



## Mathematical Modeling and Stability Analysis for the Transmission Dynamics of Listeriosis

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**Abstract:** *Listeria monocytogenes* is the bacteria that causes listeriosis, a food-borne illness. Eating contaminated food products can spread the infection to humans. Nevertheless, coming into touch with sick humans or animals might also result in a transfer. In this work, a deterministic mathematical model for the dynamics of listeriosis transmission was developed and assessed in order to evaluate the effect and contribution of treatment interventions for afflicted persons. The model was proven to be well-posed both mathematically and epidemiologically. The contaminated food threshold  $R_c$ , which is an important threshold was obtained using next generation matrix method. The model possessed two equilibrium points namely, the disease-free equilibrium and listeria free-equilibrium points. The disease free- equilibrium was found to be locally and globally stable when the contaminated food threshold is less than unity ( $R_c < 1$ ).

**Key words:** Listeriosis, Mathematical Model, Contaminated food threshold, Stability Analysis.

### 1. Introduction

Food products, both raw and processed, can harbor pathogens such as *Listeria monocytogenes*. These products can infect both people and animals when consumed. Gram-positive bacteria are the kind that this pathogen belongs to [1]. Low temperatures and dirty conditions are ideal habitats for *Listeria* growth. High concentrations of salt and acidic environments are other favorable conditions for their survival. They are specifically found in soil, vegetation, refrigerators, dirty water bodies (lakes, rivers, etc.), animal feces, and meals like smoked salmon, cold meats, and soft cheeses. Poultry and cattle are among the animals that can harbor this pathogen. They readily affect raw meals since they are found in soil and vegetation. Furthermore, using dirty processing tools and the presence of contaminated raw food might contaminate processed food [2] Such tainted food products can spread to humans through consumption. To a lesser level, contact with diseased humans or animals can also result in a transmission. Unborn children can contract these illnesses from infected pregnant mothers and female animals, a process often referred to as vertical transmission [3]. People with weakened immune systems from conditions including cancer, diabetes, kidney disease, HIV

AIDS patients, and the elderly are among the groups of persons who are most likely to contract this disease [4].

Listeriosis is characterized by fever, u-like symptoms, vomiting, nausea, and diarrhea. As with most bacterial diseases, listeriosis can be treated and avoided by following basic food storage procedures, such as not putting items in the refrigerator after their expiration date, properly preparing meat and poultry, and separating raw food from other foods and utensils [5]. While Listeriosis is a relatively uncommon disease, it is often severe and has high rates of hospitalization and death. For example, in 2006, the Czech Republic reported 10 cases of the disease linked to the distribution of local soft cheese; in the same year, Germany reported 16 cases linked to pre-sliced ready-to-eat meat products [6]. Additionally, the National Institute of Communicable Disease (NICD) in South Africa reported that by February 28, 2018, there had been 943 laboratory-confirmed cases of Listeriosis with 176 deaths from the disease [7].

The transmission dynamics of infectious diseases have previously been examined using mathematical models. Listeriosis transmission has been studied using a variety of statistical techniques and mathematical models, as seen, for example, in [8–12]. It can be seen from [13] that the proliferation of *Listeria monocytogens* is dependent upon the interplay of five different variables: Potential of hydrogen (pH), temperature, atmosphere, sodium nitrate, and sodium chloride. When a high concentration of salt, an acidic pH, vacuum packing, and sufficient refrigeration are combined. A study by [14] examined the relationship between a few related food processing parameters and the contamination of ready-to-eat food (smoked ham) with *Listeria monocytogens*. In their investigation, they created a model in which they identified staff gloves and surfaces that come into touch with food as critical components of food contamination. It was shown that ensuring that raw food and processing materials are free of contamination before they are used in food processing is the best method of preventing food contamination. Ready-to-eat meats and soft cheese are considered to be high-risk foods for vulnerable persons, based on the outbreak of the disease that has been documented in Japan, Europe, and North America [15]. In addition, [16] noted that in order to successfully implement food safety measures against listeriosis, food workers must receive education and training on how to guard against *Listeria monocytogen* contamination of ready-to-eat foods as well as how to advise customers to be vigilant about their own food safety. In order to create a predictive model that mimics the proliferation of *Listeria monocytogene* in soft blue-white cheese, a study was carried out in [17]. Additionally, [12] created a mathematical model to investigate the impact of animal vaccinations on the transmission of listeriosis to people and other animals (as *Listeria* vectors). According to their data, a drop in both the rate of animal and human mortality and the pace of animal recovery can lead to an increase in secondary infections.

There are currently very few mathematical models that take into account the effects of contaminated food products and the environment. The goal of developing a basic model that calculates the involvement of food products and bacteria in the dynamics of listeriosis transmission in the human population is to examine the function of the few existing control measures in the case of an outbreak. The control measures include lowering the infection rate, getting rid of contaminated food products, and maintaining cleanliness throughout the food production processes. Although the model on this research provides a crude representation

of the dynamics of listeriosis infection, the findings have a significant impact on the long-term management and quality assessment of control measures. Even though the model is basic, we contend that it provides important insights into the modeling of listeriosis caused by tainted food goods and environmental microorganisms.

## 2. Listeriosis Model Description and Formulation

This section presents the development of a mathematical model for the dynamics of listeriosis, which is composed of three parts: the human population, Listeria, and industrial products. The human population is made up of four vulnerable compartments. susceptible humans  $S_h(t)$ , infected humans  $I_h(t)$ , Treated human  $T_h(t)$ , and the recovered humans  $R_h(t)$ . Individuals are recruited into the susceptible class at a rate proportional to the total human population  $N(t)$ , so that the recruitment is modelled by  $\Lambda_H$ , where  $\mu$  is the natural birth/mortality rate. The susceptible humans can move into the infected class either by acquiring Listeriosis through eating contaminated products or through contact with contaminated material from the environment with a force of infection  $\lambda_h(t)$ , defined after system (2). Furthermore, the infected humans population can either die naturally or die of the disease at a rate of  $\mu I$  and  $\delta_i$  respectively or recover at a rate of  $\gamma$  and join the recovered class. Treated individual population  $T_h(t)$  is increased by progression rate from infected individuals  $\phi$  and decreased by natural death  $\mu T$  and by recovery rate  $\pi$  due to treatment. Individual from the Recovered class become susceptible at a rate  $\sigma R$ . We are assuming that there is no human to human transmission. Considering the factory dynamics, we are assuming that the amount of food products,  $F(t)$  comprises of non-contaminated,  $F_n(t)$  and contaminated  $F_c(t)$  food products. We assume that factory products are manufactured or produced at a rate of  $\delta_2 F$ . By contact with contaminated surfaces and contaminated products, the non-contaminated products can become contaminated with Listeria bacteria. Non-contaminated factory products become contaminated with a force of infection  $\lambda_f(t)$  defined after system (2). The Listeria bacteria in the environment is assumed to grow at a rate of  $r_b$  and die at a rate  $\mu_b$ . The growth of the bacteria is assumed to be logistic with a carrying capacity of  $K$ . We assume that, at any time,  $t$ , the human population and factory food products are constant and respectively given by:

$$\left. \begin{aligned} N(t) &= S_h(t) + I_h(t) + T_h(t) + R_h(t) \\ F(t) &= F_n(t) + F_c(t) \end{aligned} \right\} \dots\dots\dots(1)$$

**Model Equations of the System**

$$\left. \begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_H + \sigma R_h - (\lambda_h + \mu)S_h \\
 \frac{dI_h}{dt} &= \lambda_h S_h - (\mu + \delta_1 + \gamma + \varphi)I_h \\
 \frac{dT_h}{dt} &= \varphi I - (\mu + \pi)T \\
 \frac{dR_h}{dt} &= \gamma I + \pi T - (\mu + \sigma)R_h \\
 \frac{dB}{dt} &= r_b B \left(1 - \frac{B}{K}\right) - \mu_b B \\
 \frac{dF_n}{dt} &= \Lambda_F - (\lambda_f + \delta_2)F_n \\
 \frac{dF_c}{dt} &= \lambda_f F_n - \delta_2 F_c
 \end{aligned} \right\} \dots\dots\dots(2)$$

Where

$$\begin{aligned}
 \lambda_f(t) &= \beta_2 B + \beta_3 F_c \\
 \lambda_h(t) &= \beta_1 F_c
 \end{aligned}$$

are two forces of infections

**Table 1**

VARIABLE	DESCRIPTION
$S_h(t)$	Population of Susceptible Individuals at time $t$
$I_h(t)$	Population of Infected individuals at time $t$
$T_h(t)$	Population of Treated individuals at time $t$
$R_h(t)$	Population of Recovered Humans at time $t$
$F_n(t)$	Non-conterminated food Product at time $t$
$F_c(t)$	Conterminated food Product at time $t$
$B$	Bacteria ( <i>L. monocytogenes</i> ) at time $t$

**Table 2**

PARAMETER	DESCRIPTION
$\Lambda_h$	Recruitment rate in to Susceptible human Population
$\beta$	Rate of Transmission
$\mu_h$	Natural death rate of human
$\mu_b$	Natural death rate of bacteria
$\pi$	Progression rate from Treated individuals to Recovered individuals
$K$	Bacteria carrying capacity
$\delta_I$	The death rate due to bacteria
$\gamma$	Progression rate of individuals from infected class to recovered class
$r_b$	Bacteria net Growth rate
$\varphi$	Progression rate from Infected individual to Treated individual
$\sigma$	Rate of Recovered individuals to be become Susceptible individuals

### 3. Basic Properties of the Model

#### 3.1 Positivity of solutions

It is important to show that the state variables of the model are non-negative for all  $t > 0$ . If this result is established, then it is sufficient to say that the model given is system (1) is mathematically and biologically reasonable. This will be achieved through the following theorem.

**Theorem 1:** Let the initial data be

$$S_h(0) > 0, I_h(0) \geq 0, T_h(0) \geq 0, R_h(0) \geq 0, B \geq 0, F_n(0) > 0, F_c(0) \geq 0.$$

Then the solutions  $(S_h, I_h, T_h, R_h, B, F_n, F_c)$  of the model given by system (1), with positive initial data, will remain positive for all time  $t > 0$ .

**Proof:** Let

$$t_1 = \sup \{t > 0 : S_h(t) > 0, I_h(t) > 0, T_h(t) > 0, R_h(t) > 0, B > 0, F_n(t) > 0, F_c(t) > 0\}$$

Thus,  $t_1 > 0$ . From the first equation of our model equations (1), we have

$$\frac{dS_h}{dt} + (\lambda_h + \mu)S_h \geq \Lambda_H \tag{3}$$

solving equation (8) using integral factor method gives

$$\frac{d}{dt} \left\{ S_h \exp \left[ \mu t + \int_0^t \lambda_h(u) du \right] \right\} \geq \Lambda \exp \left[ \mu t + \int_0^t \lambda_h(u) du \right]$$

integrating both sides, we have

$$S_h(t) \exp \left[ \mu t + \int_0^t \lambda_h(u) du \right] - S_h(0) \geq \Lambda_H \int_0^t \left[ \exp \left\{ \mu x + \int_0^x \lambda_h(u) du \right\} \right] dx \quad (4)$$

so that

$$S_h(t) \geq S_h(0) \exp \left[ - \left\{ \mu t + \int_0^t \lambda_h(u) du \right\} \right] + \exp \left[ - \left\{ \mu t + \int_0^t \lambda_h(u) du \right\} \right] \times \Lambda_H \int_0^t \left[ \exp \left\{ \mu x + \int_0^x \lambda_h(u) du \right\} \right] dx$$

This implies that but  $S_h(t) > 0$  for  $t > 0$ , hence,  $S_h(t) > 0$  for  $t > 0$ .

From the second equation of system (1), we have,

$$\frac{dI_h}{dt} \geq -(\mu + \delta_1 + \gamma + \varphi) I_h \quad (5)$$

Using separation of variable method, equation (5) becomes

$$\int_0^{t_1} \left( \frac{1}{I_h} \right) dI_h \geq -(\mu + \delta_1 + \gamma + \varphi) \int_0^{t_1} dt$$

solving the above equation yields,

$$\ln \left( \frac{I_h(t_1)}{I_h(0)} \right) \geq -(\mu + \delta_1 + \gamma + \varphi) t_1$$

Exponentiating both sides gives tha,

$$I_h(t_1) \geq I_h(0) \exp \left[ -(\mu + \delta_1 + \gamma + \varphi) t_1 \right] > 0$$

Thus,  $I_h(t) > 0$  for  $t > 0$ , therefore,  $I_h(t) > 0$  for  $t > 0$ . In the same manner, it can be shown that:

$$\left. \begin{aligned} T_h(t_1) &\geq T_h(0) \exp \left[ -(\mu + \pi) t_1 \right] > 0, \\ R_h(t_1) &\geq R_h(0) \exp \left[ -(\mu + \sigma) t_1 \right] > 0, \\ B(t_1) &\geq B(0) \exp \left[ -\mu_b t_1 \right] > 0 \end{aligned} \right\} \quad (6)$$

Also, from the sixth equation of system (1), we have

$$\frac{dF_n}{dt} + (\lambda_F + \delta_2) F_n \geq \Lambda_F \quad (7)$$

solving equation (7) using integral factor method gives

$$\frac{d}{dt} \left\{ F_n \exp \left[ \delta_2 t + \int_0^t \lambda_F(u) du \right] \right\} \geq \Lambda_F \exp \left[ \delta_2 t + \int_0^t \lambda_F(u) du \right]$$

integrating both sides, we have

$$F_n(t) \exp \left[ \delta_2 t + \int_0^t \lambda_F(u) du \right] - F_n(0) \geq \Lambda_F \int_0^t \left[ \exp \left\{ \delta_2 x + \int_0^x \lambda_F(u) du \right\} \right] dx \quad (8)$$

so that

$$F_n(t) \geq F_n(0) \exp \left[ - \left\{ \delta_2 t + \int_0^t \lambda_F(u) du \right\} \right] + \exp \left[ - \left\{ \delta_2 t + \int_0^t \lambda_F(u) du \right\} \right] \times \Lambda_F \int_0^t \left[ \exp \left\{ \delta_2 x + \int_0^x \lambda_F(u) du \right\} \right] dx$$

This implies that  $F_n(t) > 0$  for  $t > 0$ , hence,  $F_n(t) > 0$  for  $t > 0$ .

Similarly, from the last equation of system (1), we have

$$\frac{dF_c}{dt} \geq -\delta_2 F_c \tag{9}$$

Using separation of variable method, and integrating both sides of equation (9), we obtained

$$\int_0^{t_1} \left( \frac{1}{F_c} \right) dF_c \geq -\delta_2 \int_0^{t_1} dt$$

So that

$$\ln \left( \frac{F_c(t_1)}{F_c(0)} \right) \geq -\delta_2 t_1$$

Hence,

$$F_c(t_1) \geq F_c(0) \exp[-\delta_2 t_1] > 0$$

This implies that  $F_c(t) > 0$  for  $t > 0$ , hence,  $F_c(t) > 0$  for  $t > 0$ .

### 3.2 Boundedness of solutions

The feasible region of the model is established by the following theorem.

**Theorem 2:** The closed set  $\Omega = \Omega_h \cup \Omega_b \cup \Omega_F \subset \mathbb{R}_+^4 \times \mathbb{R}_+ \times \mathbb{R}_+^2$ , with

$$\Omega_h = \left\{ S_h, I_h, T_h, R_h \in \mathbb{R}_+ : N_H \leq \frac{\Lambda_H}{\mu} \right\}, \Omega_F = \left\{ F_n, F_c \in \mathbb{R}_+ : F \leq \frac{\Lambda_F}{\delta_2} \right\}, \Omega_b = \{ B \in \mathbb{R}_+ : B \leq K \}$$

is positively invariant with respect to model equations (1).

**Proof:** The total human population is denoted by  $N_H$  and is given by

$$N_H(t) = S_h(t) + I_h(t) + T_h(t) + R_h(t)$$

and is differentiated and summed together to have

$$\frac{dN_H(t)}{dt} = \Lambda_H - \mu N_H - \delta_1 I_h \tag{10}$$

in absence of mortality due to Listeria (i.e.  $\delta_1 = 0$ ) then by standard comparison theorem and rearranging equation (10), we obtain

$$\frac{dN_H(t)}{dt} + \mu N_H \leq \Lambda_H \tag{11}$$

solving equation (11) by integral factor method, we have

$$N_H(t) \leq \frac{\Lambda_H}{\mu} [1 - e^{-\mu t}] + N_H(0) e^{-\mu t}$$

as  $t \rightarrow \infty$ , we have that

$$N_H(t) \leq \frac{\Lambda_H}{\mu} \quad (12)$$

Similarly, taking the bacteria population from system (1), i.e.

$$\frac{dB}{dt} \leq r_b \left(1 - \frac{B}{K}\right) B$$

Using separation of variables, we have

$$B(t) \leq \frac{KB(0)}{B(0) + (K - B(0))e^{-r_b t}} \quad (13)$$

Taking the limit as  $t \rightarrow \infty$  equation (13) becomes

$$B(t) \leq K$$

following a similar approach yields similar result for food products.

$$F(t) \leq \frac{\Lambda_F}{\delta_2} \quad (14)$$

Thus, we have shown that  $\Omega$  is positively invariant and attracts all solutions of model equation (1) in finite time. This guarantees that our investigation and analyses will be carried out in a feasible region and that every solution of our model having initial conditions in  $\Omega$  will always remain in  $\Omega$  for all time  $t > 0$ .

### 3.3 Existence of Disease Free Equilibrium Point

To obtain the equilibrium point of the model equations given by system (1), we set the RHS to zero. Also, at equilibrium state we set,

$$\begin{aligned} \Lambda_H + \sigma R_h - (\lambda_h + \mu)S_h &= 0 \\ \lambda_h S_h - (\mu + \delta_1 + \gamma + \varphi)I_h &= 0 \\ \varphi I - (\mu + \pi)T &= 0 \\ \gamma I + \pi T - (\mu + \sigma)R_h &= 0 \\ r_b B \left(1 - \frac{B}{K}\right) - \mu_b B &= 0 \\ \Lambda_F - (\lambda_f + \delta_2)F_n &= 0 \\ \lambda_f F_n - \delta_2 F_c &= 0 \end{aligned} \quad (15)$$

From the fifth equation of (15), we have

$$B \left[ r_b \left(1 - \frac{B}{K}\right) - \mu_b \right] = 0 \quad (16)$$

Equation (14) gives the following cases:

- i. Case I:  $B = 0$
- ii. Case II:  $r_b \left(1 - \frac{B}{K}\right) - \mu_b = 0$

Now, using case I, i.e.  $B = 0$  in the sixth equation of system (15) we obtain



$$F_n^* = \frac{\Lambda_F}{\beta_2 F_c^* + \delta_2} \quad (17)$$

Substituting equation (17) into the last equation of system (15), we obtain

$$F_c^* = 0 \text{ or } F_c^* = \frac{\Lambda_F \beta_2 - \delta_2^2}{\beta_2 \delta_2} \quad (18)$$

### 3.4 Disease-free Equilibrium Point

If  $B = 0$  and  $F_c^* = 0$ , then there exists a unique Listeriosis-free equilibrium point,  $\chi^0$  of the model system (1) given below

$$\chi^0 = \left[ \frac{\Lambda_H}{\mu}, 0, 0, 0, 0, \frac{\Lambda_F}{\delta_2}, 0 \right]$$

### 3.5 Listeria-free Equilibrium Point

Considering the second case,  $r_b \left(1 - \frac{B}{K}\right) - \mu_b = 0$ , solving for  $B$ , we have

$$B = \frac{K \mu_b}{r_b} (R_b - 1) \quad (19)$$

Where  $R_b = \frac{r_b}{\mu_b}$ . It follows that  $B > 0$  whenever  $R_b > 1$ . The quantity  $R_b$  in equation (19) is called the bacteria reproduction rate. Thus, the listeria-free equilibrium point of the model system (15) denoted by  $\chi^1$  is given as

$$\chi^1 = \left[ \frac{\Lambda_H}{\mu}, 0, 0, 0, \frac{K \mu_b}{r_b} [R_b - 1], \frac{\Lambda_F}{\delta_2}, 0 \right] \quad (20)$$

### 3.6 Contaminated Food Threshold

Let  $R_c$  denotes the food products contaminated threshold. In this study, food products contamination threshold is equivalent to the basic reproduction number used in infectious disease modeling which is usually denoted by  $R_0$ .

Consider the following equations from model system (1)

$$\left. \begin{aligned} \frac{dI}{dt} &= \lambda_h S_h - (\mu + \delta_1 + \gamma + \varphi) I_h \\ \frac{dT}{dt} &= \varphi I - (\mu + \pi) T \\ \frac{dB}{dt} &= r_b B \left(1 - \frac{B}{K}\right) - \mu_b B \\ \frac{dF_c}{dt} &= \lambda_f F_n - \delta_2 F_c \end{aligned} \right\} \quad (21)$$

Let

$$F = \begin{bmatrix} \lambda_h S_h \\ 0 \\ 0 \\ \lambda_f F_c \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} (\mu + \delta_1 + \gamma + \varphi) I_h \\ -\varphi I_h + (\mu + \pi) T \\ -r_b B \left(1 - \frac{B}{K}\right) + \mu_b B \\ \delta_2 F_c \end{bmatrix}$$

Evaluating the Jacobian of  $F$  and  $V$  at the disease-free equilibrium point  $\chi^0$ , we obtain

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_1 \Lambda_H}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_3 \Lambda_F}{\delta_2} & \frac{\beta_2 \Lambda_F}{\delta_2} \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \mu + \delta_1 + \gamma + \varphi & 0 & 0 & 0 \\ -\varphi & \mu + \pi & 0 & 0 \\ 0 & 0 & -r_b + \mu_b & 0 \\ 0 & 0 & 0 & \delta_2 \end{bmatrix} \quad (22)$$

So that

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu + \delta_1 + \gamma + \varphi} & 0 & 0 & 0 \\ \frac{\varphi}{(\mu + \delta_1 + \gamma + \varphi)(\mu + \pi)} & \frac{1}{\mu + \pi} & 0 & 0 \\ 0 & 0 & \frac{1}{-r_b + \mu_b} & 0 \\ 0 & 0 & 0 & \frac{1}{\delta_2} \end{bmatrix} \quad (23)$$

Computing the eigenvalues of  $FV^{-1}$  to obtain the dominant eigenvalue which will be the food product contaminated threshold denoted by  $R_c = \rho(FV^{-1})$  and is given as

$$R_c = \frac{\beta_2 \Lambda_F}{\delta_2}$$

### 3.7 Local Stability Analysis of Disease-free Equilibrium Point

**Theorem 3.** The disease-free equilibrium point  $\chi^0$  of the model is locally asymptotically stable whenever  $R_b < 1$ ,  $R_c < 1$  and unstable otherwise.

**Proof:** The Jacobian matrix of model system (1) evaluated at disease-free equilibrium is given as

$$J(\chi^0) = \begin{bmatrix} -\mu & 0 & 0 & \sigma & 0 & 0 & \frac{-\beta_1 \Lambda_H}{\mu} \\ 0 & -(\mu + \delta_1 + \gamma + \varphi) & 0 & 0 & 0 & 0 & \frac{\beta_1 \Lambda_H}{\mu} \\ 0 & \varphi & -(\mu + \pi) & 0 & 0 & 0 & 0 \\ 0 & \gamma & \pi & -(\mu + \sigma) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_b (R_b - 1) & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{-\beta_3 \Lambda_F}{\delta_2} & -\delta_2 & \frac{-\beta_2 \Lambda_F}{\delta_2} \\ 0 & 0 & 0 & 0 & \frac{\beta_3 \Lambda_F}{\delta_2} & 0 & \frac{\beta_2 \Lambda_F}{\delta_2} - \delta_2 \end{bmatrix}$$

(24)

From the above Jacobian matrix, it is obvious that the eigenvalues are given as

$$\lambda_1 = -\mu, \lambda_2 = -(\mu + \delta_1 + \gamma + \varphi), \lambda_3 = -(\mu + \pi), \lambda_4 = -(\mu + \sigma), \lambda_5 = -\delta_2$$

$$\lambda_6 = \mu_b (R_b - 1) < 0 \text{ whenever } R_b < 1 \text{ and } \lambda_7 = \delta_2 (R_c - 1) < 0 \text{ when } R_c < 1.$$

### 3.8 Global Stability Analysis of Disease-free Equilibrium Point

**Theorem 4.** The disease-free equilibrium point  $\chi^0$  of the model is globally asymptotically stable whenever  $R_c < 1$  and unstable otherwise.

**Proof:** First, the model equations are written in the form

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

$$\frac{dZ}{dt} = G(X, Z), G(X, 0) = 0$$

where  $X = (S_h, R_h, F_n) \in \mathbb{R}^3$  represents the number on uninfected components  $Z = (I_h, T_h, B, F_c) \in \mathbb{R}^4$  denotes the number of infected components. The following assumptions must be satisfied for the disease free equilibrium point of model system (1) to be globally asymptotically stable:

$$H_1 : \frac{dX}{dt} = F(X, 0), \chi^0 \text{ is locally asymptotically stable}$$

$$H_2 : \frac{dZ}{dt} = G(X, Z) = AW - \mathcal{G}(X, Z) \text{ where } \mathcal{G}(X, Z) \geq 0$$

for  $\mathcal{G}(X, Z) \in \Omega$  and  $A = D_w G(X, 0)$  is an M-matrix (the off-diagonal elements are non-negative). Now, consider the reduced system

$$\frac{dX}{dt} = F(X, 0) = \begin{bmatrix} \Lambda_H - \mu S_h \\ 0 \\ \Lambda_F - \delta_2 F_n \end{bmatrix} \quad (25)$$

The reduced system (15) is globally asymptotically stable at the disease-free equilibrium point  $\chi^0 = \left[ \frac{\Lambda_H}{\mu}, 0, 0, 0, 0, \frac{\Lambda_F}{\delta_2}, 0 \right]$ . It can be shown from the first equation of (16) that  $S_h(t) = \frac{\Lambda_H}{\mu} + \left( S_h(0) - \frac{\Lambda_H}{\mu} \right) e^{-\mu t}$ , this implies that  $S_h(t) \rightarrow \frac{\Lambda_H}{\mu}$  as  $t \rightarrow \infty$ . Similarly, from the second and third equations of (16),  $F_n(t) \rightarrow \frac{\Lambda_F}{\mu}$ . It can be observed that this particular dynamic does not depend on the initial conditions. Hence, the convergence of the solutions of reduced system (18) is global in  $\Omega$ .

For the second condition  $H_2$ , we have

$$\frac{dZ}{dt} = G(X, Z) = \begin{bmatrix} \beta_1 F_c S_h - (\mu + \delta_1 + \gamma + \varphi) I_h \\ \varphi I - (\mu + \pi) T \\ r_b B \left( 1 - \frac{B}{K} \right) - \mu_b B \\ (\beta_3 B + \beta_2 F_c) F_n - \delta_2 F_c \end{bmatrix} \quad (26)$$

$\mathcal{G}(X, Z) = AZ - G(X, Z)$  where  $A = D_w G(X, 0)$  is a metzler matrix.

$$AZ = \begin{bmatrix} \beta_1 F_c S_h^0 - (\mu + \delta_1 + \gamma + \varphi) I_h \\ \varphi I - (\mu + \pi) T \\ B(r_b - \mu_b) \\ (\beta_3 B + \beta_2 F_c) F_n^0 - \delta_2 F_c \end{bmatrix} \quad (27)$$

So that

$$\mathcal{G}(X, Z) = \begin{bmatrix} \beta_1 F_c S_h^0 \left( 1 - \frac{S_h}{S_h^0} \right) \\ 0 \\ \frac{r_b B^2}{K} \\ (\beta_3 B + \beta_2 F_c) F_n^0 \left( 1 - \frac{F_n}{F_n^0} \right) \end{bmatrix} \quad (28)$$

Since,  $S_h \leq S_h^0, F_n \leq F_n^0$ , then  $\mathcal{G}(X, Z) > 0$ . Hence, the disease-free equilibrium point is globally asymptotically stable.

## Conclusion

In this work, a deterministic mathematical model for the dynamics of food-borne listeriosis transmission was formulated and analysed in order to evaluate the effect and contribution of treatment interventions for afflicted persons. The model was proven to be well-posed both mathematically and epidemiologically. The contaminated food threshold  $R_c$ , which is an important threshold was obtained using next generation matrix method. The model possessed two equilibrium points namely, the disease-free equilibrium and listeria free-equilibrium points. The disease free- equilibrium was found to be locally and globally stable when the contaminated food threshold is less than unity ( $R_c < 1$ ).

## Recommendations

To decrease the spread of listeriosis, Healthcare agencies should take all the necessary measures in terms of sensitizing the general public about preventions which includes:

1. Pregnant women should avoid eating high-risk foods such as raw sprouts, Deli meats and soft cheeses
2. For the older Adult, they should be taking extra precautions to handle and cook food safely
3. Wash hands thoroughly before and after handling food, make sure to clean and sanitize all utensils and surfaces
4. Separate raw meat, poultry and seafood from ready-to-eat foods to prevent cross-contamination.
5. Listeriosis is typically treated with antibiotics, such as ampicillin or penicillin, gentamicin and Trimethoprim-sulfamethoxazole may be used as an alternative antibiotic in certain cases.

## Acknowledgement

Authors are grateful to the Gombe State Polytechnic, Bajoga and Tetfund IBR Grants 2024 intervention for their financial support throughout this research.

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