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Stability Analysis and Modelling of Listeriosis Dynamics in Human and Animal Populations

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Abstract

Listeria monocytogene is the causative agent of Listeriosis. It can be found mostly in the environment and it is responsible for meningoencephalitis and stillbirths in animals and humans.The objective of this study is to develop a mathematical model that explains the dynamics of Listeriosis in human and animal population. The model comprises of essential components like vaccination of susceptible vectors, livestock(vector) compartment and human compartment. We investigated the existence of the disease free equilibrium and the basic reproductive number. The disease free equilibrium was found to be locally asymptotically stable whenever the the basic reproduction number is less than unity. The model exhibited the existence of multiple endemic equilibrium. Backward bifurcation diagram showed the existence of multiple endemic equilibrium. Sensitivity analysis was used to determine the impact of each parameter on the basic reproductive number. We further carried out numerical simulations of the system of differential equations for interpretations. The effects of force of infection was analysed by varying the value of the force of infection. A decrease in the value decreases the number of infectious vector and

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human population. Our results showed a continuous increase in the number of susceptible vector, human population. The number of infectious vector and infectious human populations increase with time. This is as a result of the absence of optimal control strategies in our model.

AMS subject classification:
Keywords: Listeriosis model, basic reproductive number, endemic equilibrium, sensitivity analysis, stability analysis.

1. Introduction

Listeriosis is an infectious disease that is mostly responsible for stillbirth in animal and human populations. The causative agent of Listeriosis is Listeria monocytogenes [11]. With the advancement in immunosuppressive drugs and chemotherapy for malignancy in the early 1960s, the disease in adult patients with compromised immune systems was appreciated. Listeriosis is less common but it continues to pose a series of problems in veterinary medicine. Rhombencephalitis in animals had been linked with ingestion of silage that are being contaminated with listeria monocytogenes. This revelation from veterinary medicine has made epidemiologists to speculate that food borne infection could be responsible for human Listeriosis ([17]). The investigations of the outbreak of the disease during the early 1980s revealed that Listeriosis is a food borne disease [18]. Some bacteria can be useful in the food processing industries [20]. They are useful in the production of cheese, chemicals, yogurt and medicines. However, most zoonotic diseases that are bacteria related can be treated by antibiotics [22].

In recent times, mathematical models describing the phenomenon and dynamics of infectious diseases have played a key role in the control of diseases in epidemiology [15]. Many authors have proposed several nonlinear incidence rates to model the disease transmission dynamics. Complex transmission dynamics of some diseases such as periodic orbits, Hoff bifurcations and multiple equilibrium have been described. [4, 8]. A study conducted by [13] showed that Listeria monocytogenes is a food borne pathogen that is responsible for the cause of serious invasive illness, mostly in certain class of individuals including elderly and immune compromised patients, new born children and pregnant women. In a study conducted by [6], showed that Listeria monocytogenes has been rated among the most increasing and major food-associated pathogen and many countries of the European Union have always recorded an annual cases of human Listeriosis.

Investigations conducted by [9] on the incidence and transmission of listeria monocytogenes in ready-to-eat products in retail and food services environments proved that contamination of food products with Listeria monocytogenes can exist or show up at multiple stages before consumption. A research conducted by [14] showed that Listeria monocytogenes is among the food borne pathogens responsible for invasive illness in certain class of people and it can cause infections in the central nervous system. The outbreaks of the disease has been reported in Japan, North America and Europe. The commonest sources of getting infected with the disease are raw milk and meats [19].

In an article published by [10], indicated that Listeriosis in human are rare but it is among the top serious food borne diseases in susceptible and vulnerable individuals in a population such as the immune compromised and pregnant women. The resurgence of food borne Listeriosis was investigated by [1]. The clinical manifestations of Listeriosis can range from febrile gastroenteritis to a severe invasive forms like meningitis, perinatal infections, abortions and sepsis. Moreover, [3], stated Listeriosis as one of leading causes of death from food borne pathogens. The most recent outbreak of Listeriosis in the United States had to do with the consumption of hot dogs. Approximately, 101 cases of illness were recorded with 21 deaths reported at the Centers for Disease Control and Prevention between the years 1998-1999. An investigation conducted by [5] showed that listeria monocytogenes are the causative agent of gastrointestinal infections. The intestinal tract can be the main point of entry for Listeria monocytogenes. These are ingested via contaminated food substances. [2] researched on the identification and reservours of pathogens for effective control of sporadic disease and epidemics. Listeria monocytogenes is among the major zoonotic food borne pathogen that is responsible for approximately twenty eight percent of most food-related deaths in the United States annually.

2. Listeriosis Model Description and Formulation

In this section, the model divides the total human and vector populations at any time (t) into seven compartments with respect to disease status in the system. The total vector population, represented by $N_v(t)$, is divided into compartments of Susceptible vector (S_v), Infectious vector (I_v), Vaccinated vector (V_v), and Recovered vector (R_v). Therefore;

$$N_v(t) = S_v(t) + V_v(t) + I_v(t) + R_v(t).$$

The total human population represented by N_h, is divided into compartments of Susceptible humans (S_h), Infected humans (I_v), and Recovered humans (R_v). This gives;
$N_h(t) = S_h(t) + I_h(t) + R_h(t)$.

The Susceptible humans are recruited into the population at a rate Λ_h. Susceptible humans acquire Listeriosis through inhalation of spores, ingestion of contaminated foods from infected animals, contact with infectious animals and humans at a rate $(I_v + I_h)\,\beta$. Individuals recover from the disease at a rate γ. Humans who are infected with Listeriosis die at a rate δ_h and the recovered humans may loose immunity and return to the susceptible compartment at a rate σ_h. The natural death rate of the entire human compartments is μ_h.

The susceptible vector S_v are recruited into the population at a rate Λ_v, but a fraction of the animals are successfully vaccinated at a rate u_3, where $u_3 \in [0, 1]$. Listeriosis can be acquired through contacts with infectious animals and humans at a rate $(I_v + I_h)\,\lambda$. The natural death rate of the animals is $\mu_v$and the death rate as a result of the disease is δ_v. The animals recover at a rate α and a fraction of the vaccinated animals may move to the infected animal compartment at a rate $b\beta_m^*\lambda$ due to waning effect. Where $(1 - b) \in [0, 1]$ is the efficacy of the vaccine. This is because the animals may loose

immunity and move back to the susceptible compartment at a rate τ.

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

The following system of ordinary differential equations were obtained from the model:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h + \sigma_h R_h - \beta_m^* \beta S_h - \mu_h S_h \\
\frac{dI_h}{dt} &= \beta_m^* \beta S_h - \left(\gamma + \mu_h + \delta_h\right) I_h \\
\frac{dR_h}{dt} &= \gamma I_h - \left(\sigma_h + \mu_h\right) R_h \\
\frac{dS_v}{dt} &= (1-u_3)\Lambda_v - \beta_m^* \lambda S_v - \mu_v S_v + \sigma_v R_v + \tau V_v \\
\frac{dI_v}{dt} &= \beta_m^* \lambda S_v + b\beta_m^* \lambda V_v - (\alpha + \mu_v + \delta_v) I_v \\
\frac{dR_v}{dt} &= \alpha I_v - (\sigma_v + \mu_v) R_v \\
\frac{dV_v}{dt} &= u_3 \Lambda_v - (\tau + \mu_v) V_v - b\lambda \beta_m^* V_v
\end{aligned}\right\} \tag{2.1}$$

3. Analysis of the Listeriosis model

3.1. Positivity and Boundedness of Solutions

In human population models, the objective is to obtain non-negative solutions. The conditions under which a system of differential equations under study has non-negative solutions is of great importance. Listeriosis model would be biologically meaningful if all the solutions with non-negative initial data remain non-negative at every time. The concept of the derivative of a function would be applied. If the derivative of a function at a point is positive, then the function is said to be increasing at that point. If the derivative of the function at a point is negative, then it is said to be decreasing and if the derivative of the function at a point is equal to zero, then the function is constant.

Theorem 3.1. Let

$$\Pi = \{ (S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), V_v(t), R_v(t)) \in \mathbb{R}_+^7 : (S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), V_v(0), R_v(0)) > 0 \}$$

then the solution of

$$\{(S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), V_v(t), R_v(t))\}$$

are non-negative for all time $t \geq 0$.

This implies that, if

$$S_h(0), I_h(0), R_h(0), S_v(0), I_v(0), V_v(0), R_v(0)$$

are non-negative, then $S_h(t), I_h(t), R_h(t), S_v(t), I_v(t), V_v(t), R_v(t)$ are also non-negative for all time $t > 0$.

Considering the human population in the model:

The total human population at any time $(t)$is given by:

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

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

The above equation can be interpreted as;

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h.$$

Absence of mortality due to Listeriosis infections;

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h. \tag{3.3}$$

Solving the above differential equation; $\Lambda_h - \mu_h N_h \geq Ae^{-\mu_h t}$, where A is constant.

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

We obtain the relation; $\Lambda_h - \mu_h N_{h(0)} = A$

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

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

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

This implies that,

$$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}$$

and

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h}.$$

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

$$\Pi_h = \{(S_h, I_h, R_h) \in \mathbb{R}_3^3 : S_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h}. \tag{3.4}$$

The total vector(livestock) population at any time (t) is given by:

$$N_v(t) = S_v(t) + V_v(t) + I_v(t) + R_v(t). \tag{3.5}$$

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dV_v}{dt} + \frac{dI_v}{dt} + \frac{dR_v}{dt}. \tag{3.6}$$

The above equation can be interpreted as;

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

Absence of mortality due to Listeriosis infections;

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

Solving the differential equation; $\Lambda_v - \mu_v N_v \geq Ae^{-\mu_v t}$, where A is constant.

Applying the initial condition, $N_v(0) = N_{v(0)}$,

We obtain the relation; $\Lambda_v - \mu_v N_{v(0)} = A$

Therefore, $\Lambda_v - \mu_v N_v \geq \left(\Lambda_v - \mu_v N_{v(0)}\right) e^{-\mu_v t}$.

$$N_v \leq \frac{\Lambda_v}{\mu_v} - \left(\frac{\Lambda_v - \mu_v N_{v(0)}}{\mu_v}\right) e^{-\mu_v t}.$$

As $t \to \infty$, the population size, $N_v \to \frac{\Lambda_v}{\mu_v}$.

This implies that, $0 \leq N_v \leq \frac{\Lambda_v}{\mu_v}$ and $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$.

Also, if $N_v(0) \leq \frac{\Lambda_v}{\mu_v}$, then $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$.

$$\Pi_v = \left\{ (S_v, I_v, R_v, V_v) \in \mathbb{R}^4_+ : S_v + I_v + R_v + V_v \leq \frac{\Lambda_v}{\mu_v} \right\}. \tag{3.8}$$

The feasible region for the system of ordinary differential equations in (0.2.1) is given by:

$$\Pi = \Pi_h \times \Pi_v \subset \mathbb{R}^3_+ \times \mathbb{R}^4_4 . \tag{3.9}$$

Where,

$$\Pi_h = \{(S_h, I_h, R_h) \in \mathbb{R}^3_3 : S_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h} \tag{3.10}$$

$$\Pi_v = \left\{ (S_v, I_v, R_v, V_v) \in \mathbb{R}^4_+ : S_v + I_v + R_v + V_v \leq \frac{\Lambda_v}{\mu_v} \right\}. \tag{3.11}$$

Where Π is positively invariant.

3.2. Disease-free equilibrium for Listeriosis model

The disease-free equilibrium of the system of ordinary differential equations in (2.1) only exists when $u_1 = 0$ and all other controls are held constant. This is computed by setting the system of differential equations in (2.1) to zero. This is given by:

$$\xi_0 = \left(\begin{array}{cccccc} S_h^*, & I_h^*, & R_h^*, & S_v^*, & I_v^* & R_v^* \quad V_v^* \end{array} \right).$$

$$\frac{dS_h}{dt} = \Lambda_h + \sigma_h R_h \quad -\beta_m^* \beta S_h \quad -\mu_h S_h \quad = 0. \tag{3.12}$$

$S_h^* = \frac{\Lambda_h}{\mu_h}$.

At disease free equilibrium (DFE), there are no infections and recovery.

$$\left.\begin{array}{rcl} I_h^* & = & 0 \\ R_h^* & = & 0 \end{array}\right\}, \qquad \left.\begin{array}{rcl} I_v^* & = & 0 \\ R_v^* & = & 0 \end{array}\right\}$$

Now considering the vector (Livestock) population: At disease free equilibrium, there are no infections and recovery.

$$\frac{dV_v}{dt} = u_3 \Lambda_v \quad -(\tau + \mu_v) V_v \quad -b\lambda \beta_m^* V_v \quad = 0 \tag{3.13}$$

$u_3 \Lambda_v \quad -(\tau + \mu_v) V_v \quad = 0$

$V_v^* = \frac{u_3 \Lambda_v}{\tau + \mu_v}$. Also, from the relation;

$$\frac{dS_v}{dt} = (1 - u_3) \Lambda_v \quad -\beta_m^* \lambda S_v \quad -\mu_v S_v + \sigma_v R_v \quad +\tau V_v \quad = 0 \tag{3.14}$$

$(1 - u_3) \Lambda_v \quad -\mu_v S_v + \tau V_v \quad = 0$

$S_v^* = \frac{(1 - u_3) \Lambda_v + \tau V_v}{\mu_v}$.

But $V_v^* = \frac{u_3 \Lambda_v}{\tau + \mu_v}$.

$S_v^* = \frac{\Lambda_v (\tau + \mu_v (1 - u_3))}{\mu_v (\tau + \mu_v)}$.

$$\xi_0 = \left(\frac{\Lambda_h}{\mu_h}, \quad 0, \quad 0, \quad \frac{\Lambda_v (\tau + \mu_v (1 - u_3))}{\mu_v (\tau + \mu_v)}, \quad 0, \quad 0, \quad \frac{u_3 \Lambda_v}{\tau + \mu_v} \right). \tag{3.15}$$

3.3. The Basic Reproductive Number

In this section, we use the concepts of Next Generation Matrix to established the linear stability of the disease-free equilibrium (ξ_0). We computed the basic reproduction number. The basic reproductive number or rate is the number of secondary infections

produced by one infected animal or person in a completely susceptible population. The reproductive number combines the biology of infections with the social and behavioral factors causing contact rates [21, 12]. The basic reproductive number is the threshold parameter that governs the spread of a disease.

The next-generation matrix is defined as; $K = FV^{-1}$ and $R_{hv} = \rho\left(FV^{-1}\right)$.

Where $\rho\left(FV^{-1}\right)$denotes the spectral radius of FV^{-1}.

The basic reproductive number R_{hv}, is defined as the spectral radius of the next-generation matrix.

Definition 3.2. The spectral radius of a matrix A is defined as the maximum of the absolute values of the eigenvalues of the matrix A : $\rho(A) = sup\{|\lambda| : \lambda \epsilon \rho(A)\}$, where $\rho(A)$ represents the set of eigenvalues of the matrix A.

Using the Next Generation Matrix, we consider only the infective classes in the system of differential equations in (0.2.1) :

$$\left.\begin{aligned} \frac{dI_h}{dt} &= (I_h + I_v)\beta S_h - \left(\gamma + \mu_h + \delta_h\right) I_h \\ \frac{dI_v}{dt} &= (I_h + I_v)\lambda S_v + (I_h + I_v) b\lambda V_v \quad - (\alpha + \mu_v + \delta_v) I_v \end{aligned}\right\} \tag{3.16}$$

$$f = \begin{bmatrix} (I_h + I_v)\beta S_h \\ \\ (I_h + I_v)\lambda S_v \quad + \quad (I_h + I_v) b\lambda V_v \end{bmatrix}, \qquad v = \begin{bmatrix} \left(\gamma + \mu_h + \delta_h\right) I_h \\ \\ (\alpha + \mu_v + \delta_v) I_v \end{bmatrix}.$$

Where f is the number of new infection coming into the system and v is the number of infectives that are leaving the system either by death or birth.

The Jacobian matrix of f and v at disease free equilibrium is obtained by F and V as follows:

$$F = \begin{bmatrix} \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial I_v} \\ \\ \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \beta S_h^* & \beta S_h^* \\ \\ \lambda S_v^* + b\lambda V_v^* & \lambda S_v^* + b\lambda V_v^* \end{bmatrix}, \tag{3.17}$$

$$V = \begin{bmatrix} \frac{\partial v_1}{\partial I_h} & \frac{\partial v_1}{\partial I_v} \\ \\ \frac{\partial v_2}{\partial I_h} & \frac{\partial v_2}{\partial I_v} \end{bmatrix} = \begin{bmatrix} \left(\gamma + \delta_h + \mu_h\right) & 0 \\ \\ 0 & (\alpha + \delta_v + \mu_v) \end{bmatrix}. \tag{3.18}$$

By computing the product of FV^{-1}.

$$FV^{-1} = \begin{bmatrix} \beta S_h^* & \beta S_h^* \\ \lambda S_v^* + b\lambda V_v^* & \lambda S_v^* + b\lambda V_v^* \end{bmatrix} \begin{bmatrix} \frac{1}{(\gamma + \delta_h + \mu_h)} & 0 \\ 0 & \frac{1}{(\alpha + \delta_v + \mu_v)} \end{bmatrix}.$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta S_h^*}{(\gamma + \delta_h + \mu_h)} & \frac{\beta S_h^*}{(\alpha + \delta_v + \mu_v)} \\ \frac{\lambda S_v^* + b\lambda V_v^*}{(\gamma + \delta_h + \mu_h)} & \frac{\lambda S_v^* + b\lambda V_v^*}{(\alpha + \delta_v + \mu_v)} \end{bmatrix} \tag{3.19}$$

Now, computing the eigenvalues of FV^{-1} and selecting the dominant eigenvalue. Let A represent the eigenvalue of the matrix.

$$\begin{vmatrix} \frac{\beta S_h^*}{(\gamma + \delta_h + \mu_h)} - A & \frac{\beta S_h^*}{(\alpha + \delta_v + \mu_v)} \\ \frac{\lambda S_v^* + b\lambda V_v^*}{(\gamma + \delta_h + \mu_h)} & \frac{\lambda S_v^* + b\lambda V_v^*}{(\alpha + \delta_v + \mu_v)} - A \end{vmatrix} = 0 \tag{3.20}$$

By expansion and re-arranging;

$$A^2 - \left[\left(\frac{\lambda S_v^* + b\lambda V_v^*}{(\alpha + \delta_v + \mu_v)} \right) + \left(\frac{\beta S_h^*}{(\gamma + \delta_h + \mu_h)} \right) \right] A = 0 \tag{3.21}$$

Solving the above quadratic equation; $A_1 = 0$ and

$$A_2 = \left[\left(\frac{\lambda S_v^* + b\lambda V_v^*}{(\alpha + \delta_v + \mu_v)} \right) + \left(\frac{\beta S_h^*}{(\gamma + \delta_h + \mu_h)} \right) \right].$$

Therefore, the dominant eigenvalue is A_2. This implies that;

$$R_{hv} = \left[\left(\frac{\beta S_h^*}{(\gamma + \delta_h + \mu_h)} \right) + \left(\frac{\lambda S_v^* + b\lambda V_v^*}{(\alpha + \delta_v + \mu_v)} \right) \right]. \tag{3.22}$$

But at disease free equilibrium,

$$S_h^* = \frac{\Lambda_h}{\mu_h},$$

$$S_v^* = \frac{\Lambda_v \left(\tau + \mu_v \left(1 - u_3\right)\right)}{\mu_v \left(\tau + \mu_v\right)},$$

and $V_v^* = \dfrac{u_3 \Lambda_v}{\tau + \mu_v}$.

By substituting the above into the basic reproductive numberR_{hv} and rearranging:

$$R_{hv} = \frac{\beta \Lambda_h}{\mu_h \left(\gamma + \delta_h + \mu_h\right)} + \frac{b\lambda \Lambda_v \left(\tau + \mu_v \left(1 - 2u_3\right)\right)}{\mu_v \left(\tau + \mu_v\right)\left(\alpha + \mu_v + \delta_v\right)} \tag{3.23}$$

Vaccination of the susceptible animals would result in the reduction of R_{hv}. Moreover, the total vaccination coverage would be:

$$u_3^* = \frac{1}{1-b}\left(\frac{R_{vq}\left(\tau + 1\right) + R_{hq} - R_{hv}}{R_{vq}}\right) \tag{3.24}$$

Where,

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

$$R_{vq} = \frac{b\lambda \Lambda_v \left(\tau + \mu_v \left(1 - 2u_3\right)\right)}{\mu_v \left(\tau + \mu_v\right)\left(\alpha + \mu_v + \delta_v\right)} \quad .$$

Proposition 3.3. The disease-free equilibrium (DFE) of model (0.2.1) is locally asymptotically stable if $R_{hv} < 1$, and unstable if $R_{hv} > 1$.

3.4. Global stability of the disease-free equilibrium

Theorem 3.4. If $R_{hv} \leq 1$, the disease-free equilibrium is globally asymptotically stable in the interior of Ω.

Proof. Considering the Lyapunov function below,

$$P\left(t\right) = \left(\alpha + \mu_v + \delta_v\right) I_h + \left(\gamma + \mu_h + \delta_h\right) I_v \tag{3.25}$$

By computing the time derivative of P along the solutions of the system of ordinary

differential equations in(2.1), the following is obtained,

$$\left.\begin{aligned}
\frac{dP(t)}{dt} = & (\alpha+\mu_v+\delta_v)\frac{dI_h}{dt} + \left(\gamma+\mu_h+\delta_h\right)\frac{dI_v}{dt} \\
= & (\alpha+\mu_v+\delta_v)\left(\beta S_h(I_h+I_v) - \left(\gamma+\mu_h+\delta_h\right)I_v\right) \\
& + \left(\gamma+\mu_h+\delta_h\right)[\lambda S_v(I_h+I_v) \\
& + b(I_h+I_v)\lambda V_v - (\alpha+\mu_v+\delta_v)I_v] \\
\leq & (\alpha+\mu_v+\delta_v)\frac{\beta\Lambda_h I_h}{\mu_h} + (\alpha+\mu_v+\delta_v)\frac{\beta\Lambda_h I_v}{\mu_h} \\
& - (\alpha+\mu_v+\delta_v)\left(\gamma+\mu_h+\delta_h\right)I_h \\
& + I_h\left(\gamma+\mu_h+\delta_h\right)\left(\frac{\lambda\Lambda_v(\tau+\mu_v(I-u_3))}{\mu_v(\tau+\mu_v)}\right) \\
& + I_v\left(\gamma+\mu_h+\delta\right)\left(\frac{\lambda\Lambda_v(\tau+\mu_v(I-u_3))}{\mu_v(\tau+\mu_v)}\right) \\
& + I_h\left(\gamma+\mu_h+\delta_h\right)\left(\frac{bu_3\lambda\Lambda_v}{\tau+\mu_v}\right) + I_v\left(\gamma+\mu_h+\delta\right)\left(\frac{bu_3\lambda\Lambda_v}{\tau+\mu_v}\right) \\
& - I_v\left(\gamma+\mu_h+\delta\right)(\alpha+\mu_v+\delta_v) \\
\leq & -I_h\left(\gamma+\mu_h+\delta_h\right)(\alpha+\mu_v+\delta_v)(1-R_{hv}) \\
& - I_v\left(\gamma+\mu_h+\delta_h\right)(\alpha+\mu_v+\delta_v)(1-R_{hv}) \\
= & -(I_h+I_v)\left(\gamma+\mu_h+\delta_h\right)(\alpha+\mu_v+\delta_v)(1-R_{hv})
\end{aligned}\right\} \tag{3.26}$$

The time derivative of P along the solutions of the system of differential equations in (2.1) gives the following:

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

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

Therefore, the highest compact invariant set in $S_h,\ I_h,\ I_v,\ \in\ \Omega,\ \frac{dP(t)}{dt} = 0$, if $R_{hv} \leq 1$, is the singleton ξ_0.

This implies that is ξ_0 globally asymptotically stable in Ω. By LaSalle's invariant principle [7].

3.5. Endemic Equilibrium

Considering the system of differential equations in 0.2.1, at equilibrium, $\beta_m^* = I_h + I_v = 0$. This corresponds to the disease free equilibrium or the relation:

$$\Omega_0 \beta_m^{*3} + \Omega_1 \beta_m^{*2} + \Omega_2 \beta_m^* + \Omega_3 = 0. \tag{3.27}$$

$$\Omega_0 = 1, \Omega_1 = \frac{Z_*}{E}(1 - R_w), \Omega_2 = \frac{G_1}{E}\left(1 - R_f\right), \Omega_3 = \chi\left(1 - R_{hv}\right).$$

Where;

$$\begin{aligned}
R_{hv}^2 &= R_{hq} + R_{vq} = \frac{\beta \Lambda_h}{\mu_h\left(\gamma + \delta_h + \mu_h\right)} + \frac{\lambda \Lambda_v \left[\left(\tau + (1 - (1 - b))\, u_3\right)\right]}{\left(\tau + \mu_v\right)\left(\alpha + \delta_v + \mu_v\right)}, \\
E &= b\beta\lambda^2 \left[\mu_v\left(\alpha + \delta_v + \mu_v\right) + \left(\mu_v + \delta_v\right)\sigma_v\right]\left[\mu_h\left(\gamma + \delta_h + \mu_h\right) + \left(\mu_h + \delta_h\right)\sigma_h\right], \\
Q_1 &= b\lambda^2\left(\gamma + \delta_h + \mu_h\right)\left(\mu_h + \sigma_h\right)\left[\mu_v\left(\alpha + \delta_v + \mu_v\right) + \left(\mu_v + \delta_v\right)\sigma_v\right], \\
Q_2 &= \frac{\beta\lambda\left[\mu_h\left(\gamma + \delta_h + \mu_h\right) + \left(\mu_h + \delta_h\right)\sigma_h\right] F_3 b\left(\mu_v + \delta_v\right)\left(\tau + \mu_v\right)\left(\alpha + \delta_v + \mu_v\right)}{\left(\tau + (1 - (1 - b))\, u_3\right)}, \\
Z_* &= Q_1 + Q_2, \\
R_w^2 &= \frac{Q_1 R_{hq} + Q_2 R_{vq}}{Z_*}, \\
F_1 &= \lambda\mu_h\left(\mu_h + \sigma_h\right)\left(\gamma + \delta_h + \mu_h\right)\left(\tau + (1 + b)\,\mu_v\right)\left[\mu_v\left(\alpha + \delta_v + \mu_v\right)\right. \\
&\quad \left. + \left(\mu_v + \delta_v\right)\sigma_v\right], \\
F_2 &= \beta\mu_h\left(\alpha + \delta_v + \mu_v\right)\left(\tau + \mu_v\right)\left(\mu_v + \sigma_v\right)\left[\mu_h\left(\gamma + \delta_h + \mu_h\right) + \left(\mu_h + \delta_h\right)\sigma_h\right], \\
F_3 &= \left[\left(\tau + (1 + b)\,\mu_v\right)\left[\mu_v\left(\alpha + \delta_v + \mu_v\right) + \left(\mu_v + \delta_v\right)\sigma_v\right] + b\alpha\mu_v\right], \\
G_1 &= \lambda\left[\mu_h\left(\mu_h + \sigma_h\right)\left(\gamma + \delta_h + \mu_h\right)\left(\tau + \mu_v\right)\left[\mu_v\left(\alpha + \delta_v + \mu_v\right) + \left(\mu_v + \delta_v\right)\sigma_v\right]\right] \\
&\quad - b\left[\Lambda_v\left(\mu_v + \sigma_v\right) + \beta\Lambda_h\alpha\mu_v\sigma_v\left(\mu_h + \sigma_h\right) + \left(\alpha + \delta_v + \mu_v\right)\left(\mu_v + \sigma_v\right)\right], \\
\chi &= \frac{\mu_h\mu_v\left(\mu_v + \sigma_v\right)\left(\mu_h + \sigma_h\right)\left(\tau + \mu_v\right)\left(\alpha + \delta_v + \mu_v\right)\left(\gamma + \delta_h + \mu_h\right)}{b\beta\lambda^2\left[\mu_v\left(\alpha + \delta_v + \mu_v\right) + \left(\mu_v + \delta_v\right)\sigma_v\right]\left[\mu_h\left(\gamma + \delta_h + \mu_h\right) + \left(\mu_h + \delta_h\right)\sigma_h\right]}, \\
R_f^2 &= \frac{F_1^2 R_{hq} + F_1^2 R_{vq}}{G_1},
\end{aligned} \tag{3.28}$$

Remark 3.5. The system of differential equations in equation (2.1) is said to have an endemic equilibrium E^*, if $R_{hv} > 1$. This is satisfied by cases (2, 4, 6) in (1) . The system of differential equations can have more than one endemic equilibrium points if $R_{hv} > 1$. This is satisfied by case (8) in table (1). The system of differential equations in equation (2.1) , have more than one equilibrium points if $R_{hv} < 1$, as satisfied by case (3, 5, 7).

Table 1: Possible positive real roots of $P\left(\beta_m^*\right)$ for $R_{hv}>1$ and $R_{hv}<1$.

Cases	Ω_0	Ω_1	Ω_2	Ω_3	R_{hv}	No. of sign change	No. of positive real roots
1	+	+	+	+	$R_{hv}<1$	0	0
2	+	+	+	-	$R_{hv}>1$	1	1
3	+	+	-	+	$R_{hv}<1$	2	0,2
4	+	+	-	-	$R_{hv}>1$	1	1
5	+	-	-	+	$R_{hv}<1$	2	0,2
6	+	-	-	-	$R_{hv}>1$	1	1
7	+	-	+	+	$R_{hv}<1$	2	0,2
8	+	-	+	-	$R_{hv}>1$	3	1,3

3.6. Global stability of endemic equilibrium

The Global behaviour of the system of differential equations in equation (2.1) is analysed.

Theorem 3.6. The system of differential equations in equation (2.1), is said to have a unique endemic equilibrium if $R_{hv}>1$, and it is globally asymptotically stable.

The endemic equilibrium can only exists if and only if $R_{hv}>1$.
So by letting $R_{hv}>1$, it implies that the endemic equilibrium exists.
Considering the non-linear Lyapunov function bellow;

$$\left.\begin{aligned} L &= S_h^{**}\left(\frac{S_h}{S_h^{**}}-ln\frac{S_h}{S_h^{**}}\right) + I_h^{**}\left(\frac{I_h}{I_h**}-ln\frac{I_h}{I_h^{**}}\right) \\ &+ \frac{f_1R_h^{**}}{\gamma}\left(\frac{R_h}{R_h^{**}}-ln\frac{R_h}{R_h**}\right) + S_v^{**}\left(\frac{S_v}{S_v^{**}}-ln\frac{S_v}{S_v^{**}}\right) \\ &+ I_v^{**}\left(\frac{I_v}{I**}-ln\frac{I_v}{I_v^{**}}\right) + R_v^{**}\left(\frac{R_v}{R_h^{**}}-ln\frac{R_v}{R**}\right) \\ &+ V_v^{**}\left(\frac{Vv}{V_v^{**}}-ln\frac{V_v}{V_v^{**}}\right). \end{aligned}\right\} \tag{3.29}$$

Where;

$$f_1 = \left(u_2\gamma+\mu_h+\delta_h\right), \qquad f_2 = \left(\sigma_2+\mu_h\right),$$

$$f_3 = \left(u_4\gamma\alpha+\mu_v+\delta_v\right), \qquad f_4 = \left(\sigma_v+\mu\right),$$

When the above Lyapunov function is differentiated with respect to time, we obtain

the equation;

$$\begin{cases} \dfrac{dL}{dt} = \left(1-\dfrac{S_h^{**}}{S_h}\right)\dfrac{dS_h}{dt} + \left(1-\dfrac{I_h^{**}}{I_h}\right)\dfrac{dI_h}{dt} + \dfrac{f_1}{\gamma}\left(1-\dfrac{R**}{R_h}\right)\dfrac{dR_h}{dt} \\ \quad + \left(1-\dfrac{S_v^{**}}{S_v}\right)\dfrac{dS_v}{dt} + \left(1-\dfrac{I_v^{**}}{I_v}\right)\dfrac{dI_v}{dt} + \left(1-\dfrac{R_v^{**}}{R_v}\right)\dfrac{dR_v}{dt} \\ \quad + \left(1-\dfrac{V_v^{**}}{V_v}\right)\dfrac{dV_v}{dt}. \end{cases} \tag{3.30}$$

Therefore, this implies that;

$$\begin{cases} \dfrac{dL}{dt} = \left(1-\dfrac{S_h^{**}}{S_h}\right)\left[\Lambda_h + \sigma_h R_h^{**} + \beta\beta_m^{**} S_h^{**} + \mu_h S_h^{**} - \Lambda_h\right. \\ \quad \left. -\sigma R_h - \beta\beta_m S_h - \mu_h S_h\right] + \left(1-\dfrac{I_h^{**}}{I_h}\right)\left[\beta\beta_m S_h + f_1 I_h\right. \\ \quad \left. + \dfrac{f_1}{\gamma}\left(1-\dfrac{R_h^{**}}{R_h}\right)\left[\gamma I_h - f_2 R_h\right] + \left(1-\dfrac{S_v^{**}}{S_v}\right)\left[(1-u_3)\,\Lambda_v\right.\right. \\ \quad \left.\left. + \lambda\beta\beta_m^{**} S_v^{**} + \mu_v S_v^{**} + \sigma_v R_v^{**} + \tau V_v^{**} - (1-u_3)\,\Lambda_v\right.\right. \\ \quad \left.\left. - \lambda\beta_m S_v - \mu_v S_v - \sigma_v R_v - \tau V_v\right] + \left(1-\dfrac{I_v^{**}}{I_v}\right)\right. \\ \quad \left. \left[\lambda\beta_m S_v + b\lambda\beta_m V_v - f_3 I_v\right] + \dfrac{f_3}{\alpha}\left(1-\dfrac{R_v^{**}}{R_v}\right)\right. \\ \quad \left. [\alpha I_v - f_4 R_v] + \left(1-\dfrac{V_v^{**}}{V_v}\right)[u_3\Lambda_v + b\lambda\beta_m^{**} V_v^{**}\right. \\ \quad \left. + (\tau+\mu_v)\,V_v^{**} - u_3\Lambda_v - b\lambda\beta_m V_v - (\tau+\mu_v)\,V_v\right]. \end{cases} \tag{3.31}$$

Moreover, by further simplification, the following is obtained, basically, however,

the arithmetic mean value exceeds the geometric mean value [16]. This follows that;

$$
\begin{aligned}
2 &- \frac{S_h^{**}}{S_h} - \frac{S_h}{S_h^{**}} \leq 0 \\
1 &- \frac{R_h}{R_h^{**}} \leq 0 \\
1 &- \frac{R_h^{**}}{R_h} - \frac{f_1 f_2 S_h}{\gamma S_h^{**}}\left(1 - \frac{R_h^{**}}{R_h}\right) \leq 0 \\
1 &- \frac{\beta_m}{\beta_h^{**}} - \frac{S_h^{**}}{S_h} - \frac{S_h \beta_m I_h^{**}}{S_h^{**}\beta_m^{**}} \leq 0 \\
1 &- \frac{I_h}{I_h^{**}} - \frac{I_h}{I_h^{**}}\left(1 - \frac{R_h^{**}}{R_h}\right) \leq 0 \\
2 &- \frac{S_v^{**}}{S_v} - \frac{S_v}{S_v^{**}} \leq 0 \\
1 &- \frac{S_v^{**}}{S_v} - \frac{R_v}{R_v^{**}} + \frac{R_v S_v^{**}}{R_v^{**} S_v} \leq 0 \\
1 &- \frac{S_v^{**}}{S_v} - \frac{V_v}{V_v^{**}} - \frac{V_v S_v^{**}}{V_v^{**} S_v} \leq 0 \\
1 &- \frac{S_v^{**}}{S_v} + \frac{\beta_m}{\beta_h^{**}} - \frac{S_v \beta_m I_v^{**}}{S_v^{**}\beta_m^{**} I_v} \leq 0 \\
1 &- \frac{I_v}{I_v^{**}} - f_3\frac{I_v}{I_v^{**}} - f_3\frac{I_v R_v^{**}}{I_v^{**} R_v} \leq 0 \\
1 &- \frac{R_v}{R_v^{**}} \leq 0 \\
2 &- \frac{V_v^{**}}{V_v} - \frac{V_v}{V_v^{**}} \leq 0 \\
1 &- \frac{V_v^{**}}{V_v} + \frac{\beta_m}{\beta_h^{**}} - \frac{V_v \beta_m I_v^{**}}{V_v^{**}\beta_m^{**} I_v}
\end{aligned}
\tag{3.32}
$$

From the assumption that all the model parameters are non-negative, it implies that

the derivative of the Lyapunov function is less than zero $\left(\frac{dL}{dt} \leq 0\right)$, if the basic reproduction number of the system of differential equation in equation (2.1) is greater than one (R_{hv}> 1). Therefore by LaSalle's Invariant Principle [7], as t approaches infinity, all the solution of the equations of the system of differential equations in the model approaches the endemic equilibrium point if R_{hv}> 1.

4. Sensitivity analysis of Listeriosis model

Basically, the essence of sensitivity analysis is to determine how robust a model is to parameter values. This is usually done to help identify the parameters with high impact on the basic reproduction number (R_{hv}). The basic reproduction number is usually analysed to find out whether or not treatment of the infectives, mortality and vaccination could help in the control or eradication of the disease in the population [12].

Definition 4.1. The normalised forward sensitivity index of a variable, q, which depends differentially on a parameter, r, defined as:

$$\Upsilon_r^q = \frac{\partial q}{\partial r} \times \frac{r}{q}. \tag{4.1}$$

4.1. Sensitivity indices of the basic reproduction number R_{hv}

In epidemiological models, the value of the basic reproductive number determines the ability of the disease or infection to spread within the population. We will determine the reduction in infection due to the diseases by computing the sensitivity indices of the basic reproduction Number R_{hv}, with respect to the parameter values in the model. The sensitivity indices serve as determinants of the significance of each parameter in the dynamics and prevalence of the diseases. They measure the change in model variables when a parameter changes. In this study, we will compute the sensitivity indices of R_{hv} to parameter values for the model which will be estimated from data available or already published papers in the literature. Considering the thirteen different parameters of the system of differential equations in model (2.1), we therefore derive the sensitivity of R_{hv} to each of the parameters in the model. Consider the parameter values in table 3 bellow:

The sensitivity indices of the basic reproduction number of R_{hv} with respect to each of the parameters of the system of differential equations in model (2.1), are given in the table bellow:

5. Numerical Results

In this section, we discuss the numerical simulations of the dynamics of Listeriosis in human and animal populations. The state equations are solved over a period of time by employing the Runge-Kutta forth order scheme. The variables and parameters in Table(3), are the descriptions of parameters of the Listeriosis model in Figure 1.

Table 2: Sensitivity indices of parameters to R_{hv}.

Parameter	Description	Sensitivity index(+ve/-ve)
Λ_h	human recruitment rate	+ve
Λ_v	livestock's recruitment rate	+ve
μ_h	death rate in humans	-ve
μ_v	death rate in livestock's	-ve
δ_h	human disease induced death rate	-ve
δ_v	livestock's disease induced death rate	-ve
u_3	proportion vaccinated	-ve
α	livestock's recovery rate	-ve
β	human transmission rate	+ve
γ	human rate of recovery	-ve
τ	waning rate	+ve
λ	livestock transmission rate	+ve
b	vaccine efficacy	+ve

Table 3: Variable and parameter values of Listeriosis model.

Parameter	Estimated value	Reference
μ_h	0.004	assumed
δ_h	0.20	Adak et al., 2002.
Λ_v	0.10	assumed
Λ_h	0.025	assumed
μ_v	0.002	assumed
δ_v	0.30	Adak et al., 2002.
α	0.002	assumed
β	0.02	assumed
τ	0.013	assumed
λ	0.27	https://www.listeria.com
b	0.005	assumed

5.1. Bifurcation Analysis

Figure 1 shows the simulations of the Listeriosis model indicating the existence of backward bifurcation. Epidemically, the implication is that, the necessary condition for anthrax eradication when the basic reproduction number is less than unity is no longer applicable. Backward bifurcation in model means it is not sufficient to look at the dynamics of Listeriosis disease based on only the basic reproduction number.

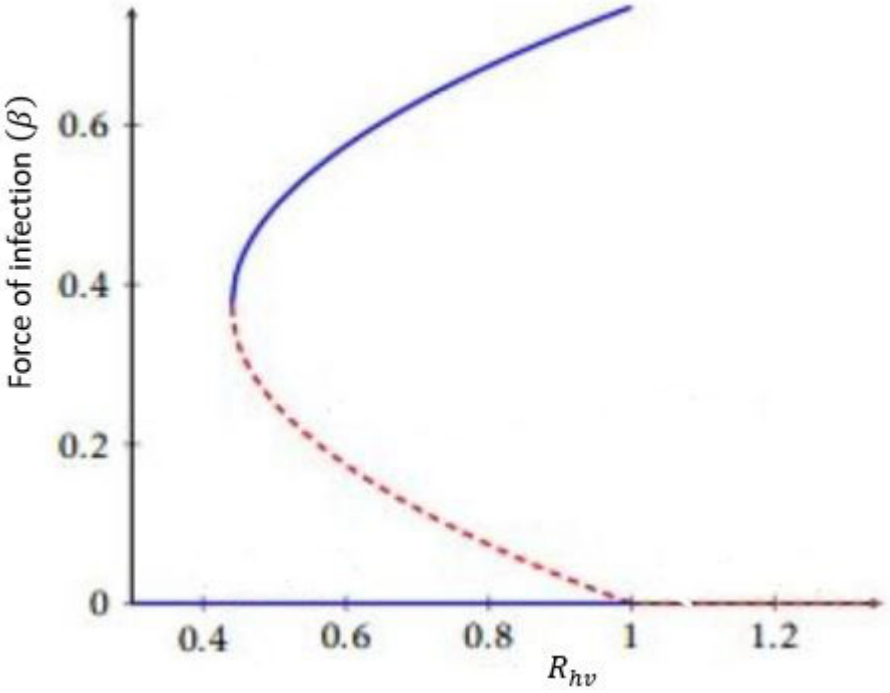

Figure 1: Simulation of the model indicating the existence of backward bifurcation.

5.2. Simulations of the model showing the effects of the force of infection (β) on infectious vector and human populations

In this section, we simulate the model by varying the value of the force of infection (β) to see its effects on the infectious human and vector populations. Figure 2 shows the effects of the force of infection on the population of the infectious human and vector populations. As the value of (β) decreases, the population of the infectious vector reduces. Moreover, a decrease in the value of (β), reduces the number of the infectious human population.

5.3. Simulations of Listeriosis model showing infectious human and vector populations

Figure 4 shows the simulations of the Listeriosis model indicating the pattern of the infectious human population and the infectious vector populations. An increase in the number of susceptible human and vector populations in the system, there are higher chances of individuals and animals to get infected with Listeriosis disease since contact rate would increases. The model assumes the concept of mass action.

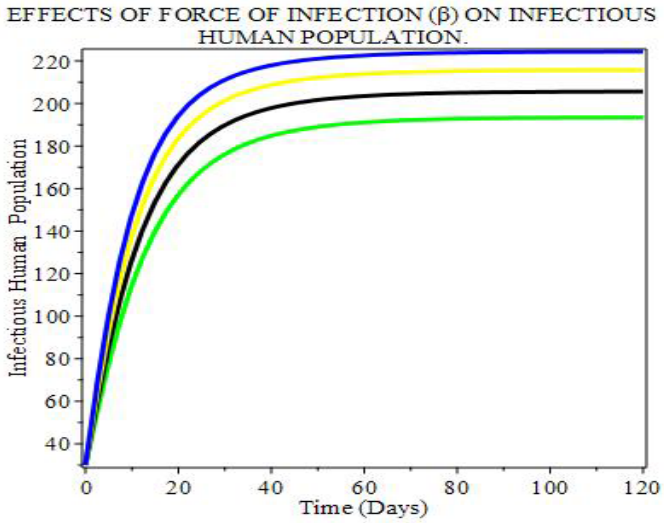

Figure 2: Simulations of Listeriosis model showing the effects of force of infection on infectious human populations.

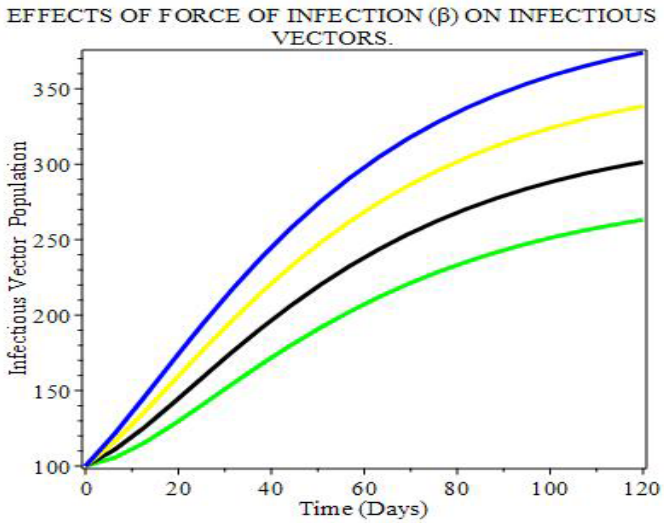

Figure 3: Simulations of Listeriosis model showing the effects of force of infection on infectious vector populations.

5.4. Simulations of Listeriosis model showing Recovered human and Recovered vector populations

The simulations in Figure 5 showed recovered human population decreases and there was a constant decreasing at some point in time. Since there are no controls in the model, this can be as a result to the number of individuals recovering from the disease been equal to the number of people getting infected from the Listeriosis disease. In the

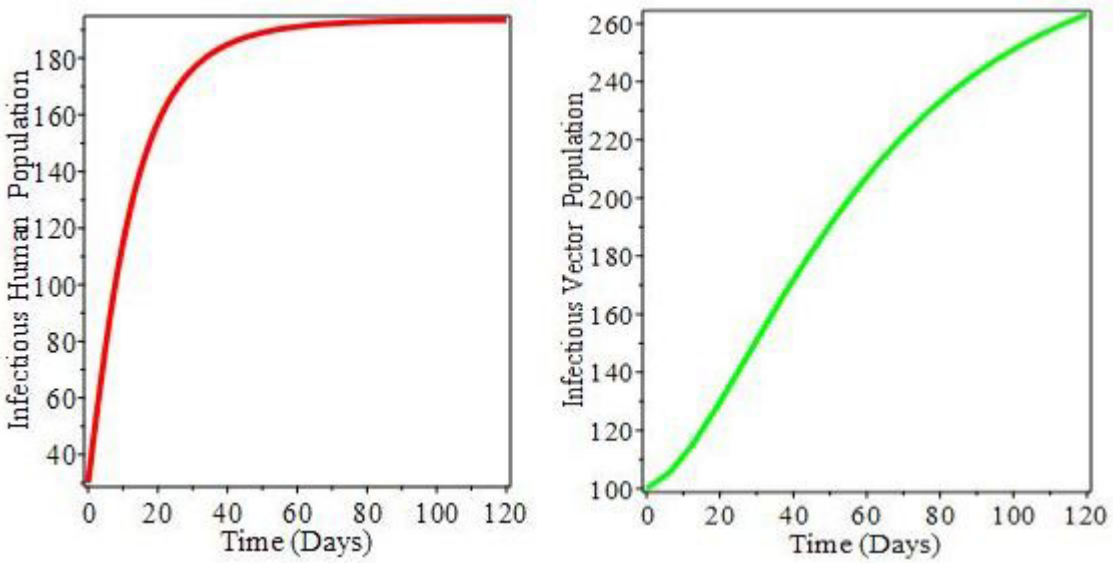

Figure 4: Simulations of the Listeriosis model showing the Infective vector population and Infective human population.

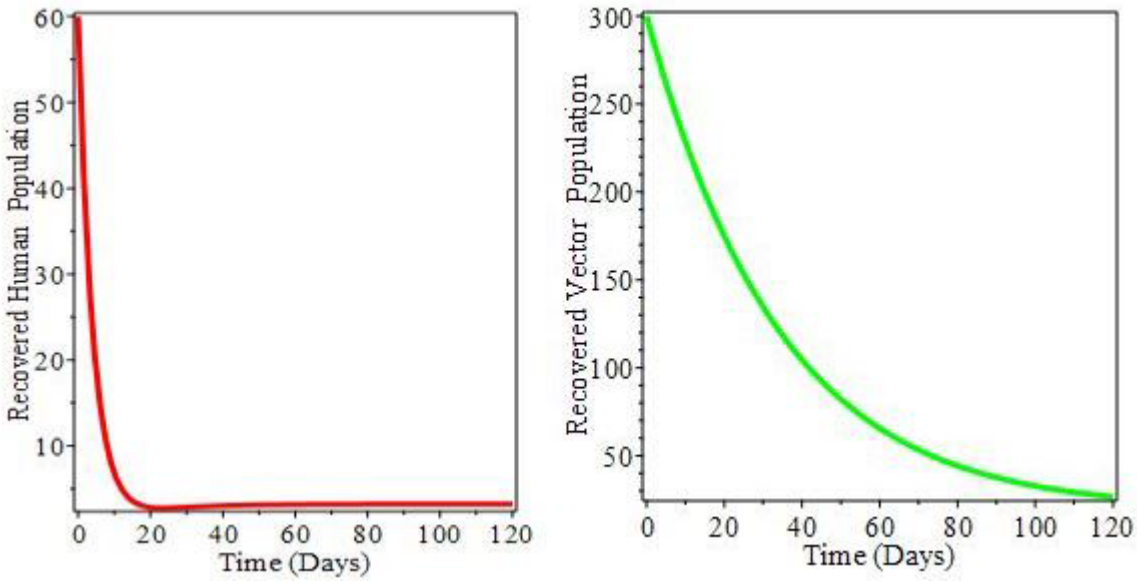

Figure 5: Simulations of the Listeriosis model indicating the Recovered vector population and the Recovered human population.

vector population, the number of animals recovering from the disease decreases with time and a sharp increase in the number of recovery.

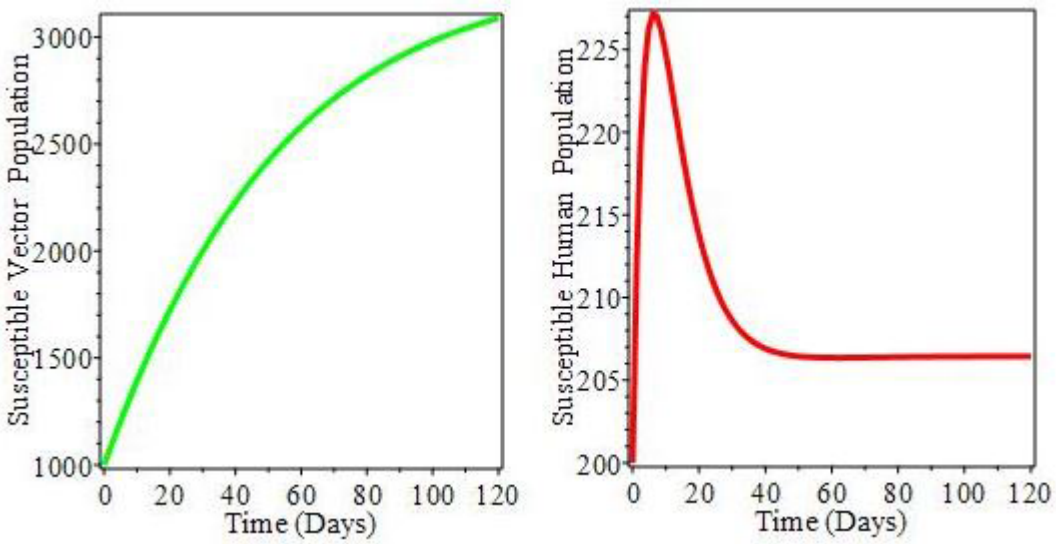

Figure 6: Simulations of Listeriosis model showing susceptible human and vector populations.

5.5. Simulations of Listeriosis model showing susceptible human and vector populations

The number of susceptible human and susceptible vector populations increases as shown in figure 6 could be attributed to the increase in the number of birth rate and the recovery rate of human and vector populations. There are more vector and humans coming into the population and this increases the number of susceptible vector and human populations.

6. Conclusion

In this paper, we developed a compartmental model for the transmission dynamics of Listeriosis infections by addition of vaccination of susceptible vector (Livestock) compartment with waning immunity was analysed. We investigated the impact of the vaccination compartment on the transmission dynamics of the disease. The basic reproductive number was analysed and we established that our model has a globally stable infection-free equilibrium when the basic reproductive number is less than one. The model indicated an existence of multiple endemic equilibrium. Epidemically, the implication is that the disease can best be curbed down whenever the basic reproductive number is always less than unity.

Sensitivity analysis of the basic reproductive number was performed to each of the parameters to determine which parameter is more sensitive to model than the other. Our analysis showed that, decreasing the vector (livestock) recovery rate, human death rate and vector (livestock) death rate would increase the basic reproductive number whilst increasing the vector (livestock) recovery rate, human death rate and vector (livestock)

death rate would decrease the basic reproductive number. However, decreasing human recruitment rate, vector (livestock) recruitment rate, vector (livestock) transmission rate and human transmission rate would cause a decrease in the basic reproduction number. Increasing human recruitment rate, vector (livestock) recruitment rate, vector (livestock) transmission rate and human transmission rate would cause an increase in the basic reproduction number.

References

[1] F. Allerberger and M. Wagner, Listeriosis: a resurgent foodborne infection, *Clinical Microbiology and Infection*, 16(1):16–23, 2010.

[2] Monica K. Borucki, James Reynolds, Clive C. Gay, Katherine L. McElwain, So Hyun Kim, Donald P. Knowles, and Jinxin Hu, Dairy farm reservoir of listeria monocytogenes sporadic and epidemic strains, *Journal of Food Protection®*, 67(11):2496–2499, 2004.

[3] Catherine W. Donnelly, Listeria monocytogenes: a continuing challenge, *Nutrition Reviews*, 59(6):183–194, 2001.

[4] Khalid Hattaf, Abid Ali Lashari, Younes Louartassi, and Noura Yousfi, A delayed sir epidemic model with general incidence rate, *Electronic Journal of Qualitative Theory of Differential Equations*, 3:1–9, 2013.

[5] H. Hof, Listeria monocytogenes: a causative agent of gastroenteritis? *European Journal of Clinical Microbiology and Infectious Diseases*, 20(6):369–373, 2001.

[6] T. Jemmi and R. Stephan, Listeria monocytogenes: food-borne pathogen and hygiene indicator, *Rev Sci Tech*, 25(2):571–80, 2006.

[7] J.P. LaSalle, The stability of dynamical systems, regional conference series in appl. *Math., SIAM, Philadelphia*, 1976.

[8] Abid Ali Lashari, Optimal control of an sir epidemic model with a saturated treatment, *Appl. Math*, 10(1):185–191, 2016.

[9] Alexandra Lianou and John N Sofos, A review of the incidence and transmission of listeria monocytogenes in ready-to-eat products in retail and food service environments, *Journal of Food Protection®*, 70(9):2172–2198, 2007.

[10] Christine L. Little, Sara M. Pires, Iain A. Gillespie, Kathie Grant, and Gordon L. Nichols, Attribution of human listeria monocytogenes infections in england and wales to ready-to-eat food sources placed on the market: adaptation of the hald salmonella source attribution model, *Foodborne pathogens and disease*, 7(7):749–756, 2010.

[11] Everitt George Dunne Murray, Robert Alexander Webb, and Meredith Blake Robson Swann, A disease of rabbits characterised by a large mononuclear leucocytosis, caused by a hitherto undescribed bacillus bacterium monocytogenes (n. sp.), *The Journal of Pathology and Bacteriology*, 29(4):407–439, 1926.

[12] Kazeem Oare Okosun, M. Mukamuri, and Daniel Oluwole Makinde, Global stability analysis and control of leptospirosis, *Open Mathematics*, 14(1):567–585, 2016.

[13] Victoria Rebagliati, Romina Philippi, Maria Rossi, Alcides Troncoso, et al., Prevention of foodborne listeriosis, *Indian Journal of Pathology and Microbiology*, 52(2):145, 2009.

[14] M.L. Rossi, Analia Paiva, Mariela Tornese, Sabrina Chianelli, and Alcides Troncoso, [listeria monocytogenes outbreaks: a review of the routes that favor bacterial presence]. *Revista chilena de infectologia: organo oficial de la Sociedad Chilena de Infectologia*, 25(5):328–335, 2008.

[15] Shigui Ruan and Wendi Wang, Dynamical behavior of an epidemic model with a nonlinear incidence rate, *Journal of Differential Equations*, 188(1):135–163, 2003.

[16] Mohammad A. Safi and Salisu M. Garba, Global stability analysis of seir model with holling type ii incidence function, *Computational and mathematical methods in medicine*, 2012.

[17] Walter F. Schlech and David Acheson, Foodborne listeriosis, *Clinical Infectious Diseases*, 31(3):770–775, 2000.

[18] Anne Schuchat, Bala Swaminathan, and CLAIRE V. Broome, Epidemiology of human listeriosis, *Clinical microbiology reviews*, 4(2):169–183, 1991.

[19] Bala Swaminathan and Peter Gerner-Smidt, The epidemiology of human listeriosis, *Microbes and Infection*, 9(10):1236–1243, 2007.

[20] Pranita D. Tamma, Sara E. Cosgrove, and Lisa L. Maragakis, Combination therapy for treatment of infections with gram-negative bacteria, *Clinical microbiology reviews*, 25(3):450–470, 2012.

[21] Pauline Van den Driessche and James Watmough, Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, *Mathematical biosciences*, 180(1):29–48, 2002.

[22] Hannah M. Wexler, Bacteroides: the good, the bad, and the nitty-gritty, *Clinical microbiology reviews*, 20(4):593–621, 2007.