Int. J. Anal. Appl. (2026), 24:160

International Journal of Analysis and Applications

Mathematical Analysis of a Co-Dynamics Model of Listeriosis and Bacterial Meningitis Starting From Ready-to-Eat Foods

Ousmane Koutou[1,*], Cheick Abdoul Kabir Kouanda[2], Adama Ouédraogo[2]

[1]*CUP-Kaya/Université Joseph KI-ZERBO, Ouagadougou, Burkina Faso*
[2]*Département de Mathématiques, Université Nazi BONI, Bobo Dioulasso, Burkina Faso*

[*]*Corresponding author:* koutousman@gmail.com

Abstract. Listeriosis is a foodborne illness that can lead to bacterial meningitis. In this paper, we propose and investigate a model of co-infection involving both listeriosis and bacterial meningitis, originating from ready-to-eat food products. We begin by examining the patterns of each disease individually, as well as the dynamics of their co-infection. From our global co-infection model, we derive separate models for bacterial meningitis and listeriosis. Each model is analyzed in terms of the existence of equilibrium points, threshold dynamics parameters, stability results, sensitivity analysis, and the presence of backward and forward bifurcations. Finally, we present numerical simulation results to support our theoretical findings.

1. Introduction

Listeriosis is a bacterial infection caused by Listeria monocytogenes, a Gram-positive bacterium. The incidence of listeriosis ranges from 0.1 to 10 cases per million inhabitants per year [22]. This pathogen was first identified in 1924 and recognized as a human infection in 1981 [16]. Among the five strains of Listeria, only Listeria monocytogenes is pathogenic to humans and animals [10]. Listeriosis is primarily contracted through the ingestion of contaminated food, direct contact with contaminated environments, or interaction with infected individuals [13, 23]. On the other hand, bacterial meningitis is a serious illness characterized by the inflammation of the meninges, the protective membranes covering the brain and spinal cord. The primary causative agent of this disease is the bacterium Neisseria meningitidis, commonly known as meningococcus, which is classified as a Gram-negative bacterium [5, 10, 27]. Symptoms of bacterial meningitis have been documented since ancient times, with the first clinically recognized epidemic occurring in Geneva

Received: May 18, 2025.

2020 *Mathematics Subject Classification.* 92D30.

Key words and phrases. Listeriosis/bacterial meningitis; co-infection modeling; bifurcation; sensitivity analysis; Numerical simulations.

https://doi.org/10.28924/2291-8639-24-2026-160
ISSN: 2291-8639

in 1805. The first recorded epidemic in Africa took place in the 1840s [1]. The african meningitis belt, comprising 26 countries, reports the highest incidence rates, with approximately 80,000 cases and over 4,000 deaths annually [20]. Bacterial meningitis can be transmitted through direct contact with an infected person via respiratory droplets, contact with contaminated environments, or through Listeria bacteria in immunocompromised individuals [5, 15].

The co-infection of bacterial meningitis and listeriosis is a significant public health concern, particularly due to their geographical overlap. Both diseases share common symptoms, including fever, headache, nausea, vomiting, drowsiness, and loss of consciousness. Specific symptoms of bacterial meningitis include a stiff neck, sensitivity to light, and a rash, while listeriosis can lead to complications in pregnant women and neurological issues such as meningitis and encephalitis. Notably, individuals infected with listeriosis may develop meningitis as a complication [21]. Numerous studies have established that Listeria monocytogenes is a frequent cause of bacterial meningitis, particularly among the elderly and immunocompromised populations. For instance, Laguna-Del Estal *et al*. [24] demonstrated that Listeria monocytogenes predominantly affects individuals with co-morbidities or weakened immune systems, although it can also occur in otherwise healthy individuals. Additionally, R. Amaya-Villar *et al*. [26] reported a high incidence of community-acquired acute Listeria meningitis in adults, recommending against the use of aminoglycosides to improve patient outcomes. A case study by A. Mancini [4] highlighted a case of Listeria monocytogenes meningitis resulting from a typical foodborne pathogen, particularly dangerous for patients with compromised cellular immunity. Despite the existing literature, there is a limited number of mathematical models that explore the dynamics of co-infection between listeriosis and bacterial meningitis. One notable study by C. W. Chukwu *et al*. [10] proposed a mathematical model to describe the co-infection dynamics, emphasizing the logistic growth of bacteria. Their results indicate that reducing Listeria pathogens and increasing the cure rate for bacterial meningitis could significantly decrease the incidence of co-infection.

In this study, we analyze a deterministic model of co-infection between listeriosis and bacterial meningitis, emphasizing key biological and epidemiological characteristics of both diseases. A novel aspect of our research is the consideration of how ready-to-eat food influences the dynamics of co-infection. With this foundation laid, the rest of the manuscript is organized as follows: Section 2 outlines the co-infection model and its well-posedness more precisely the existence, uniqueness, positivity, and boundlessness of solutions. Section 3 focuses on the mathematical analysis of the bacterial meningitis-only model, followed by the listeriosis-only model in Section 4, and the co-infection model in Section 5. Section 6 presents numerical simulations that validate several analytical findings. Finally, we end with some concluding remarks in Section 7.

2. Co-infection model formulation and its well-posedness

2.1. **Model formulation.** Our model consists of eight compartments organized into three main components: the human population $N(t)$, the bacterial population (Listeria) $B(t)$, and the read-

to-eat food products $F(t)$. The human population is divided into five compartments: susceptible individuals $S(t)$, individuals suffering from listeriosis $I_l(t)$, bacterial meningitis infected people $I_m(t)$, listeriosis and bacterial meningitis co-infected individuals $I_{lm}(t)$, and those who have recovered from bacterial meningitis-only are denoted by $R_m(t)$. Thus, the total human population reads as: $N(t) = S(t) + I_m(t) + R_m(t) + I_l(t) + I_{lm}(t)$. The total amount of ready-to-eat food products is split into two compartments: uncontaminated food products $F_u(t)$ and contaminated food products $F_c(t)$, therefore $F(t) = F_u(t) + F_c(t)$. However, individuals enter the susceptible compartment at a constant recruitment rate Λ, while all compartments of the human population experience a natural death rate μ. Moreover, a susceptible individual can become infected with listeriosis and bacterial meningitis at infection forces $\lambda_l = \beta_l(B + \alpha_3 F_c)$ and $\lambda_m = \beta_m(I_m + \alpha_1 I_{lm} + \alpha_2 B)$, respectively, where β_l and β_m represent the effective contact rates for listeriosis and bacterial meningitis infections. The parameters, α_1 and α_2 account for the relative transmissibility of bacterial meningitis from individuals in the I_{lm} and B compartments, respectively, while α_3 represents the relative transmissibility of listeriosis from the contaminated food products F_c. Individuals from compartments I_m and I_l can progress to the co-infected compartment I_{lm} at the infection forces λ_l and λ_m, respectively, or die at rates δ_m and δ_l. The mortality rate due to co-infection is denoted as δ_{lm}. Additionally, we assume that individuals from compartments I_{lm} and I_m can only recover from bacterial meningitis and move to the recovery compartment R_m at rates σ_{lm} and σ_m, respectively. Individuals in compartment R_m and I_l can return to the susceptible class S at a rate γ_m and γ_l, respectively. The total ammount of food production occurs at a rate $p_f F$. Uncontaminated foods get contaminated at a rate $\lambda_f = B + \alpha_4 F_c$, where α_4 represents the relative contamination potential of compartment F_c . The interactions and transmission pathways between all compartments are summarized by the flowchart of FIGURE 1.

Making the input-output balance from model diagram in FIGURE 1 leads to the following system of nonlinear differential equations:

$$\frac{dS}{dt} = \Lambda + \gamma_m R_m + \gamma_l I_l - (\mu + \lambda_m + \lambda_l)S, \tag{2.1a}$$

$$\frac{dI_m}{dt} = \lambda_m S - (\mu + \delta_m + \sigma_m + \lambda_l)I_m, \tag{2.1b}$$

$$\frac{dR_m}{dt} = \sigma_m I_m + \sigma_{lm} I_{lm} - (\mu + \gamma_m)R_m, \tag{2.1c}$$

$$\frac{dI_l}{dt} = \lambda_l S - (\mu + \delta_l + \gamma_l + \lambda_m)I_l, \tag{2.1d}$$

$$\frac{dI_{lm}}{dt} = \lambda_l I_m + \lambda_m I_l - (\mu + \sigma_{lm} + \delta_{lm})I_{lm}, \tag{2.1e}$$

$$\frac{dB}{dt} = \xi_l I_l + \xi_{lm} I_{lm} - d_B B, \tag{2.1f}$$

$$\frac{dF_u}{dt} = p_f F - (\lambda_f + p_f)F_u, \tag{2.1g}$$

$$\frac{dF_c}{dt} = \lambda_f F_u - p_f F_c, \tag{2.1h}$$

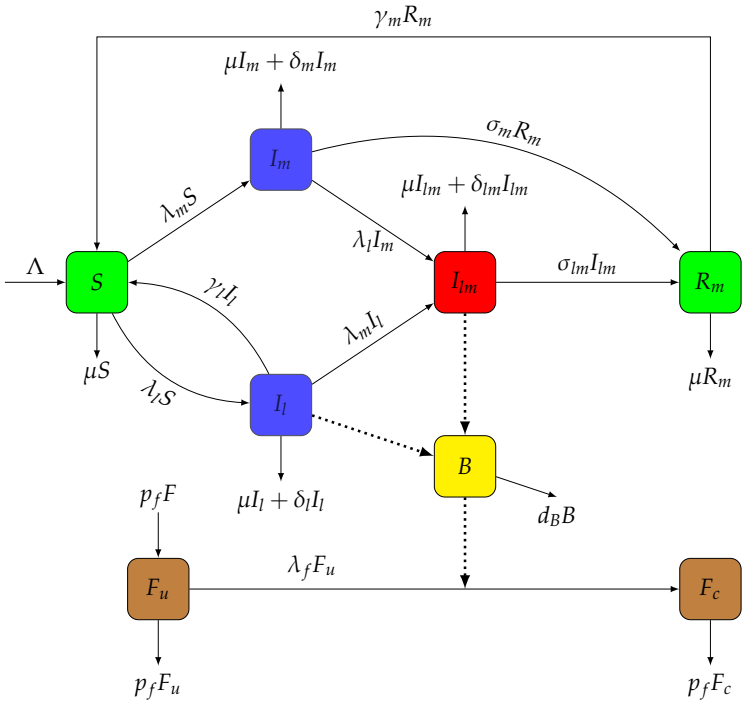

FIGURE 1. Diagram representing listeriosis/bacterial meningitis co-infection dynamics with the consideration of ready-to-eat food products.

with the following initial conditions

$$S(0) > 0, I_m(0) > 0, R_m(0) > 0, I_l(0) > 0, I_{lm}(0) > 0, B(0) > 0, F_u(0) > 0, F_c(0) > 0.$$

The different parameters of model (2.1) are defined with its values according to extinction and the persistence of two diseases in TABLE 1.

2.2. **Well-posedness results.** Let $X = (S, I_m, R_m, I_l, I_{lm}, B, F_u, F_c)^T$ and function f be the right-hand side of co-infection model (2.1). Consider the following Cauchy's problem

$$\begin{cases} \dfrac{dX(t)}{dt} = \mathbb{H}(t, X(t)), \\ (t_0, X_0) \in \mathbb{R}_+ \times \mathbb{R}^8_+. \end{cases} \tag{2.2}$$

Theorem 2.1. *The Cauchy problem* (2.2) *admits a unique maximal solution with respect to the initial condition* $(t_0, X_0) \in \mathbb{R}_+ \times \mathbb{R}^8_+$.

Proof. To prove the existence and uniqueness of solution for model (2.1), we use a similar method as in [12, 28]. Therefore, we consider the vector field of the proposed model $\mathbb{H} : (-\infty, \infty) \times \mathbb{R}^8 \to \mathbb{R}^8$

Parameters	Extinction	Persistence	References
Λ	50	50	Assumed
μ	0.092	0.092	Assumed
β_m	0.000145	0.0005	[6,24]
β_l	0.0805	0.15	[10,11]
δ_m	0.014	0.014	[6,11]
δ_l	0.9	0.9	[10,11]
δ_{lm}	0.0004	0.04	[10,11]
γ_l	0.0009	0.0009	[10,11]
γ_m	0.0061	0.0061	[6,10]
σ_m	0.007	0.007	[6,10]
σ_{lm}	0.09	0.09	[10,11]
d_B	0.52	0.52	[10,11]
ξ_l	0.007	0.009	[10,11]
ξ_{lm}	0.1	0.0074	[10,11]
p_f	0.08	0.08	[10,11]
α_1	0.00091	0.0091	[10,11]
α_2	0.00080	0.70	Assumed
α_3	0.0003	0.0003	Assumed
α_4	0.0006	0.6	Assumed

TABLE 1. Parameters of listeriosis/bacterial co-infection dynamics

as

$$\mathbb{H}(t,X)=\begin{pmatrix}\Lambda+\gamma_m R_m+\gamma_l I_l-k_1 S\\ \lambda_m S-(k_2+\lambda_l)I_m\\ \sigma_m I_m+\sigma_{lm}I_{lm}-k_3 R_m\\ \lambda_l S-(k_4+\lambda_m)I_l\\ \lambda_l I_m+\lambda_m I_l-k_5 I_{lm}\\ \xi_l I_l+\xi_{lm}I_{lm}-d_B B\\ p_f F-k_6 F_u\\ \lambda_f F_u-p_f F_c\end{pmatrix}, \tag{2.3}$$

where

$$k_1=\mu+\lambda_m+\lambda_l, k_2=\mu+\delta_m+\sigma_m, k_3=\mu+\gamma_m,$$
$$k_4=\mu+\delta_l+\gamma_l, k_5=\mu+\sigma_{lm}+\delta_{lm} \text{ and } k_6=\lambda_f+p_f.$$

The right-hand side of the equation (2.3) implies that the function $\mathbb{H}$ is continuous and therefore ensures the existence of solution $(S, I_m, R_m, I_l, I_{lm}, B, F_u, F_c)$ within the interval $[0,\infty)$. In addition, calculating the derivative of $\mathbb{H}$ with respect to the model state variables gives the Jacobian matrix

as:

$$D\mathbb{H} = \begin{pmatrix} -k_1 & -\beta_m S & \gamma_m & \gamma_l & -\beta_m\alpha_1 S & -(\beta_m\alpha_2+\beta_l)S & 0 & -\beta_l\alpha_3 S \\ \lambda_m & \beta_m S-(k_2+\lambda_l) & 0 & 0 & \beta_m\alpha_1 S & \beta_m\alpha_2 S-\beta_l I_m & 0 & -\beta_l\alpha_3 I_m \\ 0 & \sigma_m & -k_3 & 0 & \sigma_{lm} & 0 & 0 & 0 \\ \lambda_l & -\beta_m I_l & 0 & -(k_4+\lambda_m) & -\beta_m\alpha_1 I_l & \beta_l S-\beta_m\alpha_2 I_l & 0 & \beta_l\alpha_3 S \\ 0 & \beta_m I_l+\lambda_l & 0 & \lambda_m & -k_5 & \beta_m\alpha_2 I_l+\beta_l I_m & 0 & \beta_l\alpha_3 I_m \\ 0 & 0 & 0 & \xi_l & \xi_{lm} & -d_B & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -F_u & -k_6\ p_f-\alpha_4 F_u & \\ 0 & 0 & 0 & 0 & 0 & F_u & k_6\ \alpha_4 F_u-p_f & \end{pmatrix} \tag{2.4}$$

Since $D\mathbb{H}$ is continuous over $\mathbb{R}^8$ and thus $\mathbb{H}$ is locally Lipschitz continuous on $(-\infty,\infty)\times\mathbb{R}^8$, therefore, the model solution $(S, I_m, R_m, I_l, I_{lm}, B, F_u, F_c)$ is uniquely determined on the interval $[0,\infty)$. □

Proposition 2.1. *The unique solution of co-infection model* (2.1) *is bounded and dwell in the following bassin*

$$\mathcal{D} = \Bigg\{((S, I_m, R_m, I_l, I_{lm}), B, (F_u, F_c)) \in \mathbb{R}^5_+ \times \mathbb{R}_+ \times \mathbb{R}^2_+ | S + I_m + R_m + I_l + I_{lm} \leq \frac{\Lambda}{\mu}, \\ B \leq \frac{(\xi_l+\xi_{lm})\Lambda}{\mu d_B}, F_u + F_c = F\Bigg\}. \tag{2.5}$$

Proof. Let $Y = (S, I_m, R_m, I_l, I_{lm}, B, F_u, F_c)^{\mathcal{T}}$. Then, model (2.1) can be written as follows:

$$\frac{dY}{dt} = MY + M_1, \tag{2.6}$$

where

$$M = \begin{pmatrix} -k_1 & 0 & \gamma_m & \gamma_l & 0 & 0 & 0 & 0 \\ \lambda_m & -(k_2+\lambda_l) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_m & -k_3 & 0 & \sigma_{lm} & 0 & 0 & 0 \\ \lambda_l & 0 & 0 & -(k_4+\lambda_m) & 0 & 0 & 0 & 0 \\ 0 & \lambda_l & 0 & \lambda_m & -k_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi_l & \xi_{lm} & -d_B & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\lambda_f & p_f \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda_f & -p_f \end{pmatrix}$$

and

$$M_1 = (\Lambda, 0, 0, 0, 0, 0, 0, 0)^{\mathcal{T}}.$$

To continue, we have:

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_l I_l - \delta_m I_m - \delta_{lm} I_{lm}, \tag{2.7a}$$

$$\frac{dB}{dt} = \xi_l I_l + \xi_{lm} I_{lm} - d_B B, \tag{2.7b}$$

$$\frac{dF}{dt} = 0. \tag{2.7c}$$

Considering equation (2.7a), we have:

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

Let M_2, be the only solution of

$$\begin{cases} \dfrac{dM_2}{dt} = \Lambda - \mu M_2, \\ \\ M_2(0) = N(0). \end{cases}$$

Therefore,

$$M_2 = \frac{\Lambda}{\mu} + \left(M_2(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t},$$

then

$$N \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t}.$$

As t tends to $+\infty$,

$$N \leq \frac{\Lambda}{\mu},$$

hence

$$S + I_m + R_m + I_l + I_{lm} \leq \frac{\Lambda}{\mu}.$$

Furthermore, equation (2.7b) gives

$$\frac{dB}{dt} \leq -d_B B + \frac{(\xi_l + \xi_{lm})\Lambda}{\mu},$$

thus

$$B \leq \frac{(\xi_l + \xi_{lm})\Lambda}{\mu d_B}.$$

□

Proposition 2.2. *The positivity of the initial conditions entails the positivitiy of $S(t)$, $I_m(t)$, $R_m(t)$, $I_l(t)$, $I_{lm}(t)$, $B(t)$, $F_u(t)$ and $F_c(t)$ for all time $t > 0$.*

Proof. To establish the positivity of the solutions, we use Duhamel's formula. So, using equation (2.1b) and employing the method of integration factor and change of variable, yields [15]

$$I_m(t) = e^{-(\mu+\delta_m+\sigma_m+\lambda_l)t}\left[I_m(0) + \lambda_m \int_0^t e^{(\mu+\delta_m+\sigma_m+\lambda_l)s} S(s)ds\right] > 0.$$

Using equations (2.1c)-(2.1h) and the same process as previously, we obtain:

$$R_m(t) = e^{-(\mu+\gamma_m)t}\left[R_m(0) + \int_0^t e^{(\mu+\gamma_m)s}(\sigma_m I_m(s) + \sigma_{lm} I_{lm}(s))ds\right] > 0,$$

$$I_l(t) = e^{-(\mu+\delta_l+\gamma_l+\lambda_m)t}\left[I_l(0) + \lambda_l \int_0^t e^{(\mu+\delta_l+\gamma_l+\lambda_m)s} S(s)ds\right] > 0,$$

$$I_{lm}(t) = e^{-(\mu+\sigma_{lm}+\delta_{lm})t}\left[I_{lm}(0) + \int_0^t e^{(\mu+\sigma_{lm}+\delta_{lm})s}(\lambda_l I_m(s) + \lambda_m I_l(s))ds\right] > 0,$$

$$B(t) = e^{-d_B t}\left[B(0) + \int_0^t e^{d_B s}(\xi_l I_l(s) + \xi_{lm} I_{lm}(s))ds\right] > 0,$$

$$F_u(t) = e^{-(\lambda_f+p_f)t}\left[F_u(0) + p_f \int_0^t e^{(\lambda_f+p_f)s} F(s)ds\right] > 0,$$

$$F_c(t) = e^{-p_f t}\left[F_c(0) + \lambda_f \int_0^t e^{p_f s} F_u(s)ds\right] > 0.$$

Thanks to equation (2.1a) and considering $I_l(t) > 0$ and $R_m(t) > 0$, we get:

$$\frac{dS(t)}{dt} \geq -(\mu + \lambda_m + \lambda_l)S(t),$$

hence

$$S(t) \geq e^{-(\mu+\lambda_m+\lambda_l)t} S(0) > 0 \text{ for all } t > 0.$$

This shows that $S(t), I_m(t), R_m(t), I_l(t), I_{lm}(t), B(t), F_u(t)$ and $F_c(t)$ are positive. □

Now, we investigate the single infection dynamics model of each disease.

3. Bacterial meningitis-only model

Setting $I_l = I_{lm} = B = F_u = F_c = 0$ in co-infection model (2.1), we have the following bacterial meningitis-only model (3.1) given by:

$$\frac{dS}{dt} = \Lambda + \gamma_m R_m - (\mu + \lambda_m)S, \tag{3.1a}$$

$$\frac{dI_m}{dt} = \lambda_m S - (\mu + \delta_m + \sigma_m)I_m, \tag{3.1b}$$

$$\frac{dR_m}{dt} = \sigma_m I_m - (\mu + \gamma_m)R_m, \tag{3.1c}$$

where

$$N = S + I_m + R_m \text{ and } \lambda_m = \beta_m I_m.$$

Bacterial meningitis-only model disease-free steady-state is given by $E_m^0 = (\frac{\Lambda}{\mu}, 0, 0)$ and thanks to the next generation matrix approach in [25], we determine its basic reproduction number $(\mathcal{R}_0^M)$ as follows:

$$\mathcal{R}_0^M = \frac{\beta_m \Lambda}{\mu(\mu + \delta_m + \sigma_m)}. \tag{3.2}$$

However, Theorem 2 in Driessche and Watmough [25], quarantees the following result:

Proposition 3.1. *The disease-free equilibrium of the bacterial meningitis-only-model is locally asymptotically stable for $\mathcal{R}_0^M < 1$ and unstable when $\mathcal{R}_0^M > 1$.*

Proposition 3.2. *If $\mathcal{R}_0^M > 1$ then bacterial meningitis-only model admits a unique endemic equilibrium $E_m^* = (S^*, I_m^*, R_m^*)$.*

Proof. To compute the components of E_m^*, we set the right-hand bacterial meningitis-only model (3.1) equals to zero. Therefore, from equations (3.1b) and (3.1c), we obtain

$$S^* = \frac{\Lambda}{\mu \mathcal{R}_0^M}, \tag{3.3}$$

$$R_m^* = \frac{\sigma_m}{\mu + \gamma_m} I_m^*. \tag{3.4}$$

Substituting (3.3) and (3.4) in equation (3.1a), we obtain:

$$I_m^* = \frac{\mu\Lambda(\mu + \gamma_m)}{\gamma_m \sigma_m \mu \mathcal{R}_0^M + \beta_m \Lambda(\mu + \gamma_m)} (\mathcal{R}_0^M - 1). \tag{3.5}$$

We observe that $\mathcal{R}_0^M = 1$ leads to the disease-free equilibrium E_m^0, whereas when $\mathcal{R}_0^M > 1$ then there exists a unique endemic equilibrium E_m^*. □

3.1. **Endemic equilibrium and its stability analysis.**

Theorem 3.1. *If $\mathcal{R}_0^M > 1$ then the endemic equilibrium E_m^* of bacterial meningitis-only model (3.1) is locally asymptotically stable and unstable otherwise.*

Proof. The Jacobian matrix at the endemic equilibrium point E_m^* reads as

$$J(E_m^*) = \begin{pmatrix} -(\mu + \beta_m I_m^*) & -\beta_m S^* & \gamma_m \\ \beta_m I_m^* & \beta_m S^* - (\mu + \delta_m + \sigma_m) & 0 \\ 0 & \sigma_m & -(\mu + \gamma_m) \end{pmatrix} \tag{3.6}$$

and the associated characteristic polynomial is:

$$P(\lambda) = a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0,$$

where

$$
\begin{aligned}
a_3 &= 1 > 0 \\
a_2 &= \frac{\beta_m \mu \Lambda(\mu+\gamma_m)(\mathcal{R}_0^M - 1)}{\gamma_m \sigma_m \mu \mathcal{R}_0^M + \beta_m \Lambda(\mu+\gamma_m)} + 2\mu + \gamma_m > 0 \\
a_1 &= \mu(\mu+\gamma_m) + \frac{\mu\Lambda\beta_m(\mu+\gamma_m)^3(\mathcal{R}_0^M - 1)}{\gamma_m \sigma_m \mu \mathcal{R}_0^M + \beta_m \Lambda(\mu+\gamma_m)} > 0 \\
a_0 &= \frac{\mu\Lambda\beta_m(\mu+\gamma_m)(\mathcal{R}_0^M - 1)}{\gamma_m \sigma_m \mu \mathcal{R}_0^M + \beta_m \Lambda(\mu+\gamma_m)} \left[\mu\sigma_m + (\mu+\gamma_m)(\mu+\delta_m)\right] > 0
\end{aligned}
$$

Futhermore,

$$
\begin{aligned}
a_2 a_1 - a_0 = (\beta_m I_m^*)^2(2\mu+\gamma_m) + \mu^2(2\beta_m I_m^* + \mu + 2\delta_m + 2\gamma_m) \\
+ \beta_m I_m^*(\mu\gamma_m + \mu\delta_m + \gamma_m^2) + \mu^2\gamma_m + \mu\gamma_m\delta_m > 0.
\end{aligned}
$$

Therefore, all the necessary criteria for Routh-Hurwitz stability are satisfied. So, endemic equilibrium E_m^* of bacterial meningitis-only model (3.1) is locally asymptotically stable if $\mathcal{R}_0^M > 1$. □

Theorem 3.2. *The endemic equilibrium E_m^* of bacterial meningitis-only model (3.1) is globally asymptotically stable if $\mathcal{R}_0^M > 1$.*

Proof. Consider Lyapounov function as following:

$$
\begin{aligned}
\mathcal{V} = \frac{1}{2}\left[(S - S^*) + (I_m - I_m^*) + (R_m - R_m^*)\right]^2 + \frac{2\mu+\delta_m}{\beta_m}\left(I_m - I_m^* - I_m^* \ln\left(\frac{I_m}{I_m^*}\right)\right) \\
+ \frac{2\mu+\delta_m}{2\sigma_m}(R_m - R_m^*)^2.
\end{aligned}
$$

It is easy to see that $\mathcal{V}(S^*, I_m^*, R_m^*) = 0$ and positive elsewhere. Furthermore, its time derivative is given by

$$
\begin{aligned}
\frac{d\mathcal{V}}{dt} = \left[(S - S^*) + (I_m - I_m^*) + (R_m - R_m^*)\right](\Lambda - \mu(S + I_m + R_m) - \delta_m I_m) \\
+ \frac{2\mu+\delta_m}{\beta_m}(I_m - I_m^*)\left[\beta_m S - (\mu + \delta_m + \sigma_m)\right] + \frac{2\mu+\delta_m}{\sigma_m}(R_m - R_m^*)(\sigma_m I_m - (\mu+\gamma_m)R_m).
\end{aligned}
$$

At the equilibrium,

$$
\begin{aligned}
\Lambda &= \mu(S^* + I_m^* + R_m^*) + \delta_m I_m^*, \\
\beta_m S^* &= (\mu + \delta_m + \sigma_m), \\
0 &= -\sigma_m I_m^* + (\mu+\gamma_m)R_m^*.
\end{aligned}
$$

Parameters	Formula	Sensitivity indices
Λ	-	1
β_m	-	1
μ	$\mathcal{K}_{\mu}^{\mathcal{R}_0^M} = -\dfrac{2\mu+\delta_m+\sigma_m}{\mu+\delta_m+\sigma_m}$	-1.807
δ_m	$\mathcal{K}_{\delta_m}^{\mathcal{R}_0^M} = -\dfrac{\delta_m}{\mu+\delta_m+\sigma_m}$	-0.122
σ_m	$\mathcal{K}_{\sigma_m}^{\mathcal{R}_0^M} = -\dfrac{\sigma_m}{\mu+\delta_m+\sigma_m}$	-0.07

TABLE 2. Indices of local sensitivity for $\mathcal{R}_0^M$ parameters.

So,

$$\begin{aligned}
\frac{d\mathcal{V}}{dt} &= [(S-S^*)+(I_m-I_m^*)+(R_m-R_m^*)]\,[\mu(S^*+I_m^*+R_m^*)+\delta_m I_m^*-\mu(S+I_m+R_m)-\delta_m I_m] \\
&+ \frac{2\mu+\delta_m}{\beta_m}\,(I_m-I_m^*)\,[\beta_m S-(\mu+\delta_m+\sigma_m)] \\
&+ \frac{2\mu+\delta_m}{\sigma_m}(R_m-R_m^*)\,[\sigma_m I_m-(\mu+\gamma_m)R_m+-\sigma_m I_m^*+(\mu+\gamma_m)R_m^*] \\
&= \{[(S-S^*)+(R_m-R_m^*)]+(I_m-I_m^*)\}\{-\mu[(S-S^*)+(R_m-R_m^*)]-(\mu+\delta_m)(I_m-I_m^*)\} \\
&+ (2\mu+\delta_m)\,(I_m-I_m^*)\,(S-S^*) \\
&+ \frac{2\mu+\delta_m}{\sigma_m}\,[\sigma_m(I_m-I_m^*)-(\mu+\gamma_m)(R_m-R_m^*)]\,(R_m-R_m^*) \\
&= -\mu\,[(S-S^*)+(R_m-R_m^*)]^2-(\mu+\delta_m)\,(I_m-I_m^*)^2-\frac{(2\mu+\delta_m)(\mu+\gamma_m)}{\sigma_m}(R_m-R_m^*)^2 \le 0.
\end{aligned}$$

Consequently, $\dfrac{d\mathcal{V}(S,I_m,R_m)}{dt} \le 0$ and $\left\{(S,I_m,R_m)\in\mathcal{D}, \dfrac{d\mathcal{V}(S,I_m,R_m)}{dt}=0\right\} = \{(S^*,I_m^*,R_m^*)\}$. Thanks to LaSalle's invariance principle, E_m^* is globally asymptotically stable in $\mathcal{D}$, [17]. □

3.2. **Local sensitivity analysis of $\mathcal{R}_0^M$.** During modeling study, a local sensitivity analysis is used to identify among the model parameters that appear within the determined threshold dynamics parameters those that are most influential, [9]. It consists in calculating the normalized sensitivity index of each parameter, [3]. So, the sensitivity index of $\mathcal{R}_0^M$ with respect to the parameter p is given by:

$$\mathcal{K}_p^{\mathcal{R}_0^M} = \frac{\partial \mathcal{R}_0^M}{\partial p}\times\frac{p}{\mathcal{R}_0^M}.$$

The local sensitivity indices with respect to the parameters of model (3.1) are given in TABLE 2 and illustrated by FIGURE 2. We note in this part that the parameters that most influence $\mathcal{R}_0^M$ are Λ, β_m, μ and δ_m. $\mathcal{K}_{\beta_m}^{\mathcal{R}_0^M}=1$, this meaning that 1% increase in β_m will produce 1% increase in $\mathcal{R}_0^M$ and

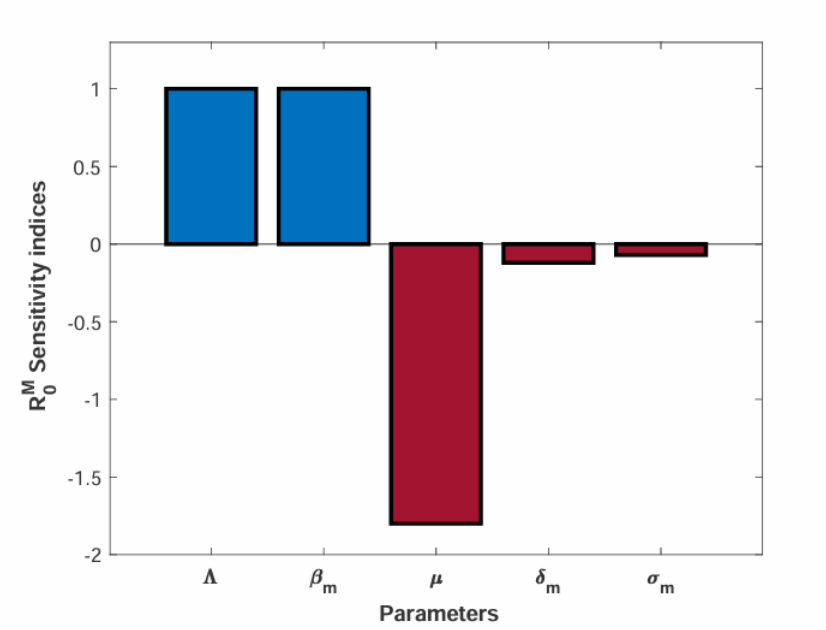

FIGURE 2. Local sensitivity analysis diagram of $\mathcal{R}_0^M$.

$\mathcal{K}_\mu^{\mathcal{R}_0^M} = -1.807$, meaning that 1% increase in μ will produce 1.807% decrease in $\mathcal{R}_0^M$. Furthermore, similar interpretation can be done for the other parameters appearing in $\mathcal{R}_0^M$.

4. LISTERIOSIS-ONLY MODEL

Listeriosis-only model (4.1) in model (2.1) is obtained by setting $I_m = R_m = I_{lm} = 0$.

$$\frac{dS}{dt} = \Lambda + \gamma_l I_l - (\mu + \lambda_l)S, \tag{4.1a}$$

$$\frac{dI_l}{dt} = \lambda_l S - (\mu + \delta_l + \gamma_l)I_l, \tag{4.1b}$$

$$\frac{dB}{dt} = \xi_l I_l - d_B B, \tag{4.1c}$$

$$\frac{dF_u}{dt} = p_f F - (\lambda_f + p_f)F_u, \tag{4.1d}$$

$$\frac{dF_c}{dt} = \lambda_f F_u - p_f F_c. \tag{4.1e}$$

In this section, $N = S + I_l$, $\lambda_l = \beta_l(B + \alpha_3 F_c)$ and $\lambda_f = B + \alpha_4 F_c$.

Disease-free equilibrium for the listeriosis-only model is given by $E_l^0 = (\frac{\Lambda}{\mu}, 0, 0, 0, 0)$ and the basic reproduction number ($\mathcal{R}_0^L$) as:

$$\mathcal{R}_0^L = \frac{\beta_l \xi_l \Lambda}{\mu d_B(\mu + \delta_l + \gamma_l)}. \tag{4.2}$$

Therefore, thanks to Theorem 2 in Driessche and Watmough [25], the following results holds:

Proposition 4.1. *Disease-free equilibrium is locally asymptotically stable for $\mathcal{R}_0^L < 1$ and unstable when $\mathcal{R}_0^L > 1$.*

Proposition 4.2. *Listeriosis-only model has a unique endemic equilibrium $E_l^* = (S^*, I_l^*, B^*, F_u^*, F_c^*)$ if and only if $\mathcal{R}_0^L > 1$.*

Proof. Endemic equilibrium for the listeriosis-only model (4.1) is obtained by setting the right hand side of listeriosis-only model (4.1) to zero.

From equations (4.1b), (4.1c) and (4.1e),

$$S^* = \frac{d_B(\mu + \delta_l + \gamma_l)B^*}{\beta_l \xi_l (B^* + \alpha_3 F_c^*)} \tag{4.3}$$

$$B^* = \frac{\xi_l}{d_B} I_l^* \tag{4.4}$$

$$F_u^* = \frac{p_f F_c^*}{B^* + \alpha_4 F_c^*} \tag{4.5}$$

Substituting (4.3), (4.4) and (4.5) in equation (4.1a), we obtain:

$$I_l^* = \frac{\mu d_B(\mu + \delta_l + \gamma_l)}{\beta_l(\mu + \delta_l)\xi_l}\left[-\frac{\beta_l(\mu + \delta_l)\alpha_3 F_c^*}{\mu(\mu + \delta_l + \gamma_l)} - 1 + \mathcal{R}_0^L\right] + \frac{d_B \mathcal{Q}}{\beta_l(\mu + \delta_l)\xi_l}, \tag{4.6}$$

with

$$\mathcal{Q} = \sqrt{\left(\beta_l(\mu + \delta_l)\alpha_3 F_c^* + \mu(\mu + \delta_l + \gamma_l) - \beta_l \frac{\Lambda \xi_l}{d_B}\right)^2 + \frac{4}{d_B}\beta_l(\mu + \delta_l)\beta_l \Lambda \xi_l \alpha_3 F_c^*}.$$

So, $I_l^* > 0$ if and only if $\mathcal{R}_0^L > \dfrac{\beta_l(\mu + \delta_l)\alpha_3 F_c^*}{\mu(\mu + \delta_l + \gamma_l)} + 1$. □

4.1. **Existence of backward and forward bifurcations.** The bifucation analysis of listeriosis-only model (4.1) can be done by applying the central manifold theory, [7]. Let $x_1 = S$, $x_2 = I_l$, $x_3 = B$, $x_4 = F_u$, $x_5 = F_c$ and $x = (x_1, x_2, x_3, x_4, x_5)^{\mathcal{T}}$. It is possible to rewrite the listeriosis-only model (4.1) as follows:

$$\frac{dx}{dt} = f(x), \tag{4.7}$$

where $f = (f_1, f_2, f_3, f_4, f_5)^{\mathcal{T}}$. We find $\xi_l = \xi^*$ as a bifurcation parameter for $\mathcal{R}_0^L = 1$ where

$$\xi^* = \frac{\mu d_B}{\beta_l \Lambda}(\mu + \delta_l + \gamma_l).$$

Linearizing the system (4.7) at the disease-free equilibrium E_l^0 and with bifurcation parameter ξ^* gives:

$$J(E_l^0) = \begin{pmatrix} -\mu & \gamma_l & -\frac{\beta_l \Lambda}{\mu} & 0 & -\frac{\beta_l \alpha_3 \Lambda}{\mu} \\ 0 & -(\mu + \delta_l + \gamma_l) & \frac{\beta_l \Lambda}{\mu} & 0 & \frac{\beta_l \alpha_3 \Lambda}{\mu} \\ 0 & \xi^* & -d_B & 0 & 0 \\ 0 & 0 & 0 & 0 & p_f \\ 0 & 0 & 0 & 0 & -p_f \end{pmatrix}. \tag{4.8}$$

The Jacobian $J(E_l^0)$ when $\xi_l = \xi^*$ has a simple zero eigenvalue and real part each of the other eigenvalues is negative. So, with Central Manifold theory, we have the right eigenvector given by $u = (u_1, u_2, u_3, u_4, u_5)^{\mathcal{T}}$ where:

$$u_1 = \frac{-d_B}{\mu \xi^*}(\mu + \delta_l) u_3,\ u_2 = \frac{d_B}{\xi^*} u_3,\ u_3 \geq 0,\ u_4 = 0,\ u_5 = 0,$$

and the left eigenvector is given by $v = (v_1, v_2, v_3, v_4, v_5)$ where:

$$v_1 = 0,\ v_2 \geq 0,\ v_3 = \frac{1}{\xi^*}(\mu + \delta_l + \gamma_l) v_2,\ v_4 \geq 0,\ v_5 = \frac{\beta_l \Lambda \alpha_3}{\mu p_f} v_2 + v_4.$$

Using the expressions

$$\mathscr{A}_1 = \sum_{k;i;j=1}^{5} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_l^0, \xi^*)$$

and

$$\mathscr{B}_1 = \sum_{k;i=1}^{5} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \xi^*}(E_l^0, \xi^*),$$

we obtain

$$\mathscr{A}_1 = -2 v_2 u_3^2 \left(\frac{d_B(\mu + \delta_l)}{\mu \xi^*} \right) < 0$$

and $\mathscr{B}_1 = v_3 u_2 > 0$.

Hence, the following theorem holds.

Theorem 4.1. *If $\mathcal{R}_0^L = 1$, listeriosis-only model undergoes a forward bifurcation.*

An example of the forward bifurcation direction is given in FIGURE 3 below: The signs of $\mathscr{A}_1$ and $\mathscr{B}_1$ as well as the bifurcation parameters allow us to make an interpretation of the stability of the endemic equilibrium. As $\mathscr{A}_1 < 0$ and $\mathscr{B}_1 > 0$, endemic equilibrium E_l^* is locally asymptotically stable to close $\mathcal{R}_0^L = 1$, [14].

The results can be summarized in the following lemma.

Lemma 4.1. *Endemic equilibrium E_l^* is locally asymptotically stable when $\mathcal{R}_0^L > 1$ but only if $\mathcal{R}_0^L$ is close to 1.*

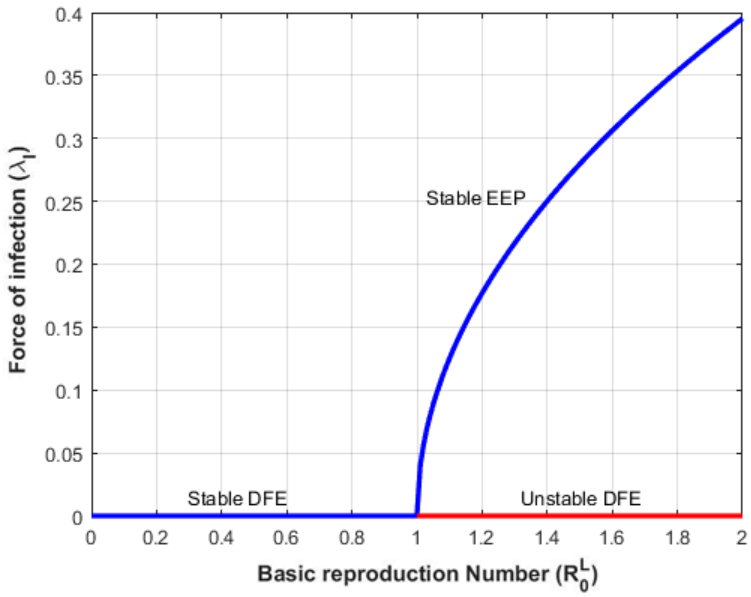

FIGURE 3. Direction of forward bifurcation.

4.2. **Local sensitivity analysis of $\mathcal{R}_0^L$.** Local sensitivity analysis in a model is often used to select the parameters to be estimated among those identified as the most influential, [9]. Of course, this study only concerns the parameters which appear in the expression of this threshold parameter. The local sensitivity analysis that we carry out here allows us to evaluate the effect of a parameter on the basic reproduction number while keeping the other parameters as constant. The method consists in calculating the sensitivity index of each parameter, [3]. So, the sensitivity index of $\mathcal{R}_0^L$ with respect to Λ is given by:

$$\mathcal{K}_{\omega}^{\mathcal{R}_0^L} = \frac{\partial \mathcal{R}_0^L}{\partial \omega} \times \frac{\omega}{\mathcal{R}_0^L}.$$

We obtain the sensitivity indices giving in the TABLE 3 and FIGURE 8.

We note in this part that the parameters that most influence $\mathcal{R}_0^L$ are Λ, β_l, ξ_l, μ, d_B and *deltal*. $\mathcal{K}_{\beta_l}^{\mathcal{R}_0^L} = 1$, this meaning that 1% increase in β_l will produce 1% increase in $\mathcal{R}_0^L$ and $\mathcal{K}_{\mu}^{\mathcal{R}_0^L} = -1.092$, meaning that 1% increase in μ will produce 1.092% decrease in $\mathcal{R}_0^L$. Morover, similar interpretation can be done for the other parameters appearing in $\mathcal{R}_0^L$.

5. Mathematical analysis of the co-infection model

The disease-free equilibrium of listeriosis and bacterial meningitis co-infection model (2.1) denoted by E_{lm}^0 is given by:

$$E_{lm}^0 = \left(S^0, I_m^0, I_l^0, I_{lm}^0, R_m^0, B^0, F_u^0, F_c^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0, 0\right).$$

Parameters	Formula	Sensitivity indices
Λ	-	1
ξ_l	-	1
β_l	-	1
d_B	-	-1
μ	$\mathcal{K}_{\mu}^{\mathcal{R}_0^L} = -\dfrac{2\mu+\delta_l+\gamma_l}{\mu+\delta_l+\gamma_l}$	-1.092
δ_l	$\mathcal{K}_{\delta_l}^{\mathcal{R}_0^L} = \dfrac{-\delta_l}{\mu+\delta_l+\gamma_l}$	-0.907
γ_l	$\mathcal{K}_{\gamma_l}^{\mathcal{R}_0^L} = \dfrac{-\gamma_l}{\mu+\delta_l+\gamma_l}$	-0.001

TABLE 3. Parameters values and sensitivity indices for $\mathcal{R}_0^L$.

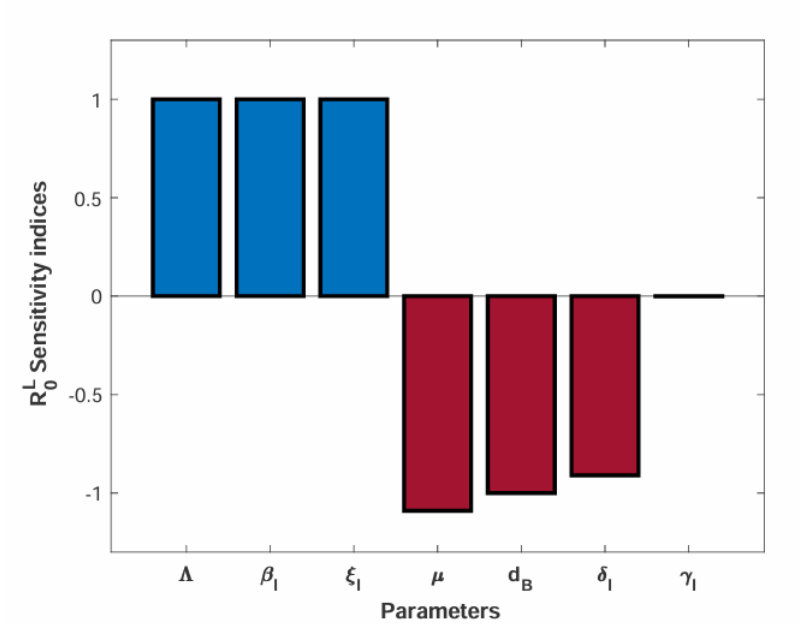

FIGURE 4. Local sensitivity analysis diagram of $\mathcal{R}_0^L$.

Using the next-generation matrix operator method on co-infection model (2.1), we have the basic reproduction number denoted by $\mathcal{R}_0^{LM}$ with

$$\mathcal{R}_0^{LM} = \max\left\{\mathcal{R}_0^L, \mathcal{R}_0^M\right\}, \tag{5.1}$$

where

$$\mathcal{R}_0^L = \frac{\beta_l \xi_l \Lambda}{\mu d_B(\mu+\delta_l+\gamma_l)} \text{ and } \mathcal{R}_0^M = \frac{\beta_m \Lambda}{\mu(\mu+\delta_m+\sigma_m)}.$$

Thus, thanks to Theorem 2 in Driessche and Watmough [25], we established the following result:

Proposition 5.1. *The disease-free equilibrium of co-infection model* (2.1) *is locally asymptotically stable (LAS) for* $\mathcal{R}_0^{LM} < 1$ *and unstable when* $\mathcal{R}_0^{LM} > 1$.

Proposition 5.2. *Co-infection model has a unique endemic equilibrium (EE) given by* $E_{lm}^* = (S^*, I_m^*, R_m^*, I_l^*, I_{lm}^*, B^*, F_u^*, F_c^*)$ *if and only if* $\mathcal{R}_0^{LM} > 1$.

Proof. Equating the derivatives of model (2.1) to zero, we can determine endemic equilibrium E_{lm}^*. Through (2.1b), we obtain:

$$S^* = \frac{\mu + \delta_m + \sigma_m + \lambda_l^*}{\lambda_m^*} I_m^*.$$

Through (2.1e), we have:

$$I_{lm}^* = \frac{\lambda_m^* I_l^* + \lambda_l^* I_m^*}{\mu + \sigma_{lm} + \delta_{lm}}.$$

Pulling B^* into (2.1f) and replacing I_{lm}^* with its value, we get:

$$B^* = \frac{\xi_l(\mu + \sigma_{lm} + \delta_{lm} + \lambda_m^*) I_l^* + \xi_l \lambda_l^* I_m^*}{d_B(\mu + \sigma_{lm} + \delta_{lm})}.$$

Following the same procedure as the previous one into (2.1c), we obtain:

$$R_m^* = \frac{\sigma_m(\mu + \sigma_{lm} + \delta_{lm}) + \sigma_{lm}\lambda_l^*}{(\mu + \gamma_m)(\mu + \sigma_{lm} + \delta_{lm})} I_m^* + \frac{\sigma_{lm}\lambda_m^* I_l^*}{(\mu + \gamma_m)(\mu + \sigma_{lm} + \delta_{lm})}.$$

Through the eighth equation, we obtain:

$$F_u^* = \frac{p_f F_c^*}{B^* + \alpha_4 F_c^*}.$$

□

5.1. **Global stability of E_{lm}^0 of co-infection model.** Model (2.1) can be rewritten as

$$\begin{cases} \dfrac{d\mathcal{X}}{dt} = \mathcal{F}(\mathcal{X}, Z) \\ \\ \dfrac{dZ}{dt} = \mathcal{G}(\mathcal{X}, Z), \quad \mathcal{G}(\mathcal{X}, 0) = 0, \end{cases}$$

where $\mathcal{X} = (S, R_m, F_u) \in \mathbb{R}^3$ and $Z = (I_m, I_l, I_{lm}, B, F_c) \in \mathbb{R}^5$. $\mathcal{X}$ denoting the number of uninfected individuals and foods and Z denoting the number of infected individuals and foods. The disease-free equilibrium of co-infection model (2.1) is denoted as:

$$U_0 = (\mathcal{X}_0, 0) \text{ with } \mathcal{X}_0 = (\frac{\Lambda}{\mu}, 0, 0).$$

The following hypothesis must be verified for $\mathcal{R}_0^{LM} < 1$:

(**H1**): For $\dfrac{d\mathcal{X}}{dt} = \mathcal{F}(\mathcal{X}, 0)$, U_0 is globally stable;

(**H2**): $\mathcal{G}(\mathcal{X}, Z) = AZ - \widehat{\mathcal{G}}(\mathcal{X}, Z)$, $\widehat{\mathcal{G}}(\mathcal{X}, Z) \geq 0$ for $(\mathcal{X}, Z) \in \mathcal{D}$, where $A = D_Z\mathcal{G}(U_0, 0)$ is a Metzler

matrix and $\mathcal{D}$ is a feasible region of the developed model. From model (2.1), we have:

$$\frac{d\mathcal{X}}{dt} = \mathcal{F}(\mathcal{X}, Z) = \begin{pmatrix} \Lambda + \gamma_m R_m + \gamma_l I_l - (\mu + \lambda_m + \lambda_l)S \\ \sigma_m I_m + \sigma_{lm} I_{lm} - (\mu + \gamma_m) R_m \\ p_f(F_u + F_c) - (\lambda_f + p_f) F_u \end{pmatrix},$$

hence

$$\mathcal{F}(\mathcal{X}, 0) = \begin{pmatrix} \Lambda - \mu S \\ 0 \\ 0 \end{pmatrix}.$$

$$\frac{dZ}{dt} = \mathcal{G}(\mathcal{X}, Z) = \begin{pmatrix} \lambda_m S - (\mu + \delta_m + \sigma_m + \lambda_l) I_m \\ \lambda_l S - (\mu + \delta_l + \gamma_l + \lambda_m) I_l \\ \lambda_l I_m + \lambda_m I_l - (\mu + \sigma_{lm} + \delta_{lm}) I_{lm} \\ \xi_l I_l + \xi_{lm} I_{lm} - d_B B \\ \lambda_f F_u - p_f F_c \end{pmatrix}.$$

Therefore,

$$A = D_Z \mathcal{G}(U_0, 0) = \begin{pmatrix} \beta_m S^0 - k_2 & 0 & \beta_m \alpha_1 S^0 & \beta_m \alpha_2 S^0 & 0 \\ 0 & -k_4 & 0 & \beta_l S^0 & \beta_l \alpha_3 S^0 \\ 0 & 0 & -k_5 & 0 & 0 \\ 0 & \xi_l & \xi_{lm} & -d_B & 0 \\ 0 & 0 & 0 & 0 & -p_f \end{pmatrix}.$$

We have $\beta_m S^0 - k_2 = k_2(\mathcal{R}_0^M - 1) < 0$ for all $\mathcal{R}_0^M < 1$. So A is a Metzler Matrix. Here,

$$\widehat{\mathcal{G}}(\mathcal{X}, Z) = AZ - \mathcal{G}(\mathcal{X}, Z),$$

and hence

$$\widehat{G}(\mathcal{X}, Z) = \begin{pmatrix} \widehat{G}_1(\mathcal{X}, Z) \\ \widehat{G}_2(\mathcal{X}, Z) \\ \widehat{G}_3(\mathcal{X}, Z) \\ \widehat{G}_4(\mathcal{X}, Z) \\ \widehat{G}_5(\mathcal{X}, Z) \end{pmatrix} = \begin{pmatrix} \lambda_m(S^0 - S) + \lambda_l I_m \\ \lambda_l(S^0 - S) + \lambda_m I_l \\ -\lambda_l I_m - \lambda_m I_l \\ 0 \\ -(B + \alpha_4 F_c) F_u \end{pmatrix}.$$

Hence $\widehat{G}_3(\mathcal{X}, Z) < 0$ and $\widehat{G}_5(\mathcal{X}, Z) < 0$, the necessary condition (**H2**) is not satisfied. Consequently U_0 and then disease free equilibrium E_{lm}^0 may not be globally asymptotic stable, [18]. However, there is a case of bifurcation which occurs at $\mathcal{R}_0^{LM} = 1$, [8].

5.2. **Existence of backward or forward bifurcations.** Bifurcation analysis of model (2.1) can be done by applying the central manifold theory. Let $y_1 = S$, $y_2 = I_m$, $y_3 = R_m$, $y_4 = I_l$, $y_5 = I_{lm}$, $y_6 = B$, $y_7 = F_u$, $y_8 = F_c$ and $y = (y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8)^{\mathcal{T}}$. So $\lambda_m = \beta_m(y_2 + \alpha_1 y_5 + \alpha_2 y_6)$, $\lambda_l = \beta_l(y_6 + \alpha_3 y_8)$ and $\lambda_f = y_6 + \alpha_4 y_8$. Co-infection model (2.1) reads as:

$$\frac{dy}{dt} = g(y), \tag{5.2}$$

where $g = (g_1, g_2, g_3, g_4, g_5, g_6, g_7, g_8)^{\mathcal{T}}$. We consider the case that $\mathcal{R}_0^M < \mathcal{R}_0^L = 1$, which amount to $\mathcal{R}_0^{LM} = \mathcal{R}_0^L = 1$. Consider now $\beta_l = \beta^*$ as a bifurcation parameter obtain from $\mathcal{R}_0^L = 1$ with

$$\beta^* = \frac{\mu d_B}{\xi_l \Lambda}(\mu + \delta_l + \gamma_l).$$

We linearize the system of equation (5.2) at the disease-free equilibrium E_{lm}^0 and with bifurcation parameter β^*. We obtain

$$J(E_{lm}^0) = \begin{pmatrix} -\mu & -\frac{\beta_m \Lambda}{\mu} & \gamma_m & \gamma_l & -\frac{\beta_m \alpha_1 \Lambda}{\mu} & -\frac{(\beta_m \alpha_2 + \beta^*)\Lambda}{\mu} & 0 & -\frac{\beta^* \alpha_3 \Lambda}{\mu} \\ 0 & \frac{\beta_m \Lambda}{\mu} - k_2 & 0 & 0 & \frac{\beta_m \alpha_1 \Lambda}{\mu} & \frac{\beta_m \alpha_2 \Lambda}{\mu} & 0 & 0 \\ 0 & \sigma_m & k_3 & 0 & \sigma_{lm} & 0 & 0 & 0 \\ 0 & 0 & 0 & k_4 & 0 & \frac{\beta^* \Lambda}{\mu} & 0 & \frac{\beta^* \alpha_3 \Lambda}{\mu} \\ 0 & 0 & 0 & 0 & k_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi_l & \xi_{lm} & -d_B & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & p_f \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -p_f \end{pmatrix}, \tag{5.3}$$

The Jacobian $J(E_{lm}^0)$ when $\beta_l = \beta^*$ has a simple zero eigenvalue and real part each of the other eigenvalues is negative. Thus, applying Central Manifold theory, the right eigenvector is given by $w = (w_1, mw_2, w_3, w_4, w_5, w_6, w_7, w_8)^{\mathcal{T}}$ where:

$$w_1 = \frac{1}{\mu}\left(\frac{\gamma_m \sigma_m}{\mu + \gamma_m} - \beta_m \frac{\Lambda}{\mu}\right) w_2 + \frac{1}{\mu}\left(\gamma_l + \frac{\xi_l \Lambda(\beta_m \alpha_2 + \beta^*)}{\mu d_B}\right) w_4,\ w_2 \geq 0,\ w_3 = \frac{\sigma_m}{\mu + \gamma_m} w_2,\ w_4 \geq 0,$$
$$w_5 = 0,\ w_6 = \frac{\xi_l}{d_B},\ w_7 \geq 0,\ w_8 = 0,$$

and left eigenvector given by $n = (n_1, n_2, n_3, n_4, n_5, n_6, n_7, n_8)$ where:

$$n_1 = n_2 = n_3 = 0,\ n_4 \geq 0,\ n_5 = \frac{\xi_{lm}(\mu + \delta_l + \gamma_l)}{\xi_l(\mu + \delta_{lm} + \sigma_{lm})} n_4,\ n_6 = \frac{1}{\xi_l}(\mu + \delta_l + \gamma_l) n_4,\ n_7 \geq 0,$$
$$n_8 = n_7 + \frac{\beta^* \Lambda \alpha_3}{\mu p_f} n_4.$$

Using the expressions

$$\mathscr{A}_2 = \sum_{k;i;j=1}^{8} n_k w_i w_j \frac{\partial^2 g_k}{\partial y_i \partial y_j}(E_{lm}^0, \beta^*)$$

and

$$\mathscr{B}_2 = \sum_{k;i=1}^{8} n_k w_i \frac{\partial^2 g_k}{\partial y_i \partial \beta^*}(E_{lm}^0, \beta^*),$$

we obtain

$$\mathscr{A}_2 = 2\beta^* w_1 w_6 n_4 + 2\beta_m w_2 w_4 n_5 + 2\beta^* w_2 w_6 n_5 + 2\beta_m \alpha_2 w_4 w_6 n_5 + 2 w_6 w_7 n_8$$
$$- (2\beta_m w_2 w_4 n_4 + 2\beta_m \alpha_2 w_4 w_6 n_4 + 2 w_6 w_7 n_7)$$

and $\mathscr{B}_2 = \dfrac{\Lambda}{\mu} n_4 w_6 > 0.$

Let

$$\mathbb{A}_1 = 2\beta^* w_1 w_6 n_4 + 2\beta_m w_2 w_4 n_5 + 2\beta^* w_2 w_6 n_5 + 2\beta_m \alpha_2 w_4 w_6 n_5 + 2 w_6 w_7 n_8,$$
$$\mathbb{A}_2 = 2\beta_m w_2 w_4 n_4 + 2\beta_m \alpha_2 w_4 w_6 n_4 + 2 w_6 w_7 n_7.$$

Hence, following result holds:

Theorem 5.1. *If $\mathcal{R}_0^{LM} = \mathcal{R}_0^L = 1$ then*

(i) *Listeriosis and bacterial meningitis co-infection model (2.1) undergoes a backward bifurcation whenever $\mathbb{A}_1 > \mathbb{A}_2$.*

(ii) *Listeriosis and bacterial meningitis co-infection model (2.1) undergoes a forward bifurcation whenever $\mathbb{A}_1 < \mathbb{A}_2$.*

5.3. **Competitive exclusion and coexistence analysis.** In this section will evaluate the conditions under which listeriosis and bacterial meningitis coexist or compete with each other, [2, 19]. To do this, we will study the impact of listeriosis on bacterial meningitis and vice versa. In addition, we will analyze the impact of the mortality rate of the bacterium Listeria monocytogenes on bacterial meningitis. As a reminder,

$$\mathcal{R}_0^L = \frac{\beta_l \xi_l \Lambda}{\mu d_B(\mu + \delta_l + \gamma_l)} \text{ and } \mathcal{R}_0^M = \frac{\beta_m \Lambda}{\mu(\mu + \delta_m + \sigma_m)}.$$

Let

$$D_1 = \beta_l \xi_l \Lambda, \quad D_2 = \delta_l + \gamma_l, \quad Q_1 = \beta_m \Lambda, \quad Q_2 = \delta_m + \sigma_m,$$
$$W = \sqrt{\frac{D_2^2}{4} + \frac{D_1}{d_B \mathcal{R}_0^L}} \quad \text{and} \quad Z = \sqrt{\frac{Q_2^2}{4} + \frac{Q_1}{\mathcal{R}_0^M}}.$$

5.3.1. *Impact of Listeriosis on Bacterial meningitis.* In this section, we evaluate the impact that any variation in infection due to listeriosis could have on bacterial meningitis. Let μ, the common parameter for $\mathcal{R}_0^M$ and $\mathcal{R}_0^L$. From $\mathcal{R}_0^L$, we get the quadratic equation in terms of μ:

$$\mu^2 + D_2 \mu - \frac{D_1}{d_B \mathcal{R}_0^L} = 0.$$

Using a conanical from, we have

$$\left(\mu + \frac{D_2}{2}\right)^2 - \left(\frac{D_2^2}{4} + \frac{D_1}{d_B \mathcal{R}_0^L}\right) = 0,$$

which gives μ in terms of $\mathcal{R}_0^L$ as follows:

$$\mu = \sqrt{\frac{D_2^2}{4} + \frac{D_1}{d_B \mathcal{R}_0^L}} - \frac{D_2}{2} > 0. \tag{5.4}$$

Thus,

$$\frac{\partial \mathcal{R}_0^M}{\partial \mathcal{R}_0^L} = \frac{\partial \mathcal{R}_0^M}{\partial \mu} \times \frac{\partial \mu}{\partial \mathcal{R}_0^L}.$$

So

$$\frac{\partial \mathcal{R}_0^M}{\partial \mathcal{R}_0^L} = \frac{2D_1\beta_m\Lambda(2\mu+\delta_m+\sigma_m)\sqrt{\frac{D_2^2}{4}+\frac{D_1}{d_B\mathcal{R}_0^L}}}{\mu^2\mathcal{R}_0^L(d_BD_2^2\mathcal{R}_0^L+4D_1)(\mu+\delta_m+\sigma_m)^2} > 0 \tag{5.5}$$

As (5.5) is strictly positive, it implies that an increase in listeriosis infection leads to an increase in bacterial meningitis.

To continue, we will analyze the impact of the variation in the mortality rate of the bacterium Listeria monocytogenes on bacterial meningitis disease. To do this, we will evaluate this impact by partially differentiating $\mathcal{R}_0^M$ from d_B. We have:

$$\frac{\partial \mathcal{R}_0^M}{\partial d_B} = \frac{D_1(-D_2+2W+Q_2)}{2d_B^2\mathcal{R}_0^LW(\mu^2-\mu Q)^2} \tag{5.6}$$

This result to:

$$\frac{\partial \mathcal{R}_0^M}{\partial d_B} < 0 \Rightarrow -D_2+2W+Q_2 < 0 \Rightarrow \mathcal{R}_0^L > \frac{4D_1}{d_B(D_2-Q_2)^2-d_BD_2^2}.$$

Let $\Phi = \frac{4D_1}{d_B(D_2-Q_2)^2-d_BD_2^2}$ then an increase of d_B when $\mathcal{R}_0^L > \Phi$ leads to a reduction in $\mathcal{R}_0^M$. Therefore, a massive elimination of listeriosis bacteria could indirectly reduce the number of cases infected with bacterial meningitis. If $\mathcal{R}_0^L$ is in $[0,\Phi[$ an increase in the mortality rate of listeriosis bacteria could lead to an increase in the basic reproduction number of bacterial meningitis. If $\mathcal{R}_0^L = \Phi$, eliminating listeriosis bacteria will have no impact on bacterial meningitis. These results are summarized in the following lemma.

Lemma 5.1. *The elimination of Listeria monocytogenes bacteria could impact bacterial meningitis as follows:*

(i) *a positive impact if* $\mathcal{R}_0^L > \Phi$,
(ii) *no impact if* $\mathcal{R}_0^L = \Phi$,
(iii) *a negative impact if* $\mathcal{R}_0^L$ *is in* $[0,\Phi[$.

5.3.2. *Impact of Bacterial on meningitis Listeriosis.* Similarly, the expression μ in terms of $\mathcal{R}_0^M$ is given by:

$$\mu = \sqrt{\frac{Q_2^2}{4}+\frac{Q_1}{\mathcal{R}_0^M}} - \frac{Q_2}{2} > 0 \tag{5.7}$$

Thus,

$$\frac{\partial \mathcal{R}_0^L}{\partial \mathcal{R}_0^M} = \frac{\partial \mathcal{R}_0^L}{\partial \mu} \times \frac{\partial \mu}{\partial \mathcal{R}_0^M}.$$

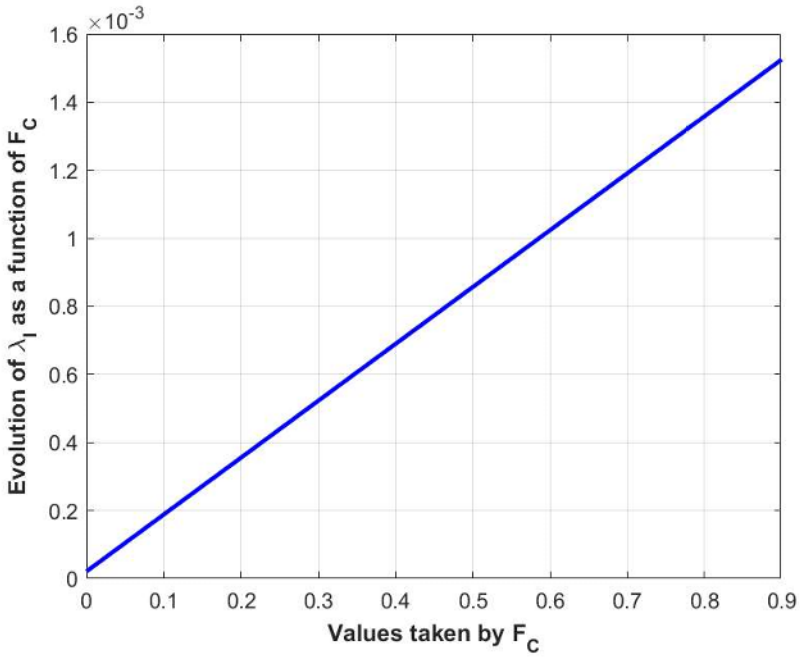

FIGURE 5. Evolution of listeriosis infection force λ_l with respect to contaminated food products F_C with $\beta_l = 0.0209$, $\alpha_3 = 0.08$ and $B = 0.001$

So

$$\frac{\partial \mathcal{R}_0^L}{\partial \mathcal{R}_0^M} = \frac{2Q_1\beta_l\xi_l\Lambda d_B(2\mu+\delta_l+\gamma_l)\sqrt{\frac{Q_2^2}{4}+\frac{Q_1}{\mathcal{R}_0^M}}}{(\mu d_B)^2(\mu+\delta_l+\gamma_l)^2(Q_2^2\mathcal{R}_0^M+4Q_1)} > 0. \tag{5.8}$$

As (5.8) is strictly positive, it implies that an increase in bacterial meningitis infection leads to an increase in listeriosis.

6. Numerical simulation of the co-infection model

6.1. **Impact of contaminated food products on listeriosis infection dynamics.** To see the impact of foods contaminated by Listeria monocytogenes on the strength of listeriosis infection, we have drawn up a graph showing the evolution of this strength of infection as a function of the foods contaminated. It is illustrated in FIGURE 5. In FIGURE 5, we can clearly see that as the number of foods contaminated by the Listeria monocytogenes bacterium increases, so does the strength of the listeriosis infection. The evolution of this force of infection can be seen in 3D as a function of the population of bacteria and contaminated food given in FIGURE 6.

This increase in the strength of infection of listeriosis logically leads to an increase in the number of basic reproduction of this disease. This can be seen in FIGURE 7 below.

In short, food contaminated with listeria monocytogenes has a considerable impact on the strength of listeriosis infection. The result is an increase in the number of basic listeriosis replicates ($\mathcal{R}_0^L$) and, by the same token, an increase in the number of infected individuals.

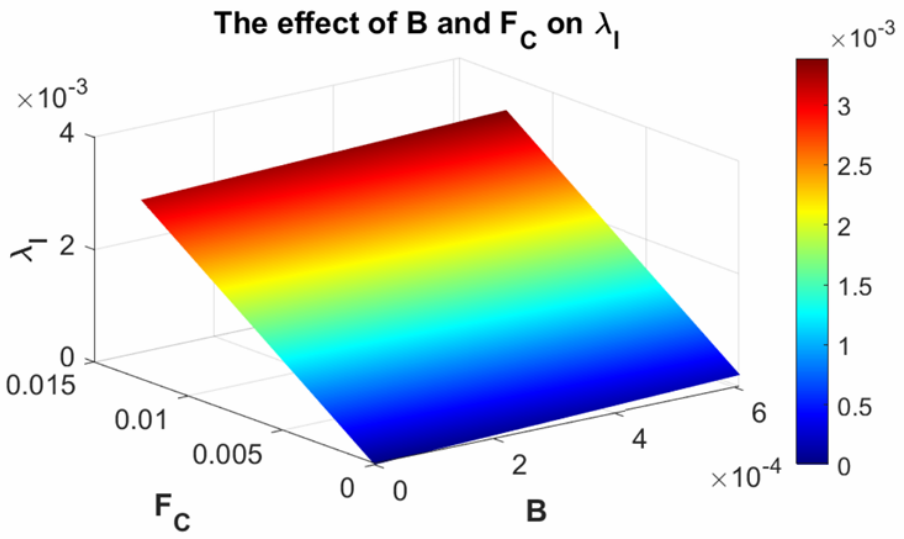

FIGURE 6. 3D representation of the strength of infection as a function of the population of bacteria and contaminated food products.

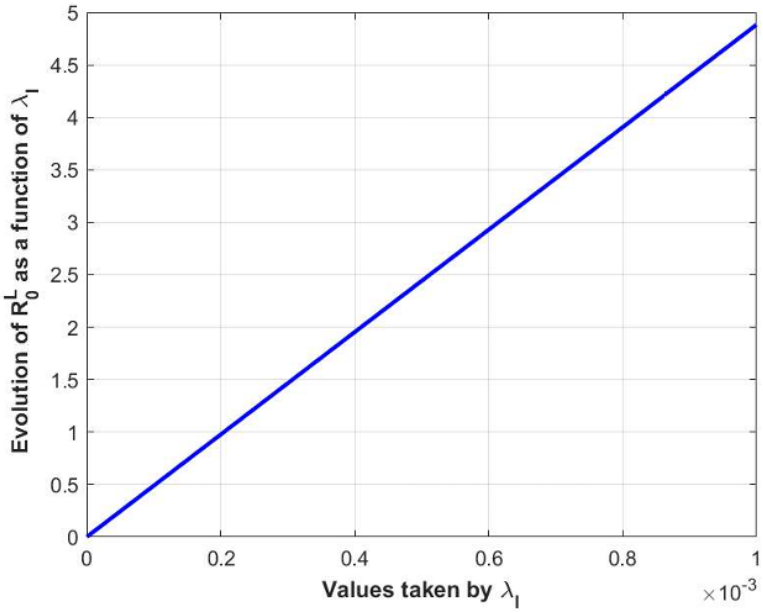

FIGURE 7. 2D representation of the evolution of the basic reproduction number of listeriosis as a function of its infection strength.

6.2. **Numerical simulation.** In this section, we present some numerical simulation results of model (2.1). In our presentation, we consider three (03) cases ranging from a very high infection situation to a situation of extinction of these diseases. Several initial conditions are considered in order

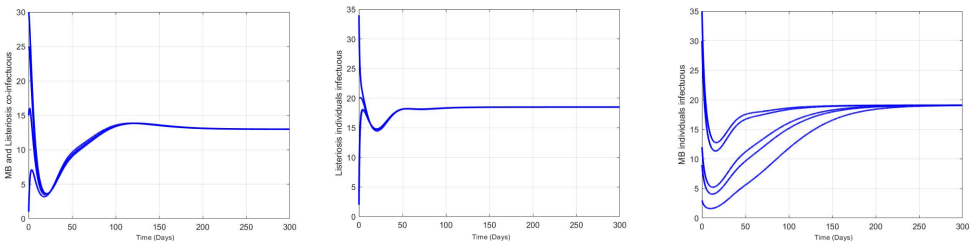

FIGURE 8. Simulations of co-infection model (2.1) with $\beta_l = 0.15$ and $\beta_m = 0.0005$. We obtain $\mathcal{R}_0^L = 1.42$ and $\mathcal{R}_0^{LM} = \mathcal{R}_0^M = 2.4$.

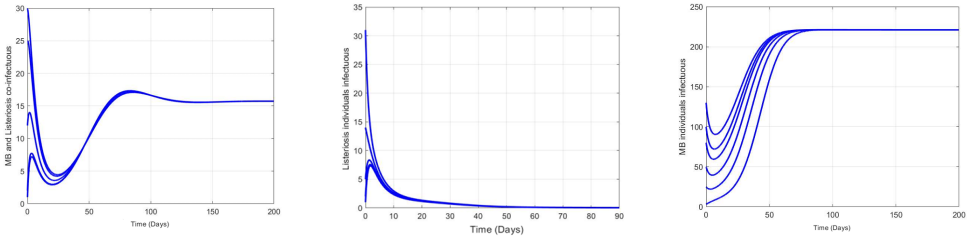

FIGURE 9. Simulations of co-infection model (2.1) with $\beta_l = 0.905$ and $\beta_m = 0.00045$. We obtain $\mathcal{R}_0^L = 0.85$ and $\mathcal{R}_0^{LM} = \mathcal{R}_0^M = 2.16$.

to illustrate compartments I_{lm}, I_l and I_m. All this will be done using the principle of competitive exclusion.

In FIGURE 8, the model simulation shows us a convergence towards a persistence of the two diseases. FIGURE 8 therefore illustrates that for $\mathcal{R}_0^M > 1$ and $\mathcal{R}_0^L > 1$, there is always coexistence of the two diseases, [3]. We have a stability of endemic equilibrium points of this co-infection, listeriosis and bacterial meningitis respectively.

In FIGURE 9, we note a convergence of endemic equilibrium of bacterial meningitis therefore a persistence of this disease only when $\mathcal{R}_0^{LM} = \mathcal{R}_0^M > 1$ and $\mathcal{R}_0^L < 1$. In this section, we note the stability of the endemic equilibrium points for co-infection and bacterial meningitis respectively. On the other hand, the disease-free equilibrium of listeriosis is stable.

In FIGURE 10, we note a convergence of endemic equilibrium of listeriosis therefore a persistence of this disease only when $\mathcal{R}_0^{LM} = \mathcal{R}_0^L > 1$ and $\mathcal{R}_0^M < 1$. In this section, we note the stability of the endemic equilibrium points for co-infection and listeriosis respectively. On the other hand, the disease-free equilibrium of bacterial meningitis is stable.

In FIGURE 11, we notice a convergence towards a disease-free equilibrium when $\mathcal{R}_0^{LM} = \mathcal{R}_0^M < 1$. We clearly observe an extinction of bacterial meningitis and listeriosis, obviously leading to an extinction of co-infection. In this section, we have the stability of disease-free equilibrium of this co-infection, listeriosis and bacterial meningitis respectively.

6.3. **Impact of varying σ_{lm} on co-infectous individuals.** FIGURE 12 show the numerical solutions of (I_{lm}) for different values treatment rates σ_{lm} to observe the impact of varying these parameters.

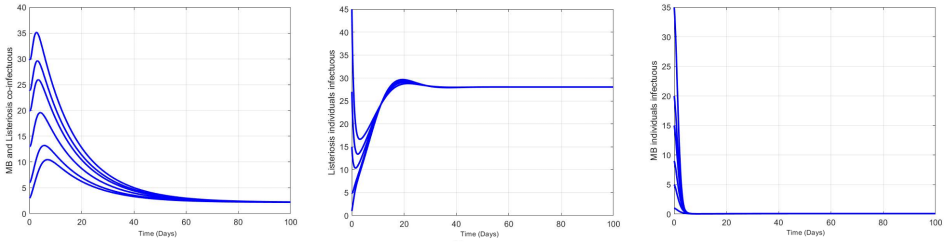

FIGURE 10. Simulations of co-infection model (2.1) with $\beta_l = 0.0209$ and $\beta_m = 0.0001585$. We obtain $\mathcal{R}_0^M = 0.76$ and $\mathcal{R}_0^{LM} = \mathcal{R}_0^L = 1.54$.

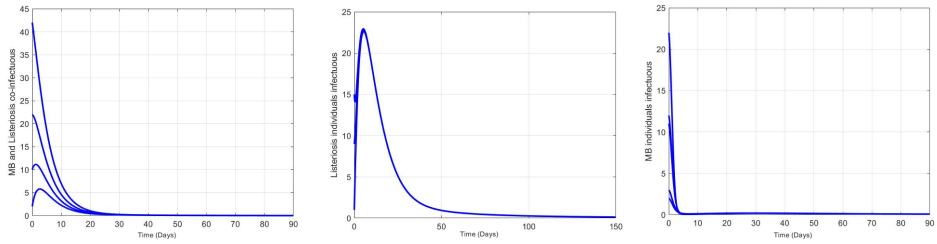

FIGURE 11. Simulations of co-infection model (2.1) with $\beta_l = 0.0805$ and $\beta_m = 0.000145$. We obtain $\mathcal{R}_0^L = 0.59$ and $\mathcal{R}_0^{LM} = \mathcal{R}_0^M = 0.69$.

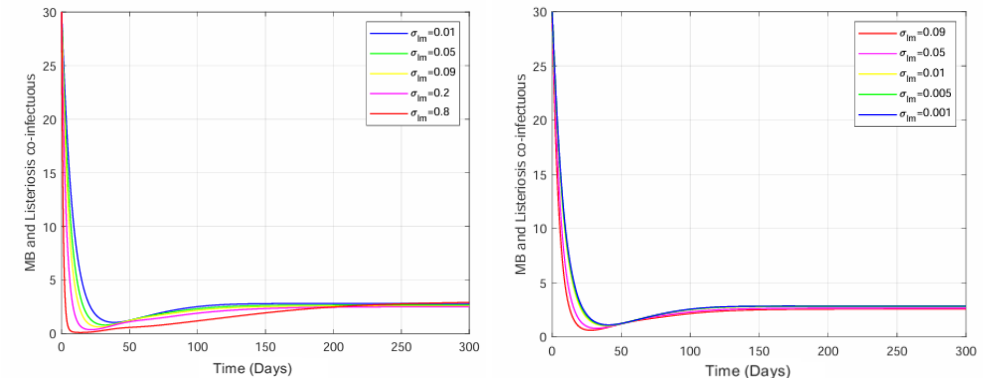

FIGURE 12. The simulation graphics varying σ_{lm} on the listeriosis and bacterial meningitis co-infected individuals (I_{lm}).

We observe that when the co-infection treatment rate σ_{lm} increases, the numbers of listeriosis and bacterial l meningitis co-infection infectuous (I_{lm}) decreases. This suggests that treatment rates need to be increased to reduce listeriosis and bacterial meningitis co-infection.

7. CONCLUSION

This work explores the co-infection of listeriosis and bacterial meningitis, presenting a model that includes infected individuals, co-infected individuals, and those cured with immunity. The model incorporates both the bacterial and food populations, distinguishing between uncontaminated and contaminated foods, [6,11]. A mathematical analysis and simulation were conducted to assess

the impact of contaminated food on listeriosis dynamics.The bacterial meningitis and listeriosis models each have a stable disease-free equilibrium when their basic reproduction numbers $\mathcal{R}_0^M$ and $\mathcal{R}_0^L$ are less than one, and unique endemic points when greater than one. The co-infection model also shows a stable disease-free equilibrium under certain conditions, with potential backward bifurcation. Sensitivity analysis identified key parameters influencing the reproduction numbers for both diseases. Overall, the study highlights the importance of considering bacterial populations and food contamination in modeling co-infections, suggesting that reducing listeriosis bacteria or contaminated foods could significantly lower listeriosis cases and related co-infections, [10, 24].

Conflicts of Interest: The authors declare that there are no conflicts of interest regarding the publication of this paper.

References

[1] Meningitis Research Foundation, The History of Meningitis, Meningitis Research Foundation. https://www.meningitis.org/blogs/the-history-of-meningitis.

[2] A.B. Diabaté, B. Sangaré, O. Koutou, Optimal Control Analysis of a Mathematical Model of Malaria and COVID-19 Co-Infection Dynamics, J. Biol. Dyn. 19 (2025), 2568392. https://doi.org/10.1080/17513758.2025.2568392.

[3] A.B. Diabaté, B. Sangaré, O. Koutou, Optimal Control Analysis of a COVID-19 and Tuberculosis (TB) Co-Infection Model with an Imperfect Vaccine for COVID-19, SeMA J. 81 (2023), 429–456. https://doi.org/10.1007/s40324-023-00330-8.

[4] S. Barocci, A. Mancini, B. Canovari, E. Petrelli, E. Sbriscia-Fioretti, et al. Listeria Monocytogenes Meningitis in an Immunocompromised Patient, New Microbiol. 38 (2015), 113–118.

[5] B. Greenwood, Meningococcal Meningitis in Africa, Trans. R. Soc. Trop. Med. Hyg. 93 (1999), 341–353. https://doi.org/10.1016/s0035-9203(99)90106-2.

[6] B.S. kotola, T.T. Mekonnen, Mathematical Model Analysis and Numerical Simulation for Codynamics of Meningitis and Pneumonia Infection with Intervention, Sci. Rep. 12 (2022), 2639. https://doi.org/10.1038/s41598-022-06253-0.

[7] C. Castillo-Chavez, B. Song, Dynamical Models of Tuberculosis and Their Applications, Math. Biosci. Eng. 1 (2004), 361–404. https://doi.org/10.3934/mbe.2004.1.361.

[8] C. Castillo-Chavez, S. Blower, P. Van Den Driessche, D. Kirschner, A.A. Yakubu, eds., Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory, Springer, New York, 2002. https://doi.org/10.1007/978-1-4613-0065-6.

[9] C. Ma, X. Li, Z. Zhao, F. Liu, K. Zhang, et al., Understanding Dynamics of Pandemic Models to Support Predictions of COVID-19 Transmission: Parameter Sensitivity Analysis of SIR-Type Models, IEEE J. Biomed. Health Inform. 26 (2022), 2458–2468. https://doi.org/10.1109/jbhi.2022.3168825.

[10] C.W. Chukwu, J. Mushanyu, M.L. Juga, Fatmawati, A Mathematical Model for Co-Dynamics of Listeriosis and Bacterial Meningitis Diseases, Commun. Math. Biol. Neurosci. 2020 (2020), 83. https://doi.org/10.28919/cmbn/5060.

[11] C.W. Chukwu, S.Y. Tchoumi, O. Koutou, F.F. Herdicho, Fatmawati, Exploring the Epidemiological Impact of Pneumonia–Listeriosis Co-Infection in the Human Population: A Modeling and Optimal Control Study, Int. J. Dyn. Control. 13 (2025), 197. https://doi.org/10.1007/s40435-025-01714-6.

[12] D.K. Das, T. Kar, Global Dynamics of a Tuberculosis Model with Sensitivity of the Smear Microscopy, Chaos Solitons Fractals 146 (2021), 110879. https://doi.org/10.1016/j.chaos.2021.110879.

[13] H. Hof, History and Epidemiology of Listeriosis, FEMS Immunol. Med. Microbiol. 35 (2003), 199–202. https://doi.org/10.1016/s0928-8244(02)00471-6.

[14] J. Njagarah, F. Nyabadza, A Metapopulation Model for Cholera Transmission Dynamics Between Communities Linked by Migration, Appl. Math. Comput. 241 (2014), 317–331. https://doi.org/10.1016/j.amc.2014.05.036.

[15] J.K.K. Asamoah, F. Nyabadza, Z. Jin, E. Bonyah, M.A. Khan, et al., Backward Bifurcation and Sensitivity Analysis for Bacterial Meningitis Transmission Dynamics with a Nonlinear Recovery Rate, Chaos Solitons Fractals 140 (2020), 110237. https://doi.org/10.1016/j.chaos.2020.110237.

[16] J. McLauchlin, R. Mitchell, W. Smerdon, K. Jewell, Listeria Monocytogenes and Listeriosis: A Review of Hazard Characterisation for Use in Microbiological Risk Assessment of Foods, Int. J. Food Microbiol. 92 (2004), 15–33. https://doi.org/10.1016/S0168-1605(03)00326-X.

[17] J.P. La Salle, The Stability of Dynamical Systems, SIAM, 1976. https://doi.org/10.1137/1.9781611970432.

[18] K.G. Mekonen, L.L. Obsu, Mathematical Modeling and Analysis for the Co-Infection of COVID-19 and Tuberculosis, Heliyon 8 (2022), e11195. https://doi.org/10.1016/j.heliyon.2022.e11195.

[19] K.O. Okosun, R. Smith, Optimal Control Analysis of Malaria-Schistosomiasis Co-Infection Dynamics, Math. Biosci. Eng. 14 (2017), 377–405. https//doi.org/10.3934/mbe.2017024.

[20] A.M. Oordt-Speets, R. Bolijn, R.C. van Hoorn, A. Bhavsar, M.H. Kyaw, Global Etiology of Bacterial Meningitis: A Systematic Review and Meta-Analysis, PLOS ONE 13 (2018), e0198772. https://doi.org/10.1371/journal.pone.0198772.

[21] M. Doganay, Listeriosis: Clinical Presentation, FEMS Immunol. Med. Microbiol. 35 (2003), 173–175. https://doi.org/10.1016/S0928-8244(02)00467-4.

[22] M.M. Koopmans, M.C. Brouwer, J.A. Vázquez-Boland, D. van de Beek, Human Listeriosis, Clin. Microbiol. Rev. 36 (2023), e00060–19. https://doi.org/10.1128/cmr.00060-19.

[23] N.G. Rouphael, D.S. Stephens, Neisseria Meningitidis: Biology, Microbiology, and Epidemiology, in: M. Christodoulides (eds), Neisseria Meningitidis, Methods in Molecular Biology, vol. 799, Humana, Totowa, NJ, 2012, pp. 1–20. https://doi.org/10.1007/978-1-61779-346-2_1.

[24] P. Laguna del Estal, G.M. Lledó Ibáñez, R. Ríos Garcés, I. Pintos Pascual, Meningitis Por Listeria Monocytogenes en Adultos, Rev. Neurol. 56 (2013), 13. https://doi.org/10.33588/rn.5601.2012446.

[25] P. van den Driessche, J. Watmough, Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission, Math. Biosci. 180 (2002), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6.

[26] R. Amaya-Villar, E. García-Cabrera, E. Sulleiro-Igual, P. Fernández-Viladrich, D. Fontanals-Aymerich, et al., Three-Year Multicenter Surveillance of Community-Acquired Listeria Monocytogenes Meningitis in Adults, BMC Infect. Dis. 10 (2010), 324. https://doi.org/10.1186/1471-2334-10-324.

[27] T.J. Irving, K.B. Blyuss, C. Colijn, C.L. Trotter, Modelling Meningococcal Meningitis in the African Meningitis Belt, Epidemiol. Infect. 140 (2011), 897–905. https://doi.org/10.1017/S0950268811001385.

[28] T. Khan, F.A. Rihan, H. Ahmad, Modelling the Dynamics of Acute and Chronic Hepatitis B with Optimal Control, Sci. Rep. 13 (2023), 14980. https://doi.org/10.1038/s41598-023-39582-9.