A Mathematical Model for Listeriosis Dynamics

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Abstract

Listeriosis is a food born disease caused by the bacteria known as Listeria monocytogens. Human and animals can be infected by consuming contaminated food products though a transmission can also occur through contact with infected animals or people although to a less extent. In this study, a mathematical model for Listeriosis dynamics was developed. The steady states and the stability of the model system were determined and analysed. The results show that the disease free equilibrium is asymptotically stable if the bacteria growth rate is less than its removal rate and also the growth rate of food contamination is less than its removal rate. It was further observed that we can still have Listeriosis driven by the contaminated food even though the Listeria bacteria population in the environment is made to go to extinct. This underscores the need to control Listeriosis by removing contaminated food, which was the policy adopted by the South African government during the recent Listeriosis outbreak.

Keywords: Listeria monocytogenes, Listeriosis, mathematical model, simulation, stability.

Declaration

I, the undersigned, hereby declare that the work contained in this research project is my original work, and that any work done by others or by myself previously has been acknowledged and referenced accordingly.

Verediana Machele Mbalilo, 23 May 2019

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1. Introduction

1.1 Background and literature review

Listeria monocytogenes are pathogens which affect raw and processed food. The consumption of such product causes infections among humans and animals. This pathogen can be characterized as a Gram-positive bacteria (Portnoy et al., 1992). Listeria strives very well at low-temperature areas and unclean environments. Other favorable conditions for their survival include high salt concentration and acidic conditions. Specifically, they are mostly found in soil, unclean water bodies (lakes, rivers, etc.), vegetation, refrigerators, feces of some animals and foods such as smoked fish, cold meats and soft cheeses. Animals including cattle and poultry can also carry this bacteria. Due to their presence in soil and vegetation, they easily affect raw foods. Moreover, processed food can be contaminated by the presence of infected raw food and the use of uncleaned processing materials (Buchanan et al., 2017). With this, humans and animals can be infected by consuming such contaminated food products though a transmission can also occur through contact with infected animals or people although to a less extent. Infected pregnant women and female animals can also transfer these infections to their unborn babies (WHO, 2019), a process often referred to as vertical transmission.

The infection caused by Listeria monocytogenes is called Listeriosis. The group of people who are at high risk to acquire this disease are elderly, pregnant women and infants as well as people with weak immune systems such as cancer, diabetes, kidney disease and HIV AIDS patients (Cartwright et al., 2013). Fever, flu-like symptoms, vomiting, nausea and diarrhea are the main symptoms of this disease. Also when pregnant women are infected with this disease can develop muscle pain, headaches and backaches which can result into miscarriages. As most bacteria infections, Listeriosis can be treated and prevented by storing food safely, avoiding storing products in the fridge beyond the use by date, cooking the meat and poultry properly and keeping raw food from touching other foods and utensils (Janakiraman, 2008). Although this disease is relatively rare, it is often severe with high hospitalization and mortality rates. For instance there were 10 cases of this disease reported from a small area of Switzerland which was due to the distributed local soft cheese. The same problem was experienced by the Czech Republic in 2006 with 78 patients of whom 13 died. Also, in 2006 and 2007, Germany reported having an outbreak of 16 cases caused by presliced ready-to-eat meat (Allerberger and Wagner, 2010). Furthermore, the National Institute of Communicable Disease (NICD) reported that by 28 February 2018 there had been 943 laboratory-confirmed cases of Listeriosis with 176 deaths from the disease in South Africa (Manganye et al., 2018).

Analyzing mathematical models related to diseases is a well-known approach to study the spread of diseases over a period of time. In view of that, there have been several proposed mathematical models and other statistical methods to study Listeria and the spread of Listeriosis. The work by Buchanan et al. (1989) shows that the growth of Listeria monocytogens depends on the interaction of five variables which are pH, temperature, sodium nitrate, atmosphere, and sodium chloride. According to their data, they found out that sodium nitrate can have high significant bacteriostatic activity against Listeria monocytogens and hence can be used to provide cured meats with a degree of protection against this bacteria and this is archived when there is a combination of high salt concentration, acidic pH, packged vacuum and adequate refrigeration. Also, Ivanek et al. (2004) conducted a study on how ready-to-eat food (smoked fish) is contaminated with Listeria monocytogens in relation to some associated factors during food processing. In their study, they developed a model where they considered food contact surface and employees groves as the key factors for food to be contaminated. They found out that the best way to prevent food contamination during food processing is to make sure that the raw food

and processing materials coming in are free from contamination. According to the inference from the outbreak of the disease reported in Japan, Europe and North America, the foods which are at high risk for susceptible humans are ready-to-eat meats and soft cheese (Swaminathan and Gerner-Smidt, 2007). Furthermore, Luber et al. (2011) pointed out that for successful employment of the food safety method against Listeriosis there is a need for food workers to be educated and trained on how to prevent (protect) the ready-to-eat foods from Listeria monocytogens contamination and also to advice the consumer to be aware and responsible for their food safety. Rosshaug et al. (2012) conducted a study with the aim of developing a predictive model that simulates the growth of Listeria monocytogen in soft blue-white cheese. In their study, they come up with a tertiary predictive model of the Listeria growth as a function of lactic acid, temperature, pH and sodium chloride. According to their analysis, they found out that the growth rate of Listeria monocytogenes is very high when present in cheese. Mateus et al. (2013) pointed out that pregnant women affected with Listeriosis are at a high risk of miscarriages and fetal death or neonatal morbidity in the form of meningitis and septicemia. Also in their study, they found out that improving education about Listeriosis transmission, control and prevention for pregnant and individuals with week immune systems will help in minimizing the mortality rate of this disease. Moreover, Osman et al. (2018) developed a mathematical model to study the effect of vaccination of animals to the spread of Listeriosis among humans and animals (as vectors of Listeria). In their analysis, they found out that the secondary infections can increase due to the decrease of human, animal death rate and animal recovery rate.

1.2 Motivation

A serious outbreak of Listeriosis was recorded in South Africa with 978 laboratory cases from the beginning of 2017 to the first quarter of 2018. It was confirmed that 183 people (thus 27%) out of 674 patients died of the disease. This case fatality ratio is high comparable to other recorded Listeriosis outbreaks globally (WHO, 2018). The main cause of the disease resulted from the consumption of processed meat products. The response by the government was to recall all meat products from companies whose factories were found to be contaminated.

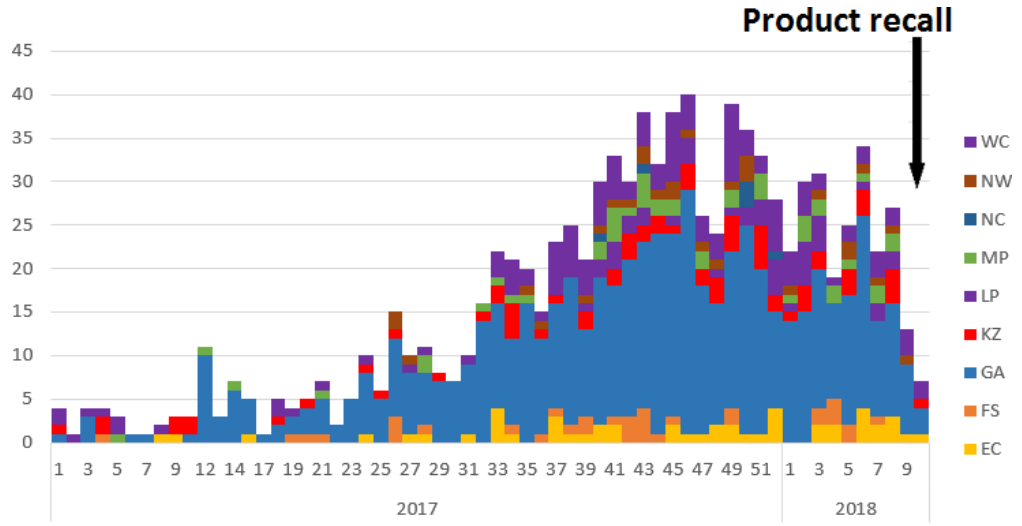

Figure 1.1: Number of Laboratory-Confirmed Cases of Listeriosis by Week of Sample Collection and Province, South Africa, 01 January 2017 to 12 March 2018 (n=978),(WHO, 2018).

It is against this background that we formulate a mathematical model for Listeriosis that considers the interaction of bacteria, humans and contaminated food.

1.3 Aims and objectives

The aims and objectives of this study includes:

(i) To develop a mathematical model for the contribution of factory products, the environment and humans in the spread of Listeriosis.

(ii) To determine the steady states and stability of the equilibrium points of the developed model.

(iii) Carry out the numerical simulations.

(iv) Create a foundation for further work on the model by linking it to data.

2. A Mathematical Model for Listeriosis

2.1 Model formulation and assumptions

In this section, we shall formulate a mathematical model that takes into account the contribution of humans, the bacteria in the environment and factory products to the spread of Listeriosis. For the model to be biologically meaningful, we also introduce some assumptions.

We consider three main groups, namely, the human population, the Listeria bacteria, and factory products and create state variables for each groups. We will have six compartments based on their interactions depicted in Figure 2.1.

In particular, the human population denoted by N, comprises of three compartments namely: the susceptible humans (S), infected humans (I) and the recovered humans (R), so that

$$N = S + I + R.$$

The susceptible humans refer to humans who at risk of being infected with Listeriosis. The infected humans are those that show symptoms and have confirmed Listeria bacteria in them. The recovered are those they would have had the disease and are treated or recover on their own. Since there is no vaccination against this disease, the recovered humans can become susceptible again. The bacteria in the environment at any time t is given by $B(t)$. The food products are divided into two classes, the non-contaminated food, $F_n(t)$ and the contaminated food $F_c(t)$.

Let μN denote the number of humans that are recruited into the susceptible compartment. The susceptible human can move into an infected group either by acquiring Listeriosis through eating contaminated products or through contact with material from the environment at rate $\lambda_h(t)$. This is given by

$$\lambda_h(t) = \frac{\beta_3 B}{\kappa + B} + \beta_4 F_c,$$

where β_3 is the infection rate of humans due to the Listeria bacteria from the environment and κ is called the half saturation constant. The half saturation constant is defined as the concentration of Listeria bacteria in the environment depicting the half saturation rate, this means that when $\kappa = B$ we obtain half the infection rate of humans due to Listeria from the environment, thus $\frac{\beta_3}{2}$. The parameter β_4 is the infection rate of susceptible humans when they take contaminated food products. We assume that there is no human to human transmission, and that all humans die naturally of a rate μ.

Once infected, humans can either die naturally, die of the disease at a rate δ_1 or recover at a rate γ and join the recovered class R.

Now considering the factory dynamics, we assume that the amount of food products F comprises of non-contaminated (F_n) and contaminated (F_c) so that

$$F = F_n + F_c.$$

By contact with contaminated surfaces and contaminated products, we assume that non-contaminated factory products can become contaminated with Listeria bacteria. We let $\lambda_f(t)$ be the rate at which non-contaminated factory products become contaminated and it is given by

$$\lambda_f(t) = \frac{\beta_1 B}{\kappa + B} + \beta_2 F_c$$

where β_1 is the rate at which non-contaminated food is contaminated by bacteria and β_2 is the rate at which contaminated food product contribute to the contamination of non-contaminated food products through the ability of contaminated food to contaminate surfaces and workers who intern contaminate non contaminated food products. We assume that the production rate δ_2 is equal to the disposal (product consumed or removal) rate from the factory.

The Listeria bacteria in the environment grows at a rate of r_b and the growth rate is assumed to be logistical. The bacteria die at a rate of μ_b. The model structure is represented schematically in Figure 2.1 where the dotted arrow shows the contributing factors to the flow of the human and food products.

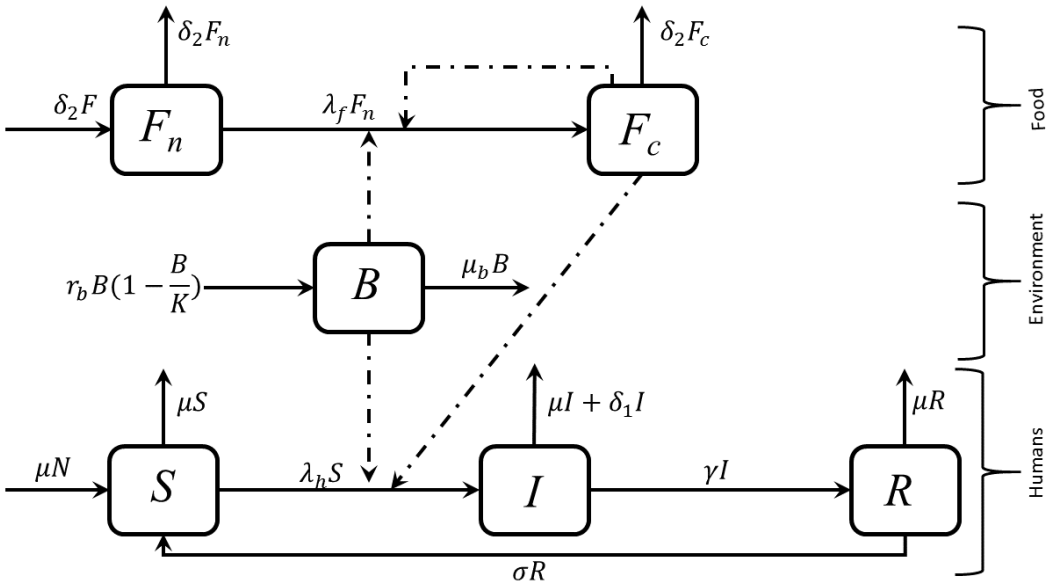

Figure 2.1

A summary descriptions of the parameters used in the model are shown in Table 2.1.

Parameter	Description
μ	Human natural death rate
γ	Human recovery rate
σ	Rate at which recovered humans return to susceptible status due to loss of immunity
r_b	Rate of Listeria bacteria to increase in the environment
μ_b	Natural death rate of bacteria
δ_1	Rate of infected human to die due to the Listeriosis
δ_2	Food removal (consuming or disposing) rate
β_1	Rate at which non-contaminated food is contaminated by bacteria
β_2	Rate of contamination of food products
β_3	Infection rate of humans by bacteria from the environment
β_4	Infection rate of susceptible humans when they take contaminated food products
κ	Half saturation constant
K	Carrying capacity of Listeria bacteria

Table 2.1: Description of parameters used in the model.

From the model shown in Figure 2.1, we obtain the following ordinary differential equations.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \mu N + \sigma R - \lambda_h S - \mu S,\\
\frac{dI}{dt} &= \lambda_h S - (\mu + \gamma + \delta_1) I,\\
\frac{dR}{dt} &= \gamma I - (\mu + \sigma) R,\\
\frac{dF_n}{dt} &= \delta_2 F - \lambda_f F_n - \delta_2 F_n,\\
\frac{dF_c}{dt} &= \lambda_f F_n - \delta_2 F_c,\\
\frac{dB}{dt} &= r_b B \left(1 - \frac{B}{K}\right) - \mu_b B.
\end{aligned}\right\} \tag{2.1.1}$$

2.2 Non-dimensionalization and model reduction

The aim of this section is to reduce the number of parameters in our system of differential equations (2.1.1). In particular, we choose new variables with the idea of eliminating the dimension of old variables without affecting the biological meaning of our system (Banasiak and Lachowicz, 2014). To do this, we introduce the following new variables;

$$f_n = \frac{F_n}{F}, \qquad f_c = \frac{F_c}{F}, \qquad b = \frac{B}{K}, \qquad s = \frac{S}{N}, \qquad i = \frac{I}{N}, \qquad r = \frac{R}{N}, \qquad \hat{\kappa} = \frac{\kappa}{K}, \qquad \hat{\beta}_2 = \beta_2 F$$

and $\hat{\beta}_4 = \beta_4 F$.

Recall that

$$\begin{aligned} N &= S + I + R, \\ 1 &= \frac{S}{N} + \frac{I}{N} + \frac{R}{N}, \\ 1 &= s + i + r, \end{aligned}$$

and

$$\begin{aligned} F &= F_n + F_c, \\ 1 &= \frac{F_n}{F} + \frac{F_c}{F}, \\ 1 &= f_n + f_c. \end{aligned}$$

By substituting these equations into our system (2.1.1) and simplifying, the system reduces to the following differential equations

$$\left.\begin{aligned} \frac{ds}{dt} &= \mu + \sigma(1 - s - i) - \mu s - \hat{\lambda}_h s, \\ \frac{di}{dt} &= \hat{\lambda}_h s - (\mu + \gamma + \delta_1) i, \\ \frac{df_c}{dt} &= \hat{\lambda}_f (1 - f_c) - \delta_2 f_c, \\ \frac{db}{dt} &= r_b b(1 - b) - \mu_b b, \end{aligned}\right\} \tag{2.2.1}$$

where $\hat{\lambda}_f = \frac{\beta_1 b}{\hat{\kappa}+b} + \hat{\beta}_2 f_c$, $\hat{\lambda}_h = \frac{\beta_3 b}{\hat{\kappa}+b} + \hat{\beta}_4 f_c$ with the initial conditions $s(0) \geq 0, i(0) \geq 0, f_c(0) \geq 0$ and $b(0) \geq 0$.

2.3 Basic properties of the model

This section explains the basic properties of our new system of differential equations (2.2.1). One important aspect is to show that, its solutions are non-negative and bounded for all time in a biologically feasible region.

2.3.1 Positivity of the solutions. The positivity of solutions to our new model is captured in the following lemma.

2.3.2 Lemma. Suppose $s(0) \geq 0, i(0) \geq 0, f_c(0) \geq 0$ and $b(0) \geq 0$ then the solutions $s(t), i(t), f_c(t)$ and $b(t)$ of system (2.2.1) are non-negative for all time $t \geq 0$.

Proof. Let $(s(t), i(t), f_c(t), b(t))$ be a solution of the system (2.2.1) with a given initial condition. In order to prove Lemma 2.3.2, we consider the equations of our system, solve and integrate them over the interval $[0, t]$.

Now from the first equation of the system (2.2.1), we have

$$\begin{aligned}\frac{ds}{dt} &= \mu + \sigma(1 - s - i) - \mu s - \hat{\lambda}_h s, \\ &= (\mu + \sigma - \sigma i) - (\sigma + \mu + \hat{\lambda}_h)s.\end{aligned}$$

This implies that

$$\frac{ds}{dt} \geq -(\sigma + \mu + \hat{\lambda}_h)s.$$

By integrating on both sides and applying the initial conditions, we have

$$s(t) \geq s(0)e^{-\int_0^t \hat{\lambda}_h(\tau)d\tau - (\sigma+\mu)t},$$

at any value of $t \geq 0$. Since $s(0) \geq 0$ then $s(t) \geq 0$.

From the second equation of the system (2.2.1) we have

$$\frac{di}{dt} = \hat{\lambda}_h s - (\mu + \gamma + \delta_2)i,$$

which implies that

$$\frac{di}{dt} \geq -(\mu + \gamma + \delta_2)i.$$

By integrating using separation of variables and then applying the initial conditions, we have

$$i(t) \geq i(0)e^{-(\mu+\gamma+\delta_1)t},$$

at any value of $t \geq 0$. Since $i(0) \geq 0$ then $i(t) \geq 0$.

From the third equation of the system (2.2.1) we have

$$\begin{aligned}\frac{df_c}{dt} &= \hat{\lambda}_f(1 - f_c) - \delta_2 f_c, \\ &= \hat{\lambda}_f - (\hat{\lambda}_f + \delta_2)f_c,\end{aligned}$$

which implies that

$$\frac{df_c}{dt} \geq -(\hat{\lambda}_f + \delta_2) f_c.$$

Integrating both sides and then applying the initial condition, we have that

$$f_c(t) \geq f_c(0) e^{-\int_0^t \hat{\lambda}_f(\tau) d\tau - \delta_2 t},$$

at any value of $t \geq 0$. Since $f_c(0) \geq 0$ then $f_c(t) \geq 0$.

Same applies to the last equation of the system where we have

$$\begin{aligned} \frac{db}{dt} &= r_b b(1-b) - \mu_b b, \\ &= -r_b b^2 - (\mu_b - r_b) b. \end{aligned}$$

This implies that

$$\frac{db}{dt} \geq -(\mu_b - r_b) b.$$

Integrating using separation of variables, we have

$$b(t) \geq b(0) e^{-(\mu_b - r_b)t},$$

at any value of $t \geq 0$. Since $b(0) \geq 0$ then $b(t) \geq 0$.

Therefore $(s(t), i(t), f_c(t), b(t))$ will never be negative for some non-negative initial conditions. □

2.3.3 Feasible region. Let us consider the biologically feasible region given by $\Omega = \{(s(t), i(t), f_c(t), b(t)) \in \mathbb{R}^4_+ : s + i \leq 1, f_c \leq 1, b \leq 1\}$.

2.3.4 Lemma. The solution of the system (2.2.1) with the non-negative initial condition are bounded for all $t \geq 0$ in the biologically feasible region, Ω.

Proof. Let $n = s + i$, such that $n \leq 1$.

We have that

$$\begin{aligned} \frac{dn}{dt} &= \frac{ds}{dt} + \frac{di}{dt}, \\ &= \mu + \sigma - \sigma(s+i) - \mu(s+i) - (\gamma + \delta_1) i, \\ &= \mu + \sigma - (\mu + \sigma) n - (\gamma + \delta_1) i. \end{aligned}$$

In the absence of mortality due to Listeria infections,

$$\frac{dn}{dt} \leq (\mu + \sigma)(1 - n).$$

Upon solving the differential equation above, we have

$$n(t) \leq 1 + Ae^{-(\sigma+\mu)t},$$

where A is a constant.

By applying the initial condition, we have

$$n(0) = 1 + A,$$

which implies that

$$A = n(0) - 1.$$

So,

$$n(t) \leq 1 + (n(0) - 1)e^{-(\sigma+\mu)t}.$$

As $t \to \infty$, $n \to 1$.

Also if $n(0) \leq 1$ then $n(t) \to 1$ from below.

Given that

$$\begin{aligned} \frac{df_c}{dt} &= \hat{\lambda}_f(1 - f_c) - \delta_2 f_c, \\ &= \hat{\lambda}_f - \hat{\lambda}_f f_c - \delta_2 f_c, \end{aligned}$$

which implies that

$$\frac{df_c}{dt} \leq \hat{\lambda}_f(1 - f_c).$$

Upon solving the differential equation above, we have

$$f_c(t) \leq 1 + Ce^{-\int \hat{\lambda}_f dt},$$

where C is a constant.

Applying the initial conditions, we have

$$f_c(0) = 1 + C,$$

which implies that

$$C = f_c(0) - 1.$$

So,

$$f_c(t) \leq 1 + (f_c(0) - 1)e^{-\int \hat{\lambda}_f dt}.$$

As $t \to \infty$, $f_c \to 1$.

This implies that, $0 \leq f_c \leq 1$.

Also if $f_c(0) \leq 1$ then $f_c(t) \to 1$ from below.

For the case of bacteria population, we consider the last equation from our system (2.2.1).

Thus

$$\frac{db}{dt} = b(r_b - \mu_b) - r_b b^2,$$

which gives us

$$b^{-2}\frac{db}{dt} = b^{-1}(r_b - \mu_b) - r_b. \tag{2.3.1}$$

We let $v(t) = b^{-1}$ and we obtain,

$$\frac{dv}{dt} = -b^{-2}\frac{db}{dt}. \tag{2.3.2}$$

Now, by substituting (2.3.2) into (2.3.1) yields

$$\frac{dv}{dt} + (r_b - \mu_b)v = r_b.$$

Solving this equation gives us

$$v(t) = \frac{r_b}{r_b - \mu_b} + Be^{-(r_b-\mu_b)t}.$$

Therefore,

$$b^{-1} = \frac{r_b}{r_b - \mu_b} + Be^{-(r_b-\mu_b)t},$$

so

$$b(t) = \frac{1}{\frac{r_b}{r_b-\mu_b} + Be^{-(r_b-\mu_b)t}},$$

where B is a constant. Applying the initial conditions gives us

$$b(0) = \frac{1}{\frac{r_b}{r_b-\mu_b} + B},$$

$$B = \frac{1 - \frac{r_b}{r_b-\mu_b}b(0)}{b(0)}.$$

Therefore

$$b(t) = \frac{1}{\frac{r_b}{r_b-\mu_b} + \left(\frac{1-\frac{r_b}{r_b-\mu_b}b(0)}{b(0)}\right)e^{-(r_b-\mu_b)t}}.$$

As $t \to \infty$, $b \to \frac{r_b-\mu_b}{r_b} = 1 - \frac{\mu_b}{r_b} \leq 1$. This means that for the Listeria bacteria population to exist their death rate μ_b should be less than their growth rate r_b. This implies that, $0 \leq b \leq 1$.

Also if $b(0) \leq 1$ then $b(t) \to 1$ from below.

Hence (s, i, f_c, b) are all bounded in the region Ω. Therefore Ω is a biologically feasible. □

3. Model Analysis

3.1 Model steady states

We know that an equilibrium point (steady state) is the point at which the function or derivative is equal to zero. So to determine the equilibrium points of the system (2.2.1) we have to set the derivatives equal to zero and then solve the resulting algebraic equation. Thus

$$\left.\begin{aligned} 0 &= \mu + \sigma(1 - s^* - i^*) - \mu s^* - \hat{\lambda}_h s^*, \\ 0 &= \hat{\lambda}_h s^* - (\mu + \gamma + \delta_1) i^*, \\ 0 &= \hat{\lambda}_f (1 - f_c^*) - \delta_2 f_c^*, \\ 0 &= r_b b^* (1 - b^*) - \mu_b b^*. \end{aligned}\right\} \tag{3.1.1}$$

From the last equation of the system (3.1.1), we have

$$\begin{aligned} r_b b^*(1 - b^*) - \mu_b b^* &= 0, \\ b^* \left(r_b(1 - b^*) - \mu_b\right) &= 0. \end{aligned}$$

This gives us

$$b_1^* = 0 \quad \text{and} \quad b_2^* = 1 - \frac{\mu_b}{r_b}.$$

Note that, b_2^* exists if and only if $\mu_b < r_b$.

Now from these two values of b^*, we have two cases to get the steady states.

Case 1: When $b^* = b_1^* = 0$ we have

$$\hat{\lambda}_f = \hat{\beta}_2 f_c^* \quad \text{and} \quad \hat{\lambda}_h = \hat{\beta}_4 f_c^*.$$

Recall that

$$\hat{\lambda}_f = \frac{\beta_1 b^*}{\hat{\kappa} + b^*} + \hat{\beta}_2 f_c^* \quad \text{and} \quad \hat{\lambda}_h = \frac{\beta_3 b^*}{\hat{\kappa} + b^*} + \hat{\beta}_4 f_c^*.$$

1 Now from the third equation of the system (3.1.1) we have

$$\begin{aligned} \hat{\lambda}_f (1 - f_c^*) - \delta_2 f_c^* &= 0, \\ \hat{\beta}_2 f_c^* (1 - f_c^*) - \delta_2 f_c^* &= 0, \\ f_c^* \left(\hat{\beta}_2 (1 - f_c^*) - \delta_2\right) &= 0, \end{aligned}$$

and this yields

$$f_{c_1}^* = 0 \quad \text{and} \quad f_{c_2}^* = 1 - \frac{\delta_2}{\hat{\beta}_2}.$$

So now we have

$b_1^* = 0, \quad f_{c_1}^* = 0$ and $b_1^* = 0, \quad f_{c_2}^* = 1 - \frac{\delta_2}{\hat{\beta}_2}$.

But $f_{c_2}^*$ exist if and only if $\delta_2 < \hat{\beta}_2$.

Now lets consider $b^* = b_1^* = 0$ and $f_c^* = f_{c_1}^* = 0$.

From the second equation of the system (3.1.1) we have

$$\hat{\lambda}_h s^* - (\mu + \gamma + \delta_1) i^* = 0,$$
$$\hat{\beta}_4 f_{c_1}^* s^* - (\mu + \gamma + \delta_1) i^* = 0.$$

We can observe that

$$\text{if} \quad f_{c_1}^* = 0 \quad \text{then} \quad i^* = 0.$$

From the first equation of the system (3.1.1) we have

$$\mu + \sigma(1 - s^*) - \mu s^* = 0,$$
$$s^* = \frac{\mu + \sigma}{\mu + \sigma},$$
$$s^* = 1.$$

Therefore we have a steady state

$$E_0^* = (s^*, i^*, f_{c_1}^*, b_1^*) = (1, 0, 0, 0),$$

which is the disease free equilibrium point because all the infected compartments are zero.

Also lets consider $b^* = b_1^* = 0$ and $f_c^* = f_{c_2}^* = 1 - \frac{\delta_2}{\hat{\beta}_2}$.

From the second equation of the system (3.1.1) we can rewrite the equation as

$$\hat{\beta}_4 f_{c_2}^* s_1^* - (\mu + \gamma + \delta_1) i_1^* = 0,$$

which yields

$$i_1^* = \frac{\hat{\beta}_4 f_{c_2}^* s_1^*}{(\mu + \gamma + \delta_1)}. \tag{3.1.2}$$

By substituting equation (3.1.2) into the first equation of the system (3.1.1) we have

$$\mu + \sigma - \sigma s_1^* - \sigma \left(\frac{\hat{\beta}_4 f_{c_2}^*}{\mu + \gamma + \delta_1} \right) s_1^* - \mu s_1^* - \hat{\beta}_4 f_{c_2}^* s_1^* = 0.$$

This gives us

$$s_1^* = \frac{(\mu + \sigma)(\mu + \gamma + \delta_1)}{(\sigma + \mu)(\mu + \gamma + \delta_1) + \hat{\beta}_4 f_{c_2}^* (\sigma + \mu + \gamma + \delta_1)},$$
$$= \frac{1}{1 + Q_1^*},$$

where $Q_1^* = \frac{(\sigma + \mu + \gamma + \delta_1)\hat{\beta}_4 f_{c_2}^*}{(\mu + \sigma)(\mu + \gamma + \delta_1)}$.

Now by substituting s_1^* into (3.1.2) we have

$$\begin{aligned} i_1^* &= \frac{\hat{\beta}_4 f_{c_2}^*}{(\mu+\gamma+\delta_1)}\left(\frac{1}{1+Q_1^*}\right), \\ &= \frac{\hat{\beta}_4 f_{c_2}^*}{(\mu+\gamma+\delta_1)\,(1+Q_1^*)}. \end{aligned}$$

So in this case, we have the steady state,

$$E_1^* = (s_1^*, i_1^*, f_{c_2}^*, 0),$$

which we call the Listeria bacteria free equilibrium point because the bacteria compartment is zero.

Case 2: When $b^* = b_2^* = 1 - \frac{\mu_b}{r_b}$. Recall that

$$\hat{\lambda}_f = \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_2 f_{c_3}^* \quad \text{and} \quad \hat{\lambda}_h = \frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^*.$$

From the third equation of the system (3.1.1), we have

$$\begin{aligned} \hat{\lambda}_f - \hat{\lambda}_f f_{c_3}^* - \delta_2 f_{c_3}^* &= 0, \\ \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_2 f_{c_3}^* - \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} f_{c_3}^* - \hat{\beta}_2 f_{c_3}^{*2} - \delta_2 &= 0, \\ \hat{\beta}_2 f_{c_3}^{*2} + \left(\delta_2 + \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} - \hat{\beta}_2\right) f_{c_3}^* - \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} &= 0. \end{aligned}$$

This can be rewritten as

$$\Upsilon_2 f_{c_3}^{*2} + \Upsilon_1 f_{c_3}^* + \Upsilon_0 = 0, \tag{3.1.3}$$

where

$$\begin{aligned} &\Upsilon_2 = \hat{\beta}_2 > 0, \\ &\Upsilon_1 = \frac{\delta_2(\hat{\kappa}+b_2^*) + \beta_1 b_2^*}{\hat{\kappa}+b_2^*} - \hat{\beta}_2 \implies \Upsilon_1 \begin{cases} > 0 & \text{if } \frac{\delta_2(\hat{\kappa}+b_2^*)+\beta_1 b_2^*}{\hat{\kappa}+b_2^*} > \hat{\beta}_2 \\ < 0 & \text{if } \frac{\delta_2(\hat{\kappa}+b_2^*)+\beta_1 b_2^*}{\hat{\kappa}+b_2^*} < \hat{\beta}_2 \end{cases}, \\ &\Upsilon_0 = -\frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} < 0. \end{aligned}$$

Solving equation (3.1.3) we have,

$$f_{c_3}^* = \frac{-\Upsilon_1 \pm \sqrt{\Upsilon_1^2 - 4\,\Upsilon_2\,\Upsilon_0}}{2\Upsilon_2}.$$

Since $\Upsilon_0 < 0$ and $\Upsilon_2 > 0$ then $\Upsilon_2\Upsilon_0 < 0$, and this implies that $f_{c_3}^*$ has one positive solution, say $f_{c_3}^{*+}$ irrespective of the conditions imposed on Υ_1.

Now from the second equation of the system (3.1.1), we have

$$\left(\frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^{*+}\right) s_2^* - (\mu+\gamma+\delta_1)\, i_2^* = 0,$$

which yields

$$i_2^* = \frac{\beta_3 b_2^* + (\hat{\kappa} + b_2^*)\hat{\beta}_4 f_{c_3}^{*+}}{(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)} s_2^*. \tag{3.1.4}$$

Now from the first equation of the system (3.1.1), we have

$$\mu + \sigma(1 - s_2^* - i_2^*) - \mu s_2^* - \sigma \frac{\beta_3 b_2^* + (\hat{\kappa} + b_2^*)\hat{\beta}_4 f_{c_3}^{*+}}{(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)} s_2^* - \mu s_2^* - \frac{\beta_3 b_2^* + (\hat{\kappa} + b_2^*)\hat{\beta}_4 f_{c_3}^{*+}}{(\hat{\kappa} + b_2^*)} s_2^* = 0.$$

This gives us

$$\begin{aligned} s_2^* &= \frac{(\mu + \sigma)(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)}{(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)(\sigma + \mu) + (\hat{\kappa} + b_2^*)(\sigma + \mu + \gamma + \delta_1)\hat{\beta}_4 f_{c_3}^{*+} + \beta_3 b_2^*(\sigma + \mu + \delta_1)}, \\ &= \frac{1}{1 + Q_2^*}. \end{aligned}$$

where $Q_2^* = \frac{(\hat{\kappa}+b_2^*)(\sigma+\mu+\gamma+\delta_1)\hat{\beta}_4 f_{c_3}^{*+} + \beta_3 b_2^*(\sigma+\mu+\delta_1)}{(\mu+\sigma)(\hat{\kappa}+b_2^*)(\mu+\gamma+\delta_1)}$.

Now by substituting s_2^* into equation (3.1.4), we get

$$\begin{aligned} i_2^* &= \frac{\beta_3 b_2^* + (\hat{\kappa} + b_2^*)\hat{\beta}_4 f_{c_3}^{*+}}{(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)} \left(\frac{1}{1 + Q_2^*}\right), \\ &= \frac{\beta_3 b_2^* + (\hat{\kappa} + b_2^*)\hat{\beta}_4 f_{c_3}^{*+}}{(\hat{\kappa} + b_2^*)(\mu + \gamma + \delta_1)(1 + Q_2^*)}, \end{aligned}$$

Therefore at this case we have a unique endemic equilibrium point

$$E_2^* = (s_2^*, i_2^*, f_{c_3}^{*+}, b_2^*),$$

whose existence is subject to $\mu_b < r_b$.

3.2 Stability analysis

In this section we carry out the stability analysis of the steady states by using the Jaccobian matrix as follows. Consider the right hand side of the equations of the system (2.2.1). Thus, we have

$$\left.\begin{aligned} f_1 &= \mu + \sigma(1 - s - i) - \mu s - \hat{\lambda}_h s, \\ f_2 &= \hat{\lambda}_h s - (\mu + \gamma + \delta_1) i, \\ f_3 &= \hat{\lambda}_f (1 - f_c) - \delta_2 f_c, \\ f_4 &= r_b b(1 - b) - \mu_b b. \end{aligned}\right\} \tag{3.2.1}$$

The Jaccobian matrix of the system (2.2.1) at any point is given by

$$J(s,i,f_c,b)=\begin{bmatrix} -(\mu+\sigma)-\left(\frac{\beta_3 b}{\hat{\kappa}+b}+\hat{\beta}_4 f_c\right) & -\sigma & -\hat{\beta}_4 s & -\frac{\hat{\kappa}\beta_3 s}{(\hat{\kappa}+b)^2} \\ \frac{\beta_3 b}{\hat{\kappa}+b}+\hat{\beta}_4 f_c & -(\mu+\gamma+\delta_1) & \hat{\beta}_4 s & \frac{\hat{\kappa}\beta_3 s}{(\hat{\kappa}+b)^2} \\ 0 & 0 & -(\frac{\beta_1 b}{\hat{\kappa}+b}+2\hat{\beta}_2 f_c+\delta_2-\hat{\beta}_2) & -\frac{\hat{\kappa}\beta_1(f_c-1)}{(\hat{\kappa}+b)^2} \\ 0 & 0 & 0 & r_b-2r_b b-\mu_b \end{bmatrix}. \tag{3.2.2}$$

3.2.1 Local stability of the disease free equilibrium (DFE).

3.2.2 Theorem. *The disease free equilibrium E_0^* is locally asymptotically stable if $\hat{\beta}_2<\delta_2$ and $r_b<\mu_b$.*

Proof. In order to determine the local stability of the disease free equilibrium we use the Jaccobian matrix technique by finding the eigenvalues of the Jaccobian matrix evaluated at the disease free equilibrium.

Since at DFE $(s,i,f_c,b)=E_0^*=(1,0,0,0)$. So by substituting the point E_0^* into (3.2.2) we have

$$J(E_0^*)=\begin{pmatrix} -(\sigma+\mu) & -\sigma & -\hat{\beta}_4 & -\frac{\beta_3}{\hat{\kappa}} \\ 0 & -(\delta_1+\gamma+\mu) & \hat{\beta}_4 & \frac{\beta_3}{\hat{\kappa}} \\ 0 & 0 & \hat{\beta}_2-\delta_2 & \frac{\beta_1}{\hat{\kappa}} \\ 0 & 0 & 0 & r_b-\mu_b \end{pmatrix}, \tag{3.2.3}$$

since the Jaccobian matrix (3.2.3) is a upper triangular matrix, then the eigenvalues will be all the diagonal elements. Therefore the eigenvalues will be

$$\lambda_1=-(\sigma+\mu),$$

$$\lambda_2=-\left(\mu+\delta_1+\gamma\right),$$

$$\lambda_3=\hat{\beta}_2-\delta_2,$$

$$\lambda_4=r_b-\mu_b.$$

Also since all parameters are positive, it is clear that λ_1 and λ_2 are negative. However λ_3 and λ_4 are negative if and only if $\hat{\beta}_2<\delta_2$ and $r_b<\mu_b$ respectively.

Therefore the disease free equilibrium will be stable if $\hat{\beta}_2<\delta_2$ and $r_b<\mu_b$, which means that, the growth rate of food contamination must be less than its removal and the growth rate of bacteria must be less than its removal (natural death rate of bacteria). □

3.2.3 Local stability of bacteria free equilibrium.

3.2.4 Theorem. *The bacteria free equilibrium E_1^* is locally asymptotically stable if $r_b < \mu_b$ and $\frac{\hat{\beta}_2}{(2\hat{\beta}_2 f_{c_2}^* + \delta_2)} < 1$.*

Proof. To show the local stability of bacteria free equilibrium we also use Jaccobian matrix technique by finding the eigenvalues of the Jaccobian matrix evaluated at the bacteria free equilibrium.

Since the bacteria free equilibrium is found at E_1^* then by substituting the point E_1^* into (3.2.2) we have

$$J(E_1^*) = \begin{bmatrix} -(\sigma+\mu) - \hat{\beta}_4 f_{c_2}^* & -\sigma & -\hat{\beta}_4 s_2^* & -\frac{\hat{\kappa}\beta_3 s_2^*}{\hat{\kappa}^2} \\ \hat{\beta}_4 f_{c_2}^* & -(\mu+\gamma+\delta_1) & \hat{\beta}_4 s_2^* & \frac{\hat{\kappa}\beta_3 s_2^*}{\hat{\kappa}^2} \\ 0 & 0 & -(2\hat{\beta}_2 f_{c_2}^* + \delta_2 - \hat{\beta}_2) & -\frac{\hat{\kappa}\beta_1(f_{c_2}^* - 1)}{\hat{\kappa}^2} \\ 0 & 0 & 0 & r_b - \mu_b \end{bmatrix}. \tag{3.2.4}$$

Since matrix (3.2.4) is a block matrix, it can be rewritten as

$$J(E_1^*) = \begin{bmatrix} H & M \\ \mathbf{0} & G \end{bmatrix}, \tag{3.2.5}$$

also as matrix (3.2.5) is an upper triangular matrix, so its eigenvalues are equal to the eigenvalues of all diagonal matrices H and G where

$$H = \begin{bmatrix} -(\sigma+\mu) - \hat{\beta}_4 f_{c_2}^* & -\sigma \\ \hat{\beta}_4 f_{c_2}^* & -(\mu+\gamma+\delta_1) \end{bmatrix}, \quad M = \begin{bmatrix} -\hat{\beta}_4 s_2^* & -\frac{\hat{\kappa}\beta_3 s_2^*}{\hat{\kappa}^2} \\ \hat{\beta}_4 s_2^* & \frac{\hat{\kappa}\beta_3 s_2^*}{\hat{\kappa}^2} \end{bmatrix}, \quad \mathbf{0} = \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix}$$

$$\text{and} \quad G = \begin{bmatrix} -(2\hat{\beta}_2 f_{c_2}^* + \delta_2 - \hat{\beta}_2) & -\frac{\hat{\kappa}\beta_1(f_{c_2}^* - 1)}{\hat{\kappa}^2} \\ 0 & r_b - \mu_b \end{bmatrix}.$$

The matrix G is an upper triangular matrix then its eigenvalues are all diagonal elements. Thus

$$\lambda_1 = r_b - \mu_b \implies \lambda_1 < 0 \quad \text{if} \quad r_b < \mu_b,$$

$$\text{and} \quad \lambda_2 = -(2\hat{\beta}_2 f_{c_2}^* + \delta_2 - \hat{\beta}_2) = \left(2\hat{\beta}_2 f_{c_2}^* + \delta_2\right)\left[\frac{\hat{\beta}_2}{\left(2\hat{\beta}_2 f_{c_2}^* + \delta_2\right)} - 1\right] \implies \lambda_2 < 0 \quad \text{if} \quad \frac{\hat{\beta}_2}{\left(2\hat{\beta}_2 f_{c_2}^* + \delta_2\right)} < 1.$$

For the eigenvalues of matrix H, we solve from

$$\det(H - \lambda I) = 0, \quad \text{where} \quad I \quad \text{is an identity matrix.}$$

So upon calculating and simplifying, we get the characteristic equation

$$\lambda^2 + \rho_1 \lambda + \rho_0 = 0, \tag{3.2.6}$$

where

$$\rho_1 = (\mu+\gamma+\delta_1)+(\mu+\sigma)+\hat{\beta}_4 f_{c_2}^* \geq 0,$$
$$\rho_0 = (\mu+\sigma)(\mu+\gamma+\delta_1)+(\mu+\sigma+\gamma+\delta_1)\hat{\beta}_4 f_{c_2}^* \geq 0.$$

So

$$\lambda = \frac{-\rho_1 \pm \sqrt{\rho_1^2 - 4\rho_0}}{2},$$

gives us

$$\lambda_3 = \frac{-\rho_1 + \sqrt{\rho_1^2 - 4\rho_0}}{2} \quad \text{and} \quad \lambda_4 = \frac{-\rho_1 - \sqrt{\rho_1^2 - 4\rho_0}}{2}.$$

So, equation (3.2.6) has roots with negative real parts by the Routh–Hurwitz stability criterion.

Hence, the eigenvalues of matrix (3.2.4) are $\lambda_1, \lambda_2, \lambda_3$ and λ_4.

Therefore the bacteria free equilibrium will be locally asymptotically stable if and only if $\mu_b > r_b$ and $\frac{\hat{\beta}_2}{(2\hat{\beta}_2 f_{c_2}^* + \delta_2)} < 1$. □

3.2.5 Local stability of endemic equilibrium .

3.2.6 Theorem. *The endemic equilibrium E_2^* is locally asymptotically stable if $r_b < (2r_b b_2^* + \mu_b)$ and $\frac{\hat{\beta}_2}{J^*} < 1$, where*

$$J^* = \frac{\beta_1 b_2^*}{\hat{\kappa} + b_2^*} + 2\hat{\beta}_2 f_{c_3}^{*+} + \delta_2.$$

Proof. To show the local stability of endemic equilibrium we also use Jaccobian matrix technique by finding the eigenvalues of the Jaccobian matrix evaluated at the endemic equilibrium.

Since the endemic equilibrium is found at E_2^* then by substituting the point E_2^* into (3.2.2) we have

$$J(E_2^*) = \begin{bmatrix} -(\sigma+\mu) - \left(\frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^{*+}\right) & -\sigma & -\hat{\beta}_4 s_2^* & -\frac{\hat{\kappa}\beta_3 s_2^*}{(\hat{\kappa}+b_2^*)^2} \\ \frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^{*+} & -(\mu+\gamma+\delta_1) & \hat{\beta}_4 s_2^* & \frac{\hat{\kappa}\beta_3 s_2^*}{(\hat{\kappa}+b_2^*)^2} \\ 0 & 0 & -(\frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} + 2\hat{\beta}_2 f_{c_3}^{*+} + \delta_2 - \hat{\beta}_2) & -\frac{\hat{\kappa}\beta_1 (f_{c_3}^{*+}-1)}{(\hat{\kappa}+b_2^*)^2} \\ 0 & 0 & 0 & r_b - 2r_b b_2^* - \mu_b \end{bmatrix}. \tag{3.2.7}$$

Since matrix (3.2.7) is a block matrix, it can be rewritten as

$$J(E_2^*) = \begin{bmatrix} A & B \\ \mathbf{0} & D \end{bmatrix}, \tag{3.2.8}$$

where

$$A = \begin{bmatrix} -(\sigma+\mu) - \left(\frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^{*+}\right) & -\sigma \\ \frac{\beta_3 b_2^*}{\hat{\kappa}+b_2^*} + \hat{\beta}_4 f_{c_3}^{*+} & -(\mu+\gamma+\delta_1) \end{bmatrix}, \quad B = \begin{bmatrix} -\hat{\beta}_4 s_2^* & -\frac{\hat{\kappa}\beta_3 s_2^*}{(\hat{\kappa}+b_2^*)^2} \\ \hat{\beta}_4 s_2^* & \frac{\hat{\kappa}\beta_3 s_2^*}{(\hat{\kappa}+b_2^*)^2} \end{bmatrix}, \quad \mathbf{0} = \begin{bmatrix} 0 & 0 \\ 0 & 0 \end{bmatrix}$$

and $D = \begin{bmatrix} -(\frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} + 2\hat{\beta}_2 f_{c_3}^{*+} + \delta_2 - \hat{\beta}_2) & -\frac{\hat{\kappa}\beta_1(f_{c_3}^{*+}-1)}{(\hat{\kappa}+b_2^*)^2} \\ 0 & r_b - 2r_b b_2^* - \mu_b \end{bmatrix}$.

Now since matrix (3.2.8) is an upper triangular matrix, its eigenvalues are equal to the eigenvalues of the diagonal matrices. Thus, the eigenvalues of matrices A and D.

Since matrix D is an upper triangular matrix, its eigenvalues are the diagonal elements. Thus

$$\lambda_1^* = r_b - (2r_b b_2^* + \mu_b) \leq 0 \quad \text{if} \quad r_b < (2r_b b_2^* + \mu_b),$$
$$\lambda_2^* = J^*\left(\frac{\hat{\beta}_2}{J^*} - 1\right) < 0 \quad \text{if} \quad \frac{\hat{\beta}_2}{J^*} < 1,$$

where

$$J^* = \frac{\beta_1 b_2^*}{\hat{\kappa}+b_2^*} + 2\hat{\beta}_2 f_{c_3}^{*+} + \delta_2.$$

Also for eigenvalues of matrix A we solve

$$\det(A - \lambda^* I) = 0,$$

and obtain the characteristic equation

$$\eta_2 \lambda^{*2} + \eta_1 \lambda^* + \eta_0 = 0, \tag{3.2.9}$$

where

$$\eta_2 = \hat{\kappa} + b_2^* > 0,$$
$$\eta_1 = \beta_3 b_2^* + (\hat{\kappa}+b_2^*)\left[(\mu+\sigma) + (\mu+\gamma+\delta_1) + \hat{\beta}_4 f_{c_3}^{*+}\right] > 0,$$
$$\eta_0 = (\mu+\gamma+\sigma+\delta_1)\left[\beta_3 b_2^* + (\hat{\kappa}+b_2^*)\hat{\beta}_4 f_{c_3}^{*+} + (\hat{\kappa}+b_2^*)(\mu+\sigma)(\mu+\gamma+\delta_1)\right] > 0.$$

So, equation (3.2.9) can be rewritten as

$$\lambda^{*2} + \phi_1 \lambda^* + \phi_0 = 0, \tag{3.2.10}$$

where $\phi_1 = \frac{\eta_1}{\eta_2} > 0$ and $\phi_0 = \frac{\eta_0}{\eta_2} > 0$.

So the eigenvalues of (3.2.10) are given by

$$\lambda^* = \frac{-\phi_1 \pm \sqrt{\phi_1^2 - 4\phi_0}}{2},$$

which means that

$$\lambda_3^* = \frac{-\phi_1 + \sqrt{\phi_1^2 - 4\phi_0}}{2} \quad \text{and} \quad \lambda_4^* = \frac{-\phi_1 - \sqrt{\phi_1^2 - 4\phi_0}}{2}.$$

Therefore equation (3.2.10) has roots with negative real parts by the Routh–Hurwitz stability criterion.

So, the eigenvalues of matrix (3.2.7) are $\lambda_1^*, \lambda_2^*, \lambda_3^*$ and λ_4^*. Therefore the endemic equilibrium will be stable if $r_b < (2r_b b_2^* + \mu_b)$ and $\frac{\hat{\beta}_2}{J^*} < 1$. □

4. Model Simulations

In this section, we simulate the mathematical model for Listeriosis dynamics in order to check the contribution of human, the environment and the factory products for the spread of Listeriosis. The system of equations are solved by using Julia software over a period of time and the estimated parameter values shown in Table 4.1.

4.1 Parameter estimation

We hypothetically choose the following parameter values. This parameters (shown in Table 4.1) are chosen in such away that they yield the results of the analysis of the steady states.

Parameter	Estimated value	Reference
μ	0.028	assumed
γ	0.07	assumed
σ	0.99	assumed
r_b	0.3	assumed
μ_b	0.2	assumed
δ_1	0.004	assumed
δ_2	0.017	assumed
β_1	0.004	assumed
$\hat{\beta}_2$	0.006	assumed
β_3	0.03	assumed
$\hat{\beta}_4$	0.05	assumed
$\hat{\kappa}$	0.0002	assumed

Table 4.1: Parameters and its values used in the Listeriosis model.

4.2 Numerical simulations

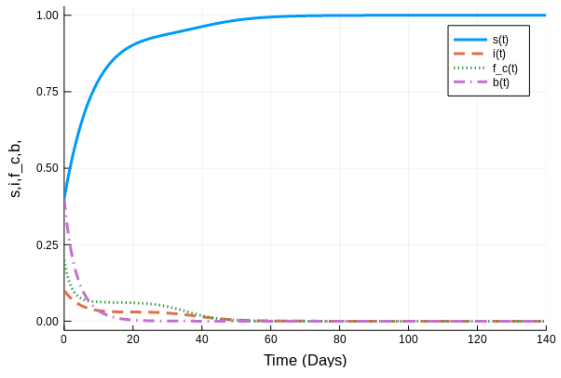

Figure 4.1: Equilibrium point E_0^* with parameter values, $\mu = 0.028, \sigma = 0.1, \gamma = 0.08, \delta_1 = 0.1, \delta_2 = 0.5, r_b = 0.18, \mu_b = 0.4, \beta_1 = 0.004, \hat{\beta}_2 = 0.006, \beta_3 = 0.03, \hat{\beta}_4 = 0.05, \hat{\kappa} = 0.0002$ and initial conditions $s(0) = 0.4, i(0) = 0.1, f_c(0) = 0.2, b(0) = 0.4$.

Figure 4.1 depicts the graph of the equilibrium point E_0^* in which all the infected compartments (group)

tends to zero. Therefore this shows the disease free equilibrium point. We can infer that this is due to the high death rate, $\mu_b = 0.4$, of the bacteria as compared to their growth rate $r_b = 0.18$. So there are less infections caused by the bacteria. Moreover, the rate of removal of contaminated food, $\delta_2 = 0.5$, is higher as compared to the growth rate of food contamination, $\hat{\beta}_2 = 0.006$.

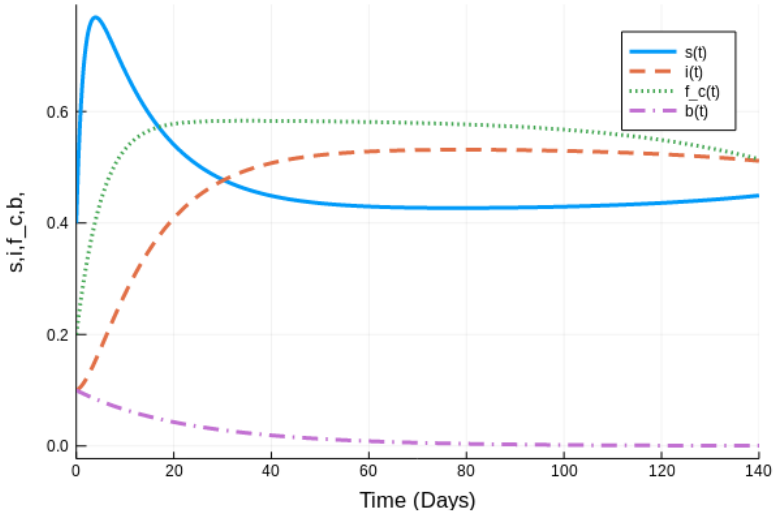

Figure 4.2: Equilibrium point E_1^* with parameter values, $\mu = 0.028, \sigma = 0.6, \gamma = 0.004, \delta_1 = 0.0035, \delta_2 = 0.1, r_b = 0.05, \mu_b = 0.089, \beta_1 = 0.004, \hat{\beta}_2 = 0.16, \beta_3 = 0.1, \hat{\beta}_4 = 0.07, \hat{\kappa} = 0.0002$ and initial conditions $s(0) = 0.4, i(0) = 0.1, f_c(0) = 0.2, b(0) = 0.1$.

Figure 4.2 depicts the graph of equilibrium point E_1^*. We can infer from this graph that the bacteria population goes to extinct since their growth rate is less that their death rate. However, Listeriosis infections exist in the population due to presence of contaminated food. This can be attributed to the fact that the rate of removal of contaminated food, $\delta_2 = 0.1$, is less as compared to the growth rate of food contamination, $\hat{\beta}_2 = 0.16$.

Figure 4.3 shows the graph of the endemic equilibrium point E_2^* in which the contaminated product, the bacteria in the environment and infectious human rate is high. Hence we can observe that the disease will persist.

Figure 4.4 shows how infectious human decrease as disposing (removal) rate increase (δ_2).

Figure 4.5 shows how infectious human decrease (going to zero) as the bacterial natural death (removal) rate μ_b increase.

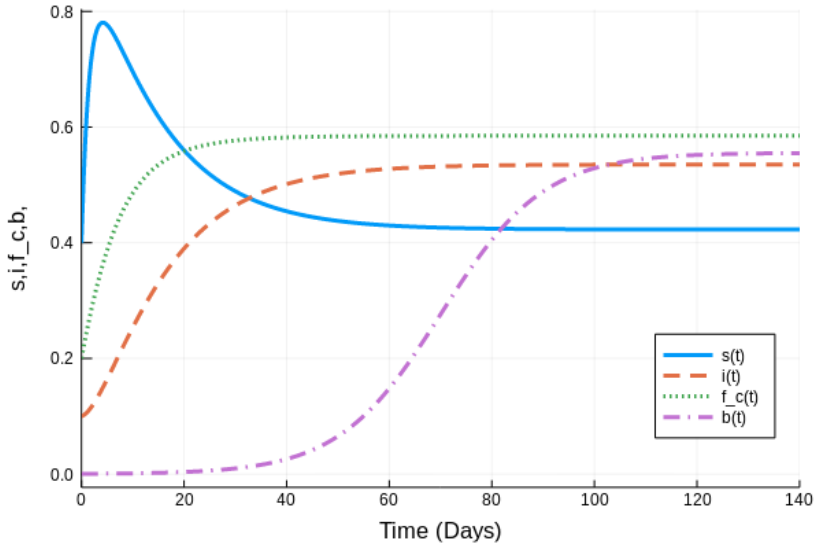

Figure 4.3: Equilibrium point E_2^* with parameter values, $\mu = 0.028, \sigma = 0.6, \gamma = 0.004, \delta_1 = 0.0035, \delta_2 = 0.1, r_b = 0.18, \mu_b = 0.08, \beta_1 = 0.004, \hat{\beta}_2 = 0.16, \beta_3 = 0.1, \hat{\beta}_4 = 0.07, \hat{\kappa} = 0.0002$ and initial conditions $s(0) = 0.4, i(0) = 0.1, f_c(0) = 0.2, b(0) = 0.0005$.

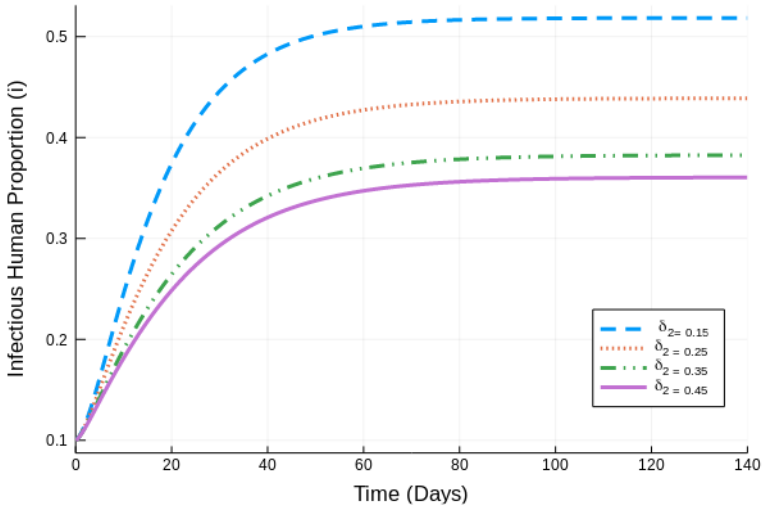

Figure 4.4: Variation of δ_2 in human infection population with parameter values, $\mu = 0.028, \sigma = 0.6, \gamma = 0.004, \delta_1 = 0.0035, \delta_2 = 0.15, 0.25, 0.35, 0.45, r_b = 0.18, \mu_b = 0.08, \beta_1 = 0.004, \hat{\beta}_2 = 0.006, \beta_3 = 0.1, \hat{\beta}_4 = 0.07, \hat{\kappa} = 0.0002$ and initial conditions $s(0) = 0.4, i(0) = 0.1, f_c(0) = 0.2, b(0) = 0.0005$.

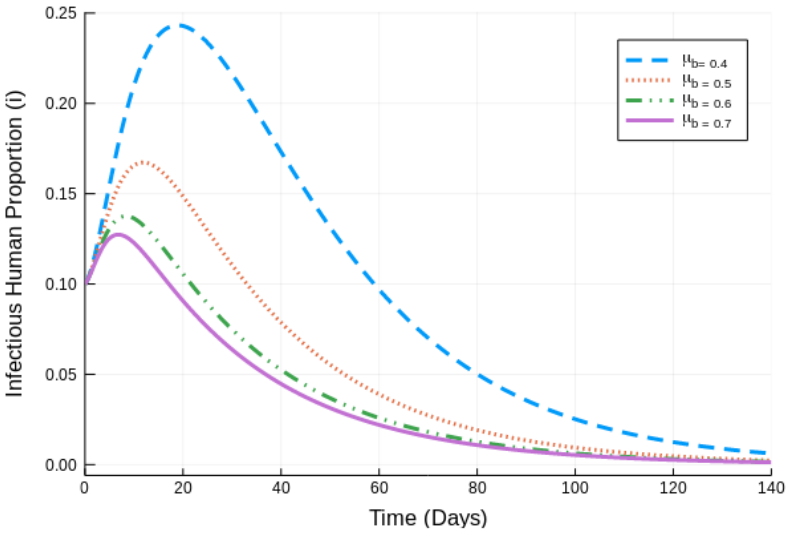

Figure 4.5: Variation of μ_b in human infectious population with parameter values, $\mu = 0.028, \sigma = 0.6, \gamma = 0.004, \delta_1 = 0.0035, \delta_2 = 0.15, r_b = 0.18, \mu_b = 0.4, 0.5, 0.6, 0.7, \beta_1 = 0.004, \hat{\beta}_2 = 0.006, \beta_3 = 0.1, \hat{\beta}_4 = 0.07, \hat{\kappa} = 0.0002$ and initial conditions $s(0) = 0.4, i(0) = 0.1, f_c(0) = 0.2, b(0) = 0.0005$.

5. Discussion, Conclusion and Future work

In this study, we formulated a mathematical model to explain the contribution of food products and bacteria in the environment to the spread of Listeriosis. The model properties were investigated and shown that all the solutions to the model were positive and bounded. As our second objective was to determine the steady states and stability of the equilibrium points of the model, so the steady states of the model were determined. We found out that there are three equilibrium points, namely, the disease free equilibrium point denoted by (E_0^*), the bacteria free equilibrium point (E_1^*) and the endemic equilibrium point (E_2^*).

The stability analyses for the steady states was done using the Jaccobian matrix. We found out that the disease free equilibrium is locally asymptotically stable when the bacteria growth rate r_b is less than its removal/death rate μ_b and the growth rate of food contamination $\hat{\beta}_2$ is less than its removal rate δ_2. The bacteria free equilibrium point is locally asymptotically stable when $r_b < \mu_b$ and $\frac{\hat{\beta}_2}{(2\hat{\beta}_2 f_{c_2}^* + \delta_2)} < 1$ and the endemic equilibrium point is locally asymptotically stable when $r_b < (2r_b b_2^* + \mu_b)$ and $\frac{\hat{\beta}_2}{J^*} < 1$, where $J^* = \frac{\beta_1 b_2^*}{\hat{\kappa} + b_2^*} + 2\hat{\beta}_2 f_{c_3}^{*+} + \delta_2$.

For the numerical simulations, we hypothetically chose the parameter values in such a way that they yield the results of the analyses of the steady states. The result show that the disease can not invade the population provided that the growth rate of food contamination is less than its removal rate, thus $\hat{\beta}_2 < \delta_2$, and the growth rate of bacteria is also less than their death rate, thus $r_b < \mu_b$. Also, the result showed that we can still have Listeriosis driven by the contaminated food even though the Listeria bacteria population in the environment is made to go to extinct. Moreover, the numerical result showed that the disease will persist in the population when $r_b > \mu_b$ and $\hat{\beta}_2 > \delta_2$.

Furthermore, the numerical result showed that as the bacteria growth rate r_b increase also the number of infectious population increase, in addition to that, the infectious population decrease as the removal rate of bacteria increase. Therefore, this study can be useful for the health policies to eradicate Listeriosis if the model is linked to data.

For the future work in the improvement of the developed model, we recommend that the model developed in this study be fitted to data on Listeriosis in order to determine the exact parameter values related to Listeriosis dynamics.

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