}{1+(\sigma_h+\mu_h)\varphi_3(\Delta t)} & 0 & 0 & 0 \\
0 & P_4 & 0 & \frac{1}{1+\mu_v\varphi_4(\Delta t)} & P_8 & \frac{\sigma_v\varphi(\Delta t)}{1+\mu_v\varphi_4(\Delta t)} \\
0 & P_5 & 0 & 0 & P_9 & 0 \\
0 & 0 & 0 & 0 & P_{10} & \frac{1}{1+(\sigma_v+\mu_v)\varphi_6(\Delta t)}
\end{pmatrix} \tag{17}
$$

Where;

$$
\left.
\begin{aligned}
P_1 = \frac{-\frac{\beta\Lambda_h}{\mu_h}\varphi_1(\Delta t)}{1+\mu_h\varphi_1(\Delta t)} \quad &, \quad P_6 = \frac{-\frac{\beta\Lambda_h}{\mu_h}\varphi_1(\Delta t)}{1+\mu_h\varphi_1(\Delta t)} \\
P_2 = \frac{1+\frac{\beta\Lambda_h}{\mu_h}\varphi_2(\Delta t)}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\Delta t)} \quad &, \quad P_7 = \frac{\frac{\beta\Lambda_h}{\mu_h}\varphi_2(\Delta t)}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\Delta t)} \\
P_3 = \frac{\gamma\varphi_3(\Delta t)}{1+(\sigma_h+\mu_h)\varphi_3(\Delta t)} \quad &, \quad P_8 = \frac{-\frac{\lambda\Lambda_v}{\mu_v}\varphi_4(\Delta t)}{1+\mu_v\varphi_4(\Delta t)} \\
P_4 = \frac{-\frac{\lambda\Lambda_v}{\mu_v}\varphi_4(\Delta t)}{1+\mu_v\varphi_4(\Delta t)} \quad &, \quad P_9 = \frac{\frac{\lambda\Lambda_v}{\mu_v}\varphi_5(\Delta t)}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\Delta t)} \\
P_5 = \frac{\frac{\lambda\Lambda_v}{\mu_v}\varphi_5(\Delta t)}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\Delta t)} \quad &, \quad P_{10} = \frac{\alpha\varphi_6(\Delta t)}{1+(\sigma_v+\mu_v)\varphi_6(\Delta t)}
\end{aligned}
\right\} \tag{18}
$$

The corresponding eigenvalues of the Jacobian matrix is obtained as;

$$\left.\begin{aligned} \lambda_1 = \frac{1}{1+\mu_h\varphi_1(\Delta t)}, \lambda_2 = \frac{1+\frac{\beta\Lambda_h}{\mu_h}\varphi_2(\Delta t)}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\Delta t)} \\ \lambda_3 = \frac{1}{1+(\sigma_h+\mu_h)\varphi_3(\Delta t)}, \lambda_4 = \frac{1}{1+\mu_v\varphi_4(\Delta t)} \\ \lambda_5 = \frac{1+\frac{\lambda\Lambda_v}{\mu_v}\varphi_5(\Delta t)}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\Delta t)}, \lambda_6 = \frac{1}{1+(\sigma_v+\mu_v)\varphi_6(\Delta t)} \end{aligned}\right\} \tag{19}$$

Local stability of the disease free equilibrium:

Theorem 5.1. *The DFE is locally asymptotically stable for every value of* $(\triangle t)$ *if the following conditions are satisfied;*

(i) $\frac{\beta\Lambda_h}{\mu_h(\gamma+\mu_h+\delta_h)} < 1$
(ii) $\frac{\lambda\Lambda_v}{\mu_v(\alpha+\mu_v+\delta_v)} < 1$

Theorem 5.2. *The DFE is locally asymptotically stable for every value of* $(\triangle t)$ *if the conditions stated in Theorem 3 are satisfied.*

Proof. The sequence;

$$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n)_n \tag{20}$$

should converge to the disease free equilibrium

$$DFE = (\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0) \tag{21}$$

for any positive initial conditions when conditions (i) and (ii) are satisfied for every value of $(\triangle t)$.

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

$$\left.\begin{array}{c} \lambda_1 = \frac{1}{1+\mu_h\varphi_1(\Delta t)}, \lambda_2 = \frac{1+\frac{\beta\Lambda_h}{\mu_h}\varphi_2(\Delta t)}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\Delta t)} \\ \lambda_3 = \frac{1}{1+(\sigma_h+\mu_h)\varphi_3(\Delta t)}, \lambda_4 = \frac{1}{1+\mu_v\varphi_4(\Delta t)} \\ \lambda_5 = \frac{1+\frac{\lambda\Lambda_v}{\mu_v}\varphi_5(\Delta t)}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\Delta t)}, \lambda_6 = \frac{1}{1+(\sigma_v+\mu_v)\varphi_6(\Delta t)} \end{array}\right\} \tag{22}$$

It shows that the DFE is locally asymptotically stable for every value of $(\triangle t)$ if the conditions (i) and (ii) are satisfied.

For λ_1

$$|\lambda_1| = \left|\frac{1}{1+\mu_h\varphi_1(\Delta t)}\right| \tag{23}$$

$|\lambda_1| < 1$ since $1+\mu_h\varphi_1(\Delta t) > 1$

For λ_2

$$\lambda_2 = \left|\frac{1+\frac{\beta\Lambda_h}{\mu_h}\varphi_2(\Delta t)}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\Delta t)}\right| \tag{24}$$

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

For λ_3

$$|\lambda_3| = \left|\frac{1}{1+(\sigma_h+\mu_h)\varphi_3(\Delta t)}\right| \tag{25}$$

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

For λ_4

$$|\lambda_4| = \left|\frac{1}{1+\mu_v\varphi_4(\Delta t)}\right| \tag{26}$$

$|\lambda_4| < 1$ since $1+\mu_v\varphi_4(\Delta t) > 1$

For λ_5

$$|\lambda_5| = \left| \frac{1 + \frac{\lambda \Lambda_v}{\mu_v} \varphi_5(\Delta t)}{1 + (\alpha + \mu_v + \delta_v)\varphi_5(\Delta t)} \right| \tag{27}$$

$|\lambda_5| \leq 1$ if and only if $1 + \frac{\lambda \Lambda_v}{\mu_v} \varphi_5(\Delta t) < 1 + (\alpha + \mu_v + \delta_v)\varphi_5(\Delta t)$

For λ_6

$$|\lambda_6| = \left| \frac{1}{1 + (\sigma_v + \mu_v)\varphi_6(\Delta t)} \right| \tag{28}$$

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

Global stability of the disease free equilibrium:

Theorem 5.3. *The disease free equilibrium is Globally asymptotically stable if the conditions stated in theorem 3 are satisfied.*

Proof. The sequence;

$$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n)_n \tag{29}$$

should converge to the disease free equilibrium;

$$\left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right) \tag{30}$$

for any positive initial condition whenever conditions (i) and (ii) are satisfied for every value of $\triangle t$. □

From the proof of theorem 1, the DFE is LAS for every value of $\triangle t$ whenever conditions (i) and (ii) hold.

Suppose that for $n > 0$;

$(S_h^n, I_h^n, R_h^n, S_v^n, I_v^n, R_v^n)$ converge to $\left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0 \right)$.

Then it can be shown that;

$$(S_h^{n+1}, I_h^{n+1}, R_h^{n+1}, S_v^{n+1}, I_v^{n+1}, R_v^{n+1}) \tag{31}$$

converges to

$$\left(\frac{\Lambda_h}{\mu_h},0,0,\frac{\Lambda_v}{\mu_v},0,0\right). \tag{32}$$

For I_h^{n+1}:

$$I_h^{n+1}=\frac{(\beta I_h^n S_h^{n+1}+\beta I_v^n S_h^{n+1})\varphi_2(\triangle t)+I_h^n}{1+(\gamma+\mu_h+\delta_h)\varphi_2(\triangle t)} \tag{33}$$

then,$\lim_{\infty} I_h^{n+1}=0$ as $n\to\infty$

For R_h^{n+1}:

$$R_h^{n+1}=\frac{\gamma I_h^{n+1}\varphi_3(\triangle t)+R_h^n}{1+(\sigma_h+\mu_h)\varphi_3(\triangle t)} \tag{34}$$

then,$\lim_{\infty} R_h^{n+1}=0$ as $n\to\infty$

For S_h^{n+1}:

$$S_h^{n+1}=\frac{(\Lambda_h+\sigma_h R_h^{n+1})\varphi_1(\triangle t)+S_h^n}{1+(\beta I_h^n+\beta I_v^n+\mu_h)\varphi_1(\triangle t)} \tag{35}$$

then,$\lim_{\infty} S_h^{n+1}=\frac{\Lambda_h}{\mu_h}$ as $n\to\infty$

For I_v^{n+1}:

$$I_v^{n+1}=\frac{(\lambda I_h^n S_v^{n+1}+\lambda I_v^n S_v^{n+1})\varphi_5(\triangle t)+I_v^n}{1+(\alpha+\mu_v+\delta_v)\varphi_5(\triangle t)} \tag{36}$$

then,$\lim_{\infty} I_v^{n+1}=0$ as $n\to\infty$

For R_v^{n+1}:

$$R_v^{n+1}=\frac{\alpha I_v^{n+1}\varphi_6(\triangle t)+R_v^n}{1+(\sigma_v+\mu_v)\varphi_6(\triangle t)} \tag{37}$$

then,$\lim_{\infty} R_v^{n+1}=0$ as $n\to\infty$

For S_v^{n+1}:

$$S_v^{n+1} = \frac{(\Lambda_v + \sigma_v R_v^{n+1})\varphi_4(\triangle t) + S_v^n}{1 + (\lambda I_h^n + \lambda I_v^n + \mu_v)\varphi_4(\triangle t)} \tag{38}$$

then, $\lim_{\infty} S_v^{n+1} = \frac{\Lambda_v}{\mu_v}$ as $n \to \infty$

Hence, the DFE is GAS since the condition (i) and (ii) are satisfied for every value of $(\triangle t)$.

6. Basic Reproductive Number

Zoonotic reproductive number is the number of secondary cases produced on average by one infected human or animal in a completely susceptible population. It combines the biology of infections with social and behavioural factors influencing contact rate [27, 28]. It is a threshold parameter that determines spread of infections.

The zoonotic reproductive number is given by the relation;

$$R_{hv} = \left(\frac{\beta\Lambda_h}{\mu_h(\gamma) + (\mu_h + \delta_h)}\right) + \left(\frac{\lambda\Lambda_v}{\mu_v[(\alpha) + (\mu_v + \delta_v)]}\right) . \tag{39}$$

where;

$$R_{hq} = \left(\frac{\beta\Lambda_h}{\mu_h(\gamma) + (\mu_h + \delta_h)}\right) \tag{40}$$

is for human population and

$$R_{vq} = \frac{\lambda\Lambda_v}{\mu_v[\alpha + (\mu_v + \delta_v)]} \tag{41}$$

for animal population.

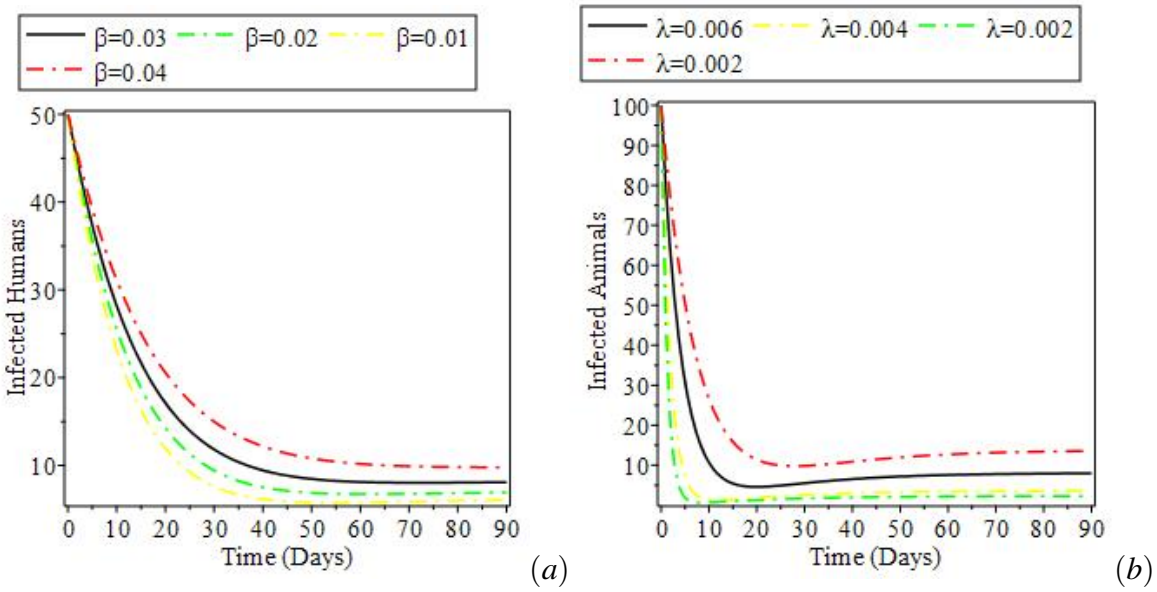
7. Numerical Analysis

In this section, we performed the quantitative analysis of the zoonotic model by solving the system of equation in Figure 1 numerically. This was done by performing the analysis of contact rates and recovery rates on humans and animals as shown in Figure 2 and Figure 3 respectively. We assume the following parameter values; $\beta = 0.03$, $\lambda = 0.004$, $\Lambda_v = 0.005$, $\Lambda_h = 0.002$, $\alpha = 0.05$, $\mu_v = 0.0002$, $\mu_h = 0.0001$, $\delta_v = 0.003$, $\delta_h = 0.001$, $\sigma_h = 0.004$ and $\sigma_v = 0.007$ in our simulations.

Analysis of contact rates; (β) and (λ) on infected humans and animals:

Figure $2(a)$ shows 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.

Figure $2(b)$ shows analysis of contact rate, (λ) on infected 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. Infected human population decreases monotonically as compared to infected animal population is a clear indication of variations in contact rate between human and animal populations as shown in Figure $2(a)$ and Figure $2(b)$.



(a) (b)

FIGURE 2. Effects of contact rates: (β) and (λ) on infected humans and animals.,

Analysis of recovery rates; (γ) and (α) on recovered humans and animals:

Figure $3(c)$ shows analysis of recovery rate, (γ) on recovered humans. As the recovery rate, (γ) increases, there is a corresponding increase in the number of recovered humans. As the recovery rate, (γ) decreases, there is a corresponding decrease in the number of recovered

humans. Hence, recovery can be achieved by ensuring that the value of recovery rate, (γ) apreciates.

Figure $3(d)$ shows analysis of recovery rate, (α) on recovered animals. As the recovery rate increases, we observe an increase in the number of recovered animals. As the value of recovery rate decreases, there is a corresponding decrease in the number of recovered animals. Hence, recovery can be achieved by ensuring that the value of recovery rate, (α) appreciates. Recovery in human increases monotonically as compared to recovery in animal population clearly indicates variations in rate of recovery in human and animal populations as shown in Figure $3(c)$ and Figure $3(d)$.

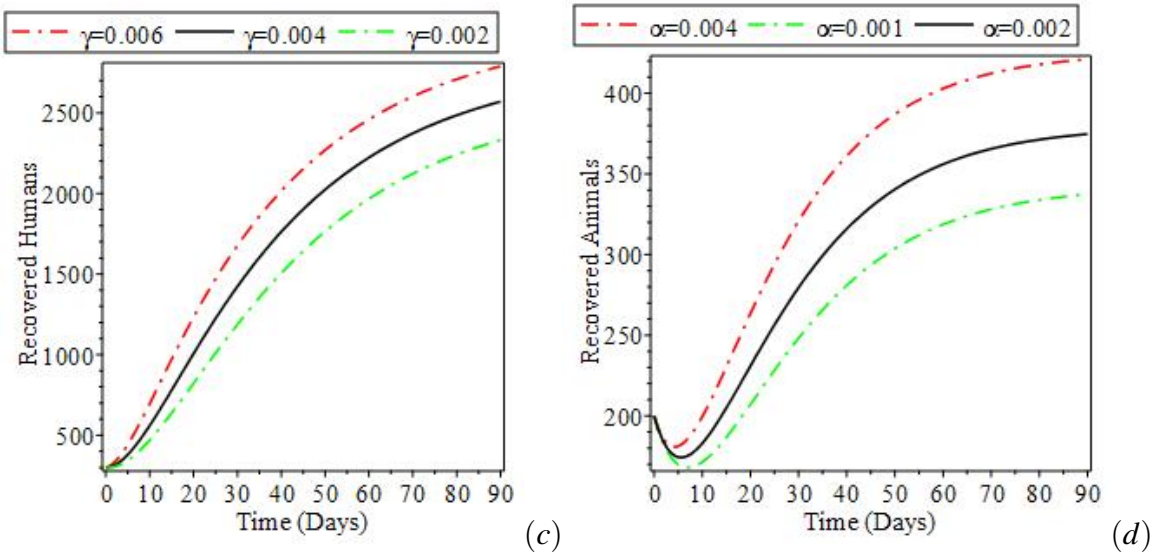


FIGURE 3. Effects of recovery rates: (γ) and (α) on recovered humans and animals.,

7.1. Bifurcation and Multiple Equilibria Analysis. The phenomenon of backward ifurcation is been considered. The idea of bifurcating endemic equilibrium exists only when the basic reproduction number is less than unity. Our zoonotic model has exhibited this property and backward bifurcation exists.

When backward bifurcation occurs, the range of the reproduction number is between $R^*_{hv} < R_{hv} < 1$. There exits at least one endemic equilibrium. Where least one is stable and the disease free equilibrium is not globally stable when the basic reproduction number is less than unity [29, 30, 31, 32, 33].

In this happenings, infection would exist even when $R_{hv} < 1$. Figure 4 is the backward bifurcation diagram of the force of infection against the basic reproduction number that our zoonotic model has exhibited.

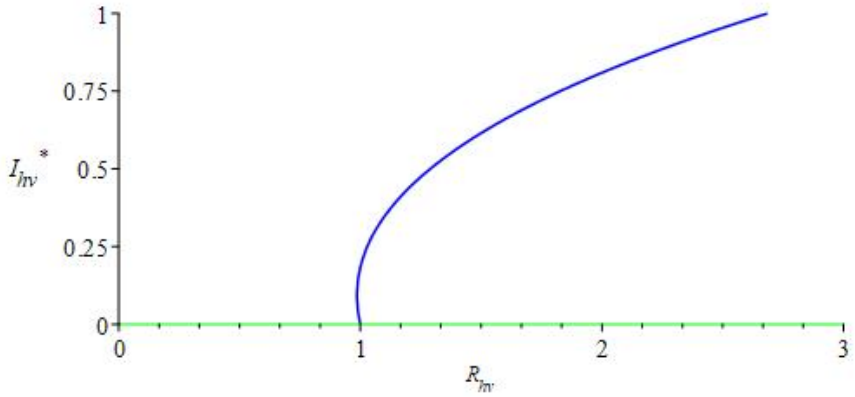


FIGURE 4. Backward bifurcation of the endemic equilibrium when $R_{hv} > 1$

8. CONCLUSION

A deterministic model that explains the spread dynamics of zoonotic diseases was formulated and analysed for qualitatively and quantitatively. The qualitative analysis of the zoonotic model was carried out using Nonstandard Finite Difference Scheme for boundedness of solution, disease free equilibrium and its local and global stability. Zoonosis free equilibrium of the scheme in its explicit form was established. Analysis of the scheme established that the zoonosis free equilibrium was both locally and globally asymptotically stable.

An analysis of the effects of contact rate between susceptible and infected animals as well 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.

ACKNOWLEDGMENT

Authors sincerely acknowledged and expressed their profound appreciation to colleagues for their reviews, comments and suggestions.

Profound appreciation goes to the UK-Africa Postgraduate Advanced Study Institute in Mathematical Sciences (UK-APASI) for providing support in the form of data and knowledge through workshop in mathematical modelling.

SOURCE OF FUNDING

No sources of funding for this research.

DATA AVAILABILITY STATEMENT

Parameter values used in this paper are assumed and others are taken from published articles. These published articles have been cited at relevant places within the text as references.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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