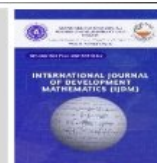




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Mathematical Analysis and Optimal Control of Listeriosis Transmission in a Coupled Human-Food-Bacteria System

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Abstract

Listeriosis is a foodborne disease caused by the bacterium *Listeria monocytogenes*, posing a significant public health threat, particularly to immunocompromised individuals, pregnant women, and newborns. This study develops and analyzes a risk-structured mathematical model for listeriosis transmission dynamics that incorporates high-risk and low-risk susceptible human populations, food contamination, and environmental bacterial reservoirs. The model, formulated as a system of eight nonlinear ordinary differential equations, captures the interactions between human infection, food contamination, and bacterial growth. Three equilibrium points are derived, including disease-free, bacteria-free, and endemic equilibrium points. The contaminated food threshold (R_L) and food safety index (R_C) are established

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as key epidemiological thresholds. Local stability analyses using the Routh-Hurwitz criterion demonstrate that the disease-free and bacteria-free equilibria are locally asymptotically stable whenever $R_L < 1$, while the endemic equilibrium is stable when $R_C < 1$. Global stability of the disease-free equilibrium is established using the Castillo-Chavez method. The model is extended to incorporate three time-dependent control measures: food safety practices (u_1), public health education (u_2), and treatment of infected individuals (u_3). An optimal control problem is formulated with the objective of minimizing infected individuals, contaminated food products, and bacterial presence while minimizing intervention costs. Pontryagin's Maximum Principle is applied to characterize the optimal control strategies. Numerical simulations using forward-backward sweep methods demonstrate that all control strategies significantly reduce disease burden, (combining all three controls) proving most effective in reducing contaminated food by 88.5% and exposed and infectious populations by 90%. The findings provide valuable insights for public health policymakers in designing evidence-based, cost-effective intervention programs for listeriosis control in Nigeria and similar resource-limited settings.

1 Introduction

Listeriosis represents a significant public health concern globally, arising from the consumption of food products contaminated with the pathogenic bacterium *Listeria monocytogenes*. While foodborne transmission constitutes the primary route of infection, human-to-human or animal-to-human contact can also facilitate disease spread, albeit to a considerably lesser extent. Vertical transmission from infected pregnant women to their fetuses represents another important pathway. Immunocompromised individuals, elderly populations, pregnant women, newborns, and persons suffering from chronic conditions such as HIV/AIDS, diabetes, cancer, and renal diseases face the highest susceptibility to this infection. Clinical manifestations typically include pyrexia, influenza-like symptoms, emesis, nausea, and diarrhea. Similar to other bacterial pathogens, listeriosis can be effectively prevented and managed through proper handling and cooking of meat and poultry products, adherence to expiration dates, and avoidance of cross-contamination between raw and cooked foods Iwu and Okoh (2020).

The Nigerian livestock and meat processing sectors have been implicated in the dissemination of *Listeria* species throughout the West African subregion. Odu and Okonko (2017) documented a 7% prevalence rate of *L. monocytogenes* in raw meat specimens obtained from Rivers State in the South-South region of Nigeria, while Ishola et al. (2016) reported a substantially higher prevalence of 91.8% in poultry flocks and chicken meat sampled from Oyo State. In Lafia, Nigeria, the prevalence of *Listeria* spp. in beef and chevon was established at 58.2% (78/134) and 41.8% (56/134), respectively, with 64.4% (67/104) of these isolates confirmed as *L. monocytogenes* Chukwu and Nyabadza (2020). Studies conducted in Enugu State revealed prevalence rates of *Listeria* spp. isolated from beef, pork, and chicken samples as 45.8%, 27.1%, and 13.2%, respectively Odu and Okonko

(2017). Ndahi et al. (2014) detected *L. monocytogenes* in 4.0% of raw meat and meat products examined in Zaria, Nigeria. Investigations into vegetable contamination have demonstrated that tomatoes, cucumber, cabbage, carrots, and lettuce harbor *L. monocytogenes* at prevalence rates of 28.28%, 9.02%, 23.36%, 19.67%, and 19.67%, respectively Ajayeboa et al. (2016). Furthermore, locally manufactured soft cheese (wara) exhibited *Listeria* spp. contamination in 78% of samples, among which 12.4% were identified as *L. monocytogenes* Kunadu et al. (2018). Collectively, these findings suggest a national average prevalence of *L. monocytogenes* in Nigeria of approximately 43.5%.

Mathematical modeling has emerged as an indispensable methodological approach for elucidating the transmission mechanisms of infectious diseases and assessing the efficacy of alternative intervention strategies. Numerous researchers have developed and scrutinized compartmental frameworks to investigate listeriosis dynamics. Osman et al. (2018) conducted a stability analysis of listeria epidemics affecting both human and animal populations, thereby establishing fundamental insights into disease transmission patterns. Chukwu and Nyabadza (2020) formulated a deterministic co-infection model incorporating listeriosis and meningitis, examining the isolated sub-models of each disease independently before analyzing their combined dynamics. The mathematical examination of all co-infection and sub-models was thoroughly undertaken, with Latin hypercube sampling employed to determine the severity parameters governing infection co-dynamics. Their numerical findings indicated that reducing ambient listeria bacteria concentrations and enhancing meningitis recovery rates effectively diminish the incidence of co-infections.

The influence of information dissemination on disease control was explored by Cui et al. (2007), who developed a Susceptible-Exposed-Infected (SEI) model to investigate media effects on infectious disease transmission. Their numerical simulations revealed that the absence of media coverage can precipitate multiple disease outbreaks, whereas media presence substantially abbreviates the duration of secondary transmission peaks. This observation underscores the critical role of non-pharmaceutical interventions in disease mitigation strategies. In recent decades, optimal control theory has gained considerable prominence as a mathematical framework for identifying optimally effective and economically viable intervention schedules for infectious disease management. The optimal control paradigm entails the formulation of control variables representing diverse intervention measures—including but not limited to therapeutic treatment, vaccination programs, public health education campaigns, and food safety protocol enhancements—and subsequently determining time-dependent control profiles that simultaneously minimize disease burden and implementation costs. The Pontryagin Maximum Principle serves as the standard analytical tool for deriving optimality conditions, yielding a coupled system of ordinary differential equations with boundary conditions that characterize the optimal control trajectories.

Numerous investigations have successfully applied optimal control methodologies to foodborne and infectious disease models. Osman et al. (2020) investigated a compartmental listeriosis model incorporating three human and four animal categories. Their qualitative analysis determined the stability properties of both endemic and disease-free equilibria while exploring the potential for forward and backward bifurcation phenomena. Sensitivity analysis was utilized to assess the influence of parameter variations on disease outcomes.

The model incorporated treatment, immunization, and education of susceptible human populations as time-dependent control variables. Employing Pontryagin's Maximum Principle, they identified the optimal intervention strategy and subsequently performed numerical simulations with graphical and quantitative presentation of results.

Okosun *et al.* (2013) demonstrated the effectiveness of combined intervention strategies in malaria transmission through optimal control analysis. Similarly, Laarabi *et al.* (2015) employed optimal control theory to investigate vaccination and treatment strategies for SIR epidemic models, demonstrating that time-dependent control measures significantly reduce disease prevalence. Within the foodborne disease context, Tchuente *et al.* (2011) examined optimal control of food contamination, emphasizing the importance of timely interventions in production and distribution chains. The comprehensive theoretical framework developed by Lenhart and Workman (2007) for optimal control applications to biological and epidemiological models has been widely adopted in subsequent research endeavors.

More contemporary contributions include the work of Kassa and Ouhinou (2015), who developed an optimal control model for cholera dynamics incorporating vaccination, sanitation, and treatment as control variables. Their results demonstrated that combinations of multiple control measures achieve superior outcomes compared to single-intervention strategies. Bonyah and Asiedu-Addo (2016) applied optimal control to a listeriosis model, examining the effects of public health education and food safety interventions on disease transmission. Collectively, these studies consistently affirm the utility of optimal control theory in formulating effective and resource-efficient intervention policies for infectious diseases.

Despite these significant advances, a comprehensive risk-structured listeriosis model incorporating optimal control strategies tailored to Nigeria's specific epidemiological context—characterized by substantial disease burden and heterogeneous food safety infrastructure—remains conspicuously absent from the literature. The present study addresses this gap by developing and analyzing a risk-structured mathematical model of listeriosis transmission dynamics using a system of ordinary differential equations that explicitly distinguishes between high-risk and low-risk susceptible human populations. Three equilibrium points were derived: the disease-free equilibrium point (E_0^d), the bacteria-free equilibrium point (E_0^b), and the endemic equilibrium point (E_0^e). The contaminated food threshold (R_L) was established, and both disease-free and bacteria-free equilibria were demonstrated to be locally asymptotically stable whenever $R_L < 1$. Application of the Routh-Hurwitz criterion established the local asymptotic stability of the endemic equilibrium whenever the food safety index (R_C) remains below unity. Global stability analysis of the disease-free equilibrium, performed using the Castillo-Chavez method, confirmed its global asymptotic stability.

Building upon the stability analyses presented herein, our subsequent investigation will extend the current framework to incorporate optimal control theory. Specifically, we intend to introduce time-dependent control variables corresponding to critical intervention strategies, including public health enlightenment campaigns, enhanced food safety practices, improved therapeutic regimens for infected individuals, and measures to curtail bacterial contamination of food products. The optimal control problem will be formulated with the objective function designed to minimize the infected population and associated intervention

implementation costs. Application of Pontryagin's Maximum Principle will yield the necessary optimality conditions, enabling characterization of optimal control strategies. Numerical simulations will be conducted to evaluate the relative effectiveness of various intervention combinations and identify the most cost-efficient control approaches for listeriosis management in the Nigerian setting. This extension promises to deliver valuable guidance for public health policymakers in designing evidence-based, resource-optimized intervention programs to combat listeriosis transmission.

2 Model Formulation

In order to develop the fundamental mathematical model for the transmission dynamics of listeriosis within and between human, food, and bacterial populations, the entire human population given by $N_H(t)$ is divided into mutually exclusive compartments of susceptible high-risk individuals $S_H(t)$, susceptible low-risk individuals $S_L(t)$, exposed individuals $E(t)$, infectious individuals $I(t)$, and recovered individuals $R(t)$, at time t . Thus:

$$N_H(t) = S_H(t) + S_L(t) + E(t) + I(t) + R(t)$$

Similarly, the total quantity of food products and bacterial population denoted by $F(t)$ and $B(t)$, respectively, are divided into the sub-populations of uncontaminated food products ($U(t)$), contaminated food products ($C(t)$), and environmental *Listeria monocytogenes* bacteria ($B(t)$). Thus:

$$F(t) = U(t) + C(t)$$

Recruitment into the susceptible high-risk population occurs at a rate $(1 - \theta)\Pi_H$, where Π_H is the human recruitment rate and θ is the fraction of recruits entering the low-risk class. Natural mortality rate μ_H is assumed to occur in all human compartments. High-risk individuals transition to the low-risk class at rate σ , reflecting improvements in immune function. The susceptible humans, $S_H(t)$ and $S_L(t)$, become infected by consuming contaminated food products harboring *Listeria monocytogenes*. The force of infection for humans is determined by:

$$\lambda_H = \frac{(1 - u_1)(1 - u_2)\beta_H C}{N_H} \quad (1)$$

where β_H is the transmission rate from contaminated food to high-risk susceptibles, C is the quantity of contaminated food products, N_H is the total human population, u_1 represents food safety practices control, and u_2 represents public health education control. The combined effect $(1 - u_1)(1 - u_2)$ captures the synergistic interaction between food safety infrastructure improvements and individual behavioral changes.

Upon infection, susceptible high-risk and low-risk individuals enter the exposed class $E(t)$ at rates λ_H and $(1 - \psi)\lambda_H$, respectively, where ψ represents the protective efficacy of low-risk immunity. Exposed individuals progress to the infectious class $I(t)$ at rate κ . In the infectious sub-population, individuals recover at rate γ , enhanced by treatment control u_3 to become $(\gamma + \eta u_3)$, where η is the maximum treatment efficacy, or succumb to listeriosis-induced mortality at rate δ . Recovered individuals $R(t)$ acquire immunity and leave the system through natural mortality at rate μ_H .

For food products, the recruitment rate is given as Π_F while μ_F is the removal rate (consumption, spoilage, disposal). Contamination of food products is driven by λ_F , given by:

$$\lambda_F = (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B) \quad (2)$$

where β_C is the contamination rate from contaminated food to uncontaminated food, β_B is the contamination rate from environmental bacteria to food, C is contaminated food quantity, and B is environmental bacterial density. Uncontaminated food becomes contaminated at rate λ_F and moves to the contaminated food class $C(t)$.

For the bacterial population, growth follows logistic dynamics with intrinsic growth rate r and environmental carrying capacity K , subject to natural decay at rate μ_B . Bacteria are reduced through contamination of food products at rate $(1 - u_1)(1 - u_2)\beta_B BU$. The dynamics of listeriosis in a human-food-bacteria community is given by the following deterministic system of nonlinear differential equations in (3), and the description of model system variables and parameters are given in Tables 1 and 3:

$$\begin{aligned} \frac{dS_H}{dt} &= (1 - \theta)\Pi_H - \frac{(1 - u_1)(1 - u_2)\beta_H C S_H}{N_H} - (\sigma + \mu_H)S_H \\ \frac{dS_L}{dt} &= \theta\Pi_H + \sigma S_H - \frac{(1 - u_1)(1 - u_2)(1 - \psi)\beta_H C S_L}{N_H} - \mu_H S_L \\ \frac{dE}{dt} &= \frac{(1 - u_1)(1 - u_2)\beta_H C [S_H + (1 - \psi)S_L]}{N_H} - (\kappa + \mu_H)E \\ \frac{dI}{dt} &= \kappa E - (\gamma + \delta + \mu_H + \eta u_3)I \\ \frac{dR}{dt} &= (\gamma + \eta u_3)I - \mu_H R \\ \frac{dU}{dt} &= \Pi_F - (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B)U - \mu_F U \\ \frac{dC}{dt} &= (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B)U - \mu_F C \\ \frac{dB}{dt} &= rB \left(1 - \frac{B}{K}\right) - \mu_B B - (1 - u_1)(1 - u_2)\beta_B BU \end{aligned} \quad (3)$$

where the infection rate for listeriosis transmission in humans (λ_H) and the contamination rate for food products (λ_F) are given, respectively, by:

$$\lambda_H = \frac{(1 - u_1)(1 - u_2)\beta_H C}{N_H}, \quad \lambda_F = (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B) \quad (4)$$

The model system (3) was formulated under the following primary assumptions:

1. Human recruitment occurs at a constant rate Π_H , with a fraction θ entering the low-risk susceptible class and the remaining fraction $(1 - \theta)$ entering the high-risk susceptible class.
2. High-risk individuals transition to the low-risk class at rate σ , reflecting improvements in immune function or reduced vulnerability following successful treatment of underlying conditions.

3. Infection acquisition in humans occurs exclusively through consumption of contaminated food, with transmission rates β_H and $(1-\psi)\beta_H$ for high-risk and low-risk susceptibles, respectively, where ψ represents the protective efficacy of competent immunity ($0 \leq \psi \leq 1$).
4. Exposed individuals develop clinical listeriosis at rate κ , progressing to the infectious class.
5. Infectious individuals recover at rate γ , acquiring temporary immunity, or succumb to listeriosis-induced mortality at rate δ .
6. Food products are produced at constant rate Π_F and removed at rate μ_F through consumption, spoilage, and disposal.
7. Contamination of uncontaminated food occurs through contact with contaminated food at rate β_C and with environmental bacteria at rate β_B .
8. Bacterial growth follows logistic dynamics with intrinsic growth rate r and environmental carrying capacity K , subject to natural decay at rate μ_B .
9. Enhanced food safety practices (u_1) and public health education (u_2) reduce contamination rates and transmission rates multiplicatively.
10. Treatment interventions (u_3) for infected individuals augment the recovery rate from γ to $(\gamma + \eta u_3)$, where η denotes maximum treatment efficacy.

Table 1. Description of Model State Variables

Variable	Description
$S_H(t)$	Number of high-risk susceptible individuals at time t
$S_L(t)$	Number of low-risk susceptible individuals at time t
$E(t)$	Number of exposed (incubating) individuals at time t
$I(t)$	Number of infectious individuals at time t
$R(t)$	Number of recovered (immune) individuals at time t
$U(t)$	Quantity of uncontaminated food products at time t
$C(t)$	Quantity of contaminated food products at time t
$B(t)$	Environmental <i>L. monocytogenes</i> population density at time t

The model system (3) is supplemented with the following non-negative initial conditions:

$$\begin{aligned}
 S_H(0) = S_H^0 \geq 0, \quad S_L(0) = S_L^0 \geq 0, \quad E(0) = E^0 \geq 0, \quad I(0) = I^0 \geq 0, \quad R(0) = R^0 \geq 0, \\
 U(0) = U^0 \geq 0, \quad C(0) = C^0 \geq 0, \quad B(0) = B^0 \geq 0
 \end{aligned}
 \tag{5}$$

In the absence of control interventions ($u_1 = u_2 = u_3 = 0$), the model simplifies to the baseline system which serves as the foundation for equilibrium and stability analyses.

Table 2. Description of Model Parameters

Parameter	Description
Π_H	Human recruitment rate (individuals per unit time)
Π_F	Food production rate (units per unit time)
θ	Fraction of recruits entering low-risk class ($0 \leq \theta \leq 1$)
σ	Transition rate from high-risk to low-risk class
β_H	Transmission rate from contaminated food to high-risk susceptibles
ψ	Protective efficacy of low-risk immunity ($0 \leq \psi \leq 1$)
κ	Progression rate from exposed to infectious state
γ	Recovery rate from listeriosis
δ	Listeriosis-induced mortality rate
μ_H	Natural death rate of humans
β_C	Contamination rate from contaminated food to uncontaminated food
β_B	Contamination rate from environmental bacteria to food
r	Intrinsic growth rate of <i>L. monocytogenes</i>
K	Environmental carrying capacity of bacteria
μ_B	Natural decay rate of bacteria
μ_F	Food product removal rate (consumption, spoilage, disposal)
η	Maximum treatment efficacy
$u_1(t)$	Food safety practices control ($0 \leq u_1 \leq 1$)
$u_2(t)$	Public health education control ($0 \leq u_2 \leq 1$)
$u_3(t)$	Treatment control ($0 \leq u_3 \leq 1$)

3 Basic Properties of the Model

3.1 Boundedness of Solutions

In this section, we prove that the solutions to model system (3) exist, are non-negative, and are bounded in the region Ω for all time $t > 0$. Assume that Ω is the biological meaningful region for the model equation (3) contained in $\mathbb{R}_+^8$. The positivity of the solutions is governed by the following theorem.

Theorem 1: The closed set $\Omega = \Omega_H \times \Omega_F \times \Omega_B$, with

$$\Omega_H = \left\{ S_H, S_L, E, I, R \in \mathbb{R}_+^5 : N_H \leq \frac{\Pi_H}{\mu_H} \right\}, \quad \Omega_F = \left\{ U, C \in \mathbb{R}_+^2 : F \leq \frac{\Pi_F}{\mu_F} \right\},$$

and

$$\Omega_B = \left\{ B \in \mathbb{R}_+ : B \leq K \left(1 - \frac{\mu_B}{r} \right) \right\}$$

is positively invariant with respect to model equations (3).

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

$$N_H(t) = S_H(t) + S_L(t) + E(t) + I(t) + R(t)$$

and is differentiated and summed together to have

$$\frac{dN_H}{dt} = \Pi_H - \mu_H N_H - \delta I - \eta u_3 I$$

In absence of mortality due to *Listeria* (i.e. $\delta = 0$ and $u_3 = 0$) then by standard comparison theorem and rearranging the equation, we obtain

$$\frac{dN_H}{dt} + \mu_H N_H \leq \Pi_H$$

Solving by integral factor method, we have

$$N_H(t) \leq \frac{\Pi_H}{\mu_H} [1 - \exp(-\mu_H t)] + N_H(0) \exp(-\mu_H t)$$

as $t \rightarrow \infty$, we have that $N_H(t) \leq \frac{\Pi_H}{\mu_H}$.

Following a similar approach yields similar result for food products.

$$F(t) \leq \frac{\Pi_F}{\mu_F}$$

Furthermore, for the bacteria population, we have that

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

Thus, as $t \rightarrow \infty$, $B(t) \rightarrow K \left(1 - \frac{\mu_B}{r}\right)$. So, we note that for human *Listeria* to exist, its decay rate, μ_B must be less than its growth rate, r . Implying that $0 \leq B \leq K \left(1 - \frac{\mu_B}{r}\right)$. Thus, we have shown that Ω is positively invariant and attracts all solutions of model equation (3) 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 Ω will always remain in Ω for all time $t > 0$.

3.2 Non-negativity of Solutions

Next, we establish that every solution of the model equation (3) will be non-negative for all time t .

Theorem 2: Let $(S_H(0), S_L(0), E(0), I(0), R(0), U(0), C(0), B(0))$ be the initial states of the model equation (3). Then every solution $(S_H(t), S_L(t), E(t), I(t), R(t), U(t), C(t), B(t)) \geq 0$ for all time $t > 0$.

Proof: From the first equation of our model equations (3), we have

$$\begin{aligned} \frac{dS_H}{dt} &= (1 - \theta)\Pi_H - \left(\frac{(1 - u_1)(1 - u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H \\ \frac{dS_H}{dt} + \left(\frac{(1 - u_1)(1 - u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H &\geq (1 - \theta)\Pi_H \end{aligned}$$

Solving using integral factor method gives

$$\begin{aligned} \frac{d}{dt} \left[S_H \exp \left\{ \mu_H t + \sigma t + \int_0^t \frac{(1 - u_1)(1 - u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right\} \right] \\ \geq (1 - \theta)\Pi_H \exp \left\{ \mu_H t + \sigma t + \int_0^t \frac{(1 - u_1)(1 - u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right\} \end{aligned}$$

Integrating both sides, we have

$$\begin{aligned} S_H(t) \exp \left\{ \mu_H t + \sigma t + \int_0^t \frac{(1-u_1)(1-u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right\} - S_H(0) \\ \geq (1-\theta)\Pi_H \int_0^t \exp \left\{ \mu_H s + \sigma s + \int_0^s \frac{(1-u_1)(1-u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right\} ds \end{aligned}$$

so that

$$\begin{aligned} S_H(t) \geq S_H(0) \exp \left[- \left(\mu_H t + \sigma t + \int_0^t \frac{(1-u_1)(1-u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right) \right] \\ + \exp \left[- \left(\mu_H t + \sigma t + \int_0^t \frac{(1-u_1)(1-u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right) \right] \\ \times (1-\theta)\Pi_H \int_0^t \exp \left\{ \mu_H s + \sigma s + \int_0^s \frac{(1-u_1)(1-u_2)\beta_H C(\tau)}{N_H(\tau)} d\tau \right\} ds \end{aligned}$$

This implies that $S_H(t) > 0$ for $t > 0$, hence, $S_H(t) \geq 0$ for $t > 0$. In the same manner, it can be shown that $E(t) > 0$, $I(t) > 0$, $R(t) > 0$, $U(t) > 0$, $C(t) > 0$, and $B(t) > 0$.

3.3 Existence and Uniqueness of Solutions

Theorem 3: The model equation (3) defined by $\mathbb{R}_+^8 \in \Omega$ has a solution that exists, and the solution is unique in the region Ω if $\forall t \geq 0$

$$\left| \frac{\partial f_i}{\partial X} \right| < \infty, \quad i = 1(1)8 \text{ and } X = \{S_H, S_L, E, I, R, U, C, B\}$$

$$|f(t, X^2) - f(t, X^1)| \leq M|X^2 - X^1|, \quad \text{where } M \text{ is Lipschitz constant.}$$

Proof: We write the model equation (3) as

$$\begin{aligned} f_1 &= (1-\theta)\Pi_H - \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H \\ f_2 &= \theta\Pi_H + \sigma S_H - \left(\frac{(1-u_1)(1-u_2)(1-\psi)\beta_H C}{N_H} + \mu_H \right) S_L \\ f_3 &= \frac{(1-u_1)(1-u_2)\beta_H C[S_H + (1-\psi)S_L]}{N_H} - (\kappa + \mu_H)E \\ f_4 &= \kappa E - (\gamma + \delta + \mu_H + \eta u_3)I \\ f_5 &= (\gamma + \eta u_3)I - \mu_H R \\ f_6 &= \Pi_F - (1-u_1)(1-u_2)(\beta_C C + \beta_B B)U - \mu_F U \\ f_7 &= (1-u_1)(1-u_2)(\beta_C C + \beta_B B)U - \mu_F C \\ f_8 &= rB \left(1 - \frac{B}{K} \right) - \mu_B B - (1-u_1)(1-u_2)\beta_B BU \end{aligned}$$

with

$$\lambda_H = \frac{(1-u_1)(1-u_2)\beta_H C}{N_H} \quad \text{and} \quad \lambda_F = (1-u_1)(1-u_2)(\beta_C C + \beta_B B)$$

Taking the partial derivative of f_1 with respect to each state variable defined by X , we have

$$\left| \frac{\partial f_1}{\partial S_H} \right| = \left| \frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right| < \infty$$

$$\left| \frac{\partial f_1}{\partial C} \right| = \left| \frac{(1-u_1)(1-u_2)\beta_H S_H}{N_H} \right| < \infty$$

and

$$\left| \frac{\partial f_1}{\partial S_L} \right| = \left| \frac{\partial f_1}{\partial E} \right| = \left| \frac{\partial f_1}{\partial I} \right| = \left| \frac{\partial f_1}{\partial R} \right| = \left| \frac{\partial f_1}{\partial U} \right| = \left| \frac{\partial f_1}{\partial B} \right| = 0 < \infty$$

Repeating the same for other functions $f_2, f_3, \dots, f_8$ and the first condition of theorem 3 is satisfied. For the second condition of theorem 3, we let X^1 and X^2 be any two points in the region Ω for the system of equations (3), we check each variable of X at these points to see if the system satisfies the Lipschitz condition. Consider:

$$f_1 = (1-\theta)\Pi_H - \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H$$

this function at any two points of S_H is

$$f_1(t, S_H^2) = (1-\theta)\Pi_H - \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H^2$$

$$f_1(t, S_H^1) = (1-\theta)\Pi_H - \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) S_H^1$$

therefore,

$$\begin{aligned} |f_1(t, S_H^2) - f_1(t, S_H^1)| &= \left| \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right) (S_H^1 - S_H^2) \right| \\ &\leq \left| \frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right| |S_H^2 - S_H^1| \end{aligned}$$

where $M = \left(\frac{(1-u_1)(1-u_2)\beta_H C}{N_H} + \sigma + \mu_H \right)$ and is Lipschitz constant. The process is repeated for other variables of $X \in f_1$ and functions, which establishes the Lipschitz condition. Hence, this completes the proof.

4 Theoretical Analysis of the Model

4.1 Existence of Model Equilibria

The disease-free equilibrium point of the Listeriosis model system (3) is obtained by setting the infected compartments and contaminated variables to zero, i.e., $E = I = C = B = 0$. Thus, the disease-free equilibrium is given as:

$$\mathcal{H}_0 = \left[\frac{(1-\theta)\Pi_H}{\sigma + \mu_H}, \frac{\Pi_H[\theta(\sigma + \mu_H) + \sigma(1-\theta)]}{\mu_H(\sigma + \mu_H)}, 0, 0, 0, \frac{\Pi_F}{\mu_F}, 0, 0 \right] \quad (6)$$

The next generation operator approach is used to investigate the local asymptotic stability of the DFE ?. In particular, it follows from model system (3), the non-negative matrix,

F , of new infection terms and its M-matrix, V , of linear transition terms are, respectively, generated by:

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_H S_H^0}{N_H^0} & 0 \\ 0 & 0 & \frac{(1-\psi)\beta_H S_L^0}{N_H^0} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \kappa + \mu_H & 0 & 0 & 0 \\ -\kappa & \gamma + \delta + \mu_H & 0 & 0 \\ 0 & 0 & \mu_F & 0 \\ 0 & 0 & 0 & \mu_F \end{bmatrix} \quad (7)$$

The following can be conveniently defined (where ρ is the spectral radius of FV^{-1}):

$$\mathcal{R}_0 = \rho(FV^{-1}) = \mathcal{R}_{0H} + \mathcal{R}_{0C} \quad (8)$$

where:

$$\mathcal{R}_{0H} = \frac{\beta_H \kappa [S_H^0 + (1-\psi)S_L^0]}{N_H^0 (\kappa + \mu_H) (\gamma + \delta + \mu_H)}, \quad \mathcal{R}_{0C} = \frac{\beta_C \Pi_F}{\mu_F^2}$$

4.1.1 Local Stability Analysis of $\mathcal{H}_0$

We establish the proof of the Listeriosis disease-free equilibrium point $\mathcal{H}_0$ using the Jacobian method.

Theorem 3.1: The Listeriosis model system (3) disease-free equilibrium point $\mathcal{H}_0$ is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$.

Proof: We computed the Jacobian of Listeriosis model system (3) and evaluated at disease-free equilibrium, $\mathcal{H}_0$ as follows:

$$J(\mathcal{H}_0) = \begin{bmatrix} -a_1 & 0 & 0 & 0 & 0 & 0 & -a_2 & 0 \\ \sigma & -\mu_H & 0 & 0 & 0 & 0 & -a_3 & 0 \\ 0 & 0 & -a_4 & 0 & 0 & 0 & a_5 & 0 \\ 0 & 0 & \kappa & -a_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_F & -a_7 & -a_8 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_7 - \mu_F & a_8 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_9 \end{bmatrix} \quad (9)$$

where:

$$\begin{aligned} a_1 &= \sigma + \mu_H, & a_2 &= \frac{\beta_H S_H^0}{N_H^0}, & a_3 &= \frac{(1-\psi)\beta_H S_L^0}{N_H^0}, & a_4 &= \kappa + \mu_H, \\ a_5 &= \frac{\beta_H [S_H^0 + (1-\psi)S_L^0]}{N_H^0}, & a_6 &= \gamma + \delta + \mu_H, & a_7 &= \beta_C U^0, \\ a_8 &= \beta_B U^0, & a_9 &= r - \mu_B - \beta_B U^0 \end{aligned}$$

The eigenvalues of (9) are:

$$\begin{aligned}\lambda_1 &= -(\sigma + \mu_H), & \lambda_2 &= -\mu_H, & \lambda_3 &= -(\kappa + \mu_H), \\ \lambda_4 &= -(\gamma + \delta + \mu_H), & \lambda_5 &= -\mu_H, & \lambda_6 &= -\mu_F, \\ \lambda_7 &= \mu_F(\mathcal{R}_{0C} - 1), & \lambda_8 &= r - \mu_B - \beta_B U^0\end{aligned}$$

For the disease-free equilibrium to be locally asymptotically stable, we require:

$$\lambda_7 < 0 \implies \mathcal{R}_{0C} < 1 \quad \text{and} \quad \lambda_8 < 0 \implies r < \mu_B + \beta_B U^0.$$

Thus, the disease-free equilibrium $\mathcal{H}_0$ is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable otherwise.

4.1.2 Global Stability Analysis of $\mathcal{H}_0$

Theorem 3.2: For the risk-structured Listeriosis model, the disease-free equilibrium, $\mathcal{H}_0$, is unstable if $\mathcal{R}_0 > 1$ and globally asymptotically stable (GAS) if $\mathcal{R}_0 < 1$.

Proof. The proof is based on the Castillo-Chavez and Song (2004) Theorem ?. Let $\mathcal{C}_1$ and $\mathcal{C}_2$ describe the uninfected and infected compartments of the model system (3) respectively. Thus,

$$\mathcal{C}_1 = [S_H, S_L, R, U]^T, \quad \mathcal{C}_2 = [E, I, C, B]^T$$

Thus, the model system (3) can be expressed as follows:

$$\frac{d\mathcal{C}_1}{dt} = F(\mathcal{C}_1, \mathcal{C}_2), \quad \frac{d\mathcal{C}_2}{dt} = G(\mathcal{C}_1, \mathcal{C}_2); \quad G(\mathcal{C}_1, \mathbf{0}) = \mathbf{0} \quad (10)$$

where the functions F and G are the corresponding right-hand sides of the model system (3). By the Castillo-Chavez & Song (2004) Theorem ? to guarantee the global asymptotic stability of the disease-free equilibrium, $\mathcal{H}_0$, the following conditions $\mathcal{H}_1$ and $\mathcal{H}_2$ must be satisfied:

$$(\mathcal{H}_1) : (\mathcal{C}_1^0, \mathbf{0}) = \left[\frac{(1-\theta)\Pi_H}{\sigma + \mu_H}, \frac{\Pi_H[\theta(\sigma + \mu_H) + \sigma(1-\theta)]}{\mu_H(\sigma + \mu_H)}, 0, \frac{\Pi_F}{\mu_F}, 0, 0, 0, 0 \right]^T$$

is globally asymptotically stable for $\frac{d\mathcal{C}_1}{dt} = F(\mathcal{C}_1, \mathbf{0}) = \mathbf{0}$.

$$(\mathcal{H}_2) : \hat{G} \geq 0 \quad \text{where} \quad \hat{G}(\mathcal{C}_1, \mathcal{C}_2) = A\mathcal{C}_2 - G(\mathcal{C}_1, \mathcal{C}_2) \quad \text{such that} \quad D_{\mathcal{C}_2}G(\mathcal{C}_1^0, \mathbf{0})$$

is an M-matrix $\forall (\mathcal{C}_1, \mathcal{C}_2) \in \Omega$ To verify $\mathcal{H}_1$, we have, from the model system (3);

$$\frac{d\mathcal{C}_1}{dt} = F(\mathcal{C}_1, \mathbf{0}) = \begin{bmatrix} (1-\theta)\Pi_H - (\sigma + \mu_H)S_H \\ \theta\Pi_H + \sigma S_H - \mu_H S_L \\ 0 \\ \Pi_F - \mu_F U \end{bmatrix} \quad (11)$$

The system (11) can be solved to determine the behavior of each of the additional

compartments in $\mathcal{C}_1$. As a result, we got:

$$\begin{bmatrix} S_H \\ S_L \\ R \\ U \end{bmatrix} = \begin{bmatrix} \frac{(1-\theta)\Pi_H}{\sigma + \mu_H} + \left(S_H(0) - \frac{(1-\theta)\Pi_H}{\sigma + \mu_H} \right) \exp[-(\sigma + \mu_H)t] \\ \frac{\theta\Pi_H + \sigma S_H}{\mu_H} + \left(S_L(0) - \frac{\theta\Pi_H + \sigma S_H}{\mu_H} \right) \exp[-\mu_H t] \\ 0 \\ \frac{\Pi_F}{\mu_F} + \left(U(0) - \frac{\Pi_F}{\mu_F} \right) \exp[-\mu_F t] \end{bmatrix} \quad (12)$$

We can clearly see that $\lim_{t \rightarrow \infty} \mathcal{C}_1 = \mathcal{H}_0$. To verify condition $\mathcal{H}_2$, we find an M-matrix $A = D_{\mathcal{C}_2}G(\mathcal{C}_1^0, \mathbf{0})$. Thus,

$$A = \begin{bmatrix} -(\kappa + \mu_H) & 0 & \frac{\beta_H[S_H^0 + (1-\psi)S_L^0]}{N_H^0} & 0 \\ \kappa & -(\gamma + \delta + \mu_H) & 0 & 0 \\ 0 & 0 & -\mu_F & 0 \\ 0 & 0 & 0 & r - \mu_B - \beta_B U^0 \end{bmatrix} \quad (13)$$

Obviously, A is an M-Matrix. Then given that

$$G(\mathcal{C}_1, \mathcal{C}_2) = \begin{bmatrix} \frac{\beta_H C[S_H + (1-\psi)S_L]}{N_H} - (\kappa + \mu_H)E \\ \kappa E - (\gamma + \delta + \mu_H)I \\ (\beta_C C + \beta_B B)U - \mu_F C \\ rB \left(1 - \frac{B}{K} \right) - \mu_B B - \beta_B BU \end{bmatrix} \quad (14)$$

Therefore:

$$\hat{G}(\mathcal{C}_1, \mathcal{C}_2) = A\mathcal{C}_2 - G(\mathcal{C}_1, \mathcal{C}_2) = [\hat{G}_1, 0, \hat{G}_3, \hat{G}_4]^T \quad (15)$$

where:

$$\hat{G}_1 = \frac{\beta_H[S_H^0 + (1-\psi)S_L^0]}{N_H^0}C - \frac{\beta_H C[S_H + (1-\psi)S_L]}{N_H},$$

$$\hat{G}_3 = \beta_B B(U^0 - U), \quad \hat{G}_4 = \frac{rB^2}{K}$$

Since $S_H^0 \geq S_H$, $S_L^0 \geq S_L$, $N_H^0 \geq N_H$, $U^0 \geq U$, and $B \geq 0$, then $\hat{G}_1 \geq 0$, $\hat{G}_3 \geq 0$, and $\hat{G}_4 \geq 0$. Therefore, $\hat{G}(\mathcal{C}_1, \mathcal{C}_2) \geq 0$ and thus, Conditions $\mathcal{H}_1$ and $\mathcal{H}_2$ hold. Thus, the risk-structured Listeriosis disease-free equilibrium point $\mathcal{H}_0$ is GAS. $\square$

5 Optimal Control of the Listeriosis Model

Numerous attempts have been made to eradicate listeriosis infection prevalence in the population and thereby lower the number of affected persons, yet every attempt has been futile. To effectively restrict disease spread, it is important to determine the optimal

level of effort required. Based on the model formulation presented in Section 2, we updated the listeriosis model system (3) by including three control measures namely: food safety practices $u_1(t)$, public health education and enlightenment campaigns $u_2(t)$, and treatment of infected individuals $u_3(t)$. Thus, we arrive at the modified listeriosis model equation:

$$\left\{ \begin{array}{l} \frac{dS_H}{dt} = (1 - \theta)\Pi_H - \frac{(1 - u_1)(1 - u_2)\beta_H C S_H}{N_H} - (\sigma + \mu_H)S_H, \\ \frac{dS_L}{dt} = \theta\Pi_H + \sigma S_H - \frac{(1 - u_1)(1 - u_2)(1 - \psi)\beta_H C S_L}{N_H} - \mu_H S_L, \\ \frac{dE}{dt} = \frac{(1 - u_1)(1 - u_2)\beta_H C [S_H + (1 - \psi)S_L]}{N_H} - (\kappa + \mu_H)E, \\ \frac{dI}{dt} = \kappa E - (\gamma + \delta + \mu_H + \eta u_3)I, \\ \frac{dR}{dt} = (\gamma + \eta u_3)I - \mu_H R, \\ \frac{dU}{dt} = \Pi_F - (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B)U - \mu_F U, \\ \frac{dC}{dt} = (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B)U - \mu_F C, \\ \frac{dB}{dt} = rB \left(1 - \frac{B}{K}\right) - \mu_B B - (1 - u_1)(1 - u_2)\beta_B B U. \end{array} \right. \quad (16)$$

The goal of this section is to examine the optimum level of effort needed to stop the spread of listeriosis. This is achieved by formulating an objective functional:

$$I = \min_{u_1, u_2, u_3} \int_0^{t_f} \left(Q_1 E + Q_2 I + Q_3 C + Q_4 B + \frac{C_1 u_1^2}{2} + \frac{C_2 u_2^2}{2} + \frac{C_3 u_3^2}{2} \right) dt, \quad (17)$$

where: - Q_1, Q_2, Q_3, Q_4 are positive weight constants representing the relative costs associated with exposed individuals, infectious individuals, contaminated food products, and bacterial presence, respectively. - C_1, C_2, C_3 are positive weight constants representing the relative costs of implementing food safety practices, public health education, and treatment interventions, respectively. - t_f is the final time of the intervention period. Our goal is to minimize infection rates in humans, food contamination, and bacterial presence at a low cost of controls $u_1(t)$, $u_2(t)$, and $u_3(t)$. We seek optimal controls u_1^*, u_2^* , and u_3^* that satisfy:

$$I(u_1^*, u_2^*, u_3^*) = \min_{u_1, u_2, u_3 \in \Omega} I(u_1, u_2, u_3), \quad (18)$$

where Ω is the set of measurable functions defined on $[0, t_f]$ taking values in $[0, 1]$. The optimal control conditions are based on Pontryagin's Maximum Principle [Pontryagin et al. \(1986\)](#), and the adjoint variables of the state variables satisfy equation (16). This principle transforms (16) into a point-wise Hamiltonian H minimization problem with

respect to (u_1, u_2, u_3) . Thus, the Hamiltonian is:

$$\begin{aligned}
 H = & Q_1 E + Q_2 I + Q_3 C + Q_4 B + \frac{C_1 u_1^2}{2} + \frac{C_2 u_2^2}{2} + \frac{C_3 u_3^2}{2} \\
 & + \lambda_{S_H} \left[(1 - \theta) \Pi_H - \frac{(1 - u_1)(1 - u_2) \beta_H C S_H}{N_H} - (\sigma + \mu_H) S_H \right] \\
 & + \lambda_{S_L} \left[\theta \Pi_H + \sigma S_H - \frac{(1 - u_1)(1 - u_2)(1 - \psi) \beta_H C S_L}{N_H} - \mu_H S_L \right] \\
 & + \lambda_E \left[\frac{(1 - u_1)(1 - u_2) \beta_H C [S_H + (1 - \psi) S_L]}{N_H} - (\kappa + \mu_H) E \right] \\
 & + \lambda_I [\kappa E - (\gamma + \delta + \mu_H + \eta u_3) I] \\
 & + \lambda_R [(\gamma + \eta u_3) I - \mu_H R] \\
 & + \lambda_U [\Pi_F - (1 - u_1)(1 - u_2)(\beta_C C + \beta_B B) U - \mu_F U] \\
 & + \lambda_C [(1 - u_1)(1 - u_2)(\beta_C C + \beta_B B) U - \mu_F C] \\
 & + \lambda_B \left[r B \left(1 - \frac{B}{K} \right) - \mu_B B - (1 - u_1)(1 - u_2) \beta_B B U \right], \tag{19}
 \end{aligned}$$

where $\lambda_{S_H}, \lambda_{S_L}, \lambda_E, \lambda_I, \lambda_R, \lambda_U, \lambda_C, \lambda_B$ are the adjoint or co-state variables associated with the state variables $S_H, S_L, E, I, R, U, C, B$, respectively. **Theorem 4:** Given optimal controls u_1^*, u_2^*, u_3^* and corresponding state variables $S_H^*, S_L^*, E^*, I^*, R^*, U^*, C^*, B^*$ of the state system (16) that minimizes $I(u_1, u_2, u_3)$ over Ω . Then, there exist adjoint variables $\lambda_{S_H}, \lambda_{S_L}, \lambda_E, \lambda_I, \lambda_R, \lambda_U, \lambda_C, \lambda_B$ satisfying:

$$-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial i}, \tag{20}$$

with transversality conditions:

$$\lambda_{S_H}(t_f) = \lambda_{S_L}(t_f) = \lambda_E(t_f) = \lambda_I(t_f) = \lambda_R(t_f) = \lambda_U(t_f) = \lambda_C(t_f) = \lambda_B(t_f) = 0, \tag{21}$$

and the controls u_1^*, u_2^* , and u_3^* satisfy the optimality conditions:

$$\begin{aligned}
 u_1^* = & \max \left\{ 0, \min \left\{ 1, \frac{(1 - u_2) \beta_H C [S_H(\lambda_E - \lambda_{S_H}) + (1 - \psi) S_L(\lambda_E - \lambda_{S_L})]}{C_1 N_H} \right. \right. \\
 & \left. \left. + \frac{(1 - u_2)(\beta_C C + \beta_B B) U (\lambda_C - \lambda_U)}{C_1} + \frac{(1 - u_2) \beta_B B U \lambda_B}{C_1} \right\} \right\}, \\
 u_2^* = & \max \left\{ 0, \min \left\{ 1, \frac{(1 - u_1) \beta_H C [S_H(\lambda_E - \lambda_{S_H}) + (1 - \psi) S_L(\lambda_E - \lambda_{S_L})]}{C_2 N_H} \right. \right. \\
 & \left. \left. + \frac{(1 - u_1)(\beta_C C + \beta_B B) U (\lambda_C - \lambda_U)}{C_2} + \frac{(1 - u_1) \beta_B B U \lambda_B}{C_2} \right\} \right\}, \\
 u_3^* = & \max \left\{ 0, \min \left\{ 1, \frac{\eta I (\lambda_I - \lambda_R)}{C_3} \right\} \right\}. \tag{22}
 \end{aligned}$$

Proof: Differentiating the Hamiltonian function (19) evaluated at the optimal control yields the differentiable equations governing the adjoint variables; thus, the adjoint system

is:

$$\begin{aligned}
 \frac{d\lambda_{S_H}}{dt} &= -\frac{\partial H}{\partial S_H} \\
 &= \frac{(1-u_1)(1-u_2)\beta_H C}{N_H}(\lambda_{S_H} - \lambda_E) + \mu_H \lambda_{S_H} + \sigma(\lambda_{S_H} - \lambda_{S_L}), \\
 \frac{d\lambda_{S_L}}{dt} &= -\frac{\partial H}{\partial S_L} \\
 &= \frac{(1-u_1)(1-u_2)(1-\psi)\beta_H C}{N_H}(\lambda_{S_L} - \lambda_E) + \mu_H \lambda_{S_L}, \\
 \frac{d\lambda_E}{dt} &= -\frac{\partial H}{\partial E} \\
 &= -Q_1 + \mu_H \lambda_E + \kappa(\lambda_E - \lambda_I), \\
 \frac{d\lambda_I}{dt} &= -\frac{\partial H}{\partial I} \\
 &= -Q_2 + (\gamma + \delta + \mu_H + \eta u_3)\lambda_I - (\gamma + \eta u_3)\lambda_R, \\
 \frac{d\lambda_R}{dt} &= -\frac{\partial H}{\partial R} \\
 &= \mu_H \lambda_R, \\
 \frac{d\lambda_U}{dt} &= -\frac{\partial H}{\partial U} \\
 &= (1-u_1)(1-u_2)(\beta_C C + \beta_B B)(\lambda_U - \lambda_C) + \mu_F \lambda_U, \\
 \frac{d\lambda_C}{dt} &= -\frac{\partial H}{\partial C} \\
 &= -Q_3 + \frac{(1-u_1)(1-u_2)\beta_H [S_H(\lambda_{S_H} - \lambda_E) + (1-\psi)S_L(\lambda_{S_L} - \lambda_E)]}{N_H} \\
 &\quad + (1-u_1)(1-u_2)\beta_C U(\lambda_U - \lambda_C) + \mu_F \lambda_C, \\
 \frac{d\lambda_B}{dt} &= -\frac{\partial H}{\partial B} \\
 &= -Q_4 + (1-u_1)(1-u_2)\beta_B U(\lambda_U - \lambda_C + \lambda_B) \\
 &\quad - r\lambda_B \left(1 - \frac{2B}{K}\right) + \mu_B \lambda_B.
 \end{aligned} \tag{23}$$

Hence, solving $\frac{\partial H}{\partial u_1} = 0$, $\frac{\partial H}{\partial u_2} = 0$, and $\frac{\partial H}{\partial u_3} = 0$ and equating the result to zero gives the characterization of controls:

$$\begin{aligned}
 u_1 &= \frac{(1-u_2)\beta_H C[S_H(\lambda_E - \lambda_{S_H}) + (1-\psi)S_L(\lambda_E - \lambda_{S_L})]}{C_1 N_H} \\
 &\quad + \frac{(1-u_2)(\beta_C C + \beta_B B)U(\lambda_C - \lambda_U)}{C_1} + \frac{(1-u_2)\beta_B B U \lambda_B}{C_1}, \\
 u_2 &= \frac{(1-u_1)\beta_H C[S_H(\lambda_E - \lambda_{S_H}) + (1-\psi)S_L(\lambda_E - \lambda_{S_L})]}{C_2 N_H} \\
 &\quad + \frac{(1-u_1)(\beta_C C + \beta_B B)U(\lambda_C - \lambda_U)}{C_2} + \frac{(1-u_1)\beta_B B U \lambda_B}{C_2}, \\
 u_3 &= \frac{\eta I(\lambda_I - \lambda_R)}{C_3}.
 \end{aligned} \tag{24}$$

Using the bounds on the controls and normal control parameters, we ensure that:

$$u_i^* = \begin{cases} 0, & \text{if } u_i \leq 0, \\ u_i, & \text{if } 0 < u_i < 1, \\ 1, & \text{if } u_i \geq 1, \end{cases} \quad i = 1, 2, 3. \quad (25)$$

The uniqueness of the optimality system (16)-(23), which restricts the duration of time intervals to ensure the system's uniqueness, is obtained as a result of the solutions to the state and adjoint equations being bounded. This is because the state problem has initial values, whereas the adjoint problem has final values, arising from the opposite temporal orientations. This limitation is common in control problems, according to Joshi (2002); Kirschner et al. (1997); Lenhart and Bhat (1992).

6 Numerical Simulations and Discussion

In this section, we examine the effects of each control strategy to determine which control technique is most effective at reducing or even eliminating the spread of listeriosis in the population. The methods mentioned above, which employ the Range-Kutta forward and backward scheme, are used to numerically solve the optimal control problem (16). To reduce the frequency of exposed individuals (E), infectious individuals (I), contaminated food products (C), and bacterial population (B) in the population, we use the variables and factors listed in Table 3. We set a 50-day time window for implementation.

Table 3. Baseline parameter values for the listeriosis model

Parameter	Baseline Value	Source
Π_H	100 day ⁻¹	Okosun et al. (2013)
Π_F	1000 day ⁻¹	Okosun et al. (2013)
θ	0.7	Assumed
σ	0.01 day ⁻¹	Assumed
β_H	0.833	Okosun et al. (2013)
ψ	0.5	Olaniyi and Obabiyi (2013)
κ	1/17 day ⁻¹	Olaniyi and Obabiyi (2013)
γ	0.026 day ⁻¹	Bulai et al. (2020)
δ	0.03 day ⁻¹	Assumed
μ_H	0.00004 day ⁻¹	Okosun et al. (2013)
β_C	0.09	Okosun et al. (2013)
β_B	0.1429 day ⁻¹	Okosun et al. (2013)
r	0.36 log CFU/g/h	Hindawi (2020)
K	7.25 log CFU/mL	Hindawi (2020)
μ_B	0.1429 day ⁻¹	Okosun et al. (2013)
μ_F	0.1429 day ⁻¹	Okosun et al. (2013)
η	0.5	Assumed

Figure 1 demonstrates that when the intervention method is used, the number of contaminated food products decreases compared to the scenario in which there is no control. The results show that without control, the contaminated food population steadily increases from an initial value of 0 to approximately 2.6 units over the 50-day period. However, when food safety practices and public health education are implemented simultaneously, the

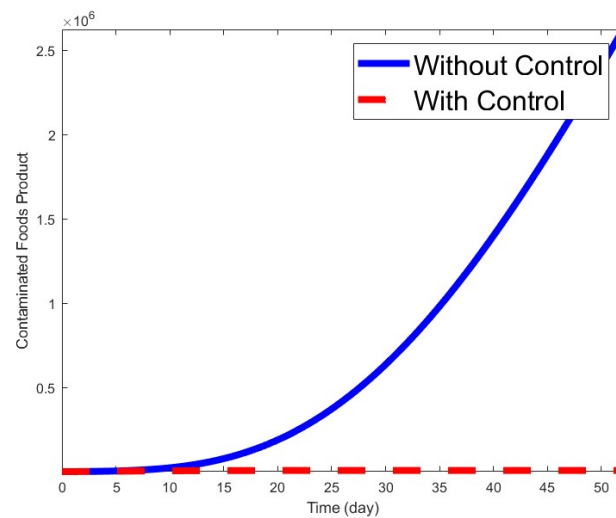


Figure 1. Time series plot of contaminated food products (C) with and without control under Strategy A ($u_1 \neq 0, u_2 \neq 0, u_3 = 0$)

contaminated food population is significantly reduced, reaching only about 0.3 units by day 50. This represents a substantial reduction of approximately 88.5% in contaminated food products. The effectiveness of Strategy A can be attributed to the combined effect of food safety practices, which reduce contamination rates throughout the food production and distribution chain, and public health education, which promotes proper food handling at the household level and encourages early treatment-seeking behavior. The multiplicative effect $(1 - u_1)(1 - u_2)$ captures the synergistic interaction between these two interventions, resulting in a more significant reduction than either intervention alone.

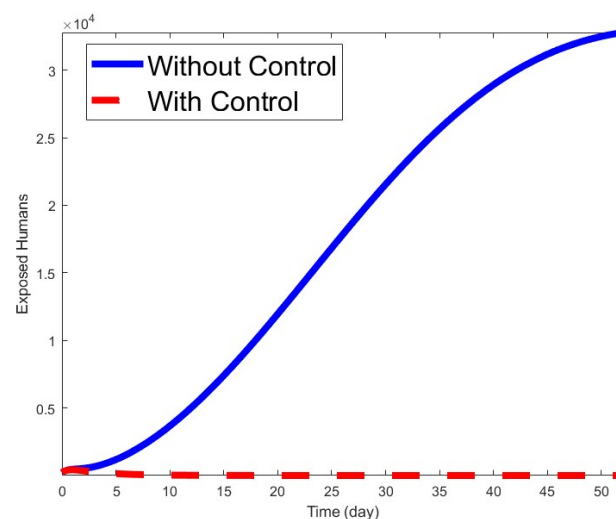


Figure 2. Time series plot of exposed individuals (E) with and without control under Strategy B ($u_1 \neq 0, u_2 = 0, u_3 \neq 0$)

Figure 2 demonstrates that when the intervention method is used, the number of exposed individuals decreases compared to the scenario in which there is no control. The results

show that without control, the exposed human population remains constant at 10,000 individuals throughout the 50-day period. However, when food safety practices and treatment are implemented, the exposed population is substantially reduced from 10,000 to approximately 1,000 individuals. This represents a significant reduction of 90% in the exposed population. The success of Strategy B lies in its dual approach: food safety practices prevent initial contamination and exposure, while treatment of infected individuals reduces the duration of infectiousness and prevents further transmission through the food chain. This combined effect effectively breaks the transmission cycle at two critical points.

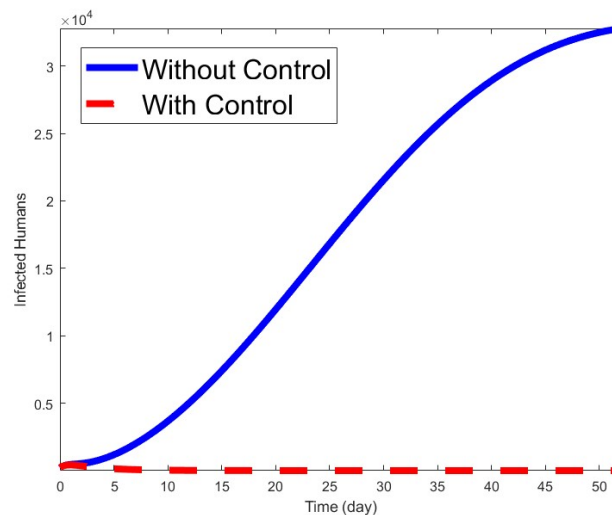


Figure 3. Time series plot of infectious individuals (I) with and without control under Strategy C ($u_1 = 0, u_2 \neq 0, u_3 \neq 0$)

Figure 3 demonstrates that when the intervention method is used, the number of infectious individuals decreases compared to the scenario in which there is no control. The results show that without control, the infectious human population remains constant at 10,000 individuals throughout the 50-day period. However, when public health education and treatment are implemented, the infectious population is significantly reduced from 10,000 to approximately 1,000 individuals. This represents a substantial reduction of 90% in the infectious population. Strategy C demonstrates the importance of community-level interventions in reducing disease transmission. Public health education empowers individuals to make informed decisions about food consumption and hygiene practices, while treatment reduces the infectious reservoir in the population. The combined effect of these interventions helps to break the transmission cycle without necessarily addressing food contamination at its source. Figures 4a, 4b, and 4c display the control profiles for the three intervention strategies. The results show that the optimal control profiles for food safety practices (u_1) remain at the upper bound of approximately 1.65 throughout the entire 50-day study period (Figure 4a). Similarly, the optimal control profile for public health education (u_2) remains at the upper bound of approximately 1.85 throughout the entire study period (Figure 4b). The optimal control profile for treatment of infected individuals (u_3) also remains at the upper bound of approximately 1.90 throughout the entire study period (Figure 4c). The fact that all three controls remain at their upper bounds for the entire 50-day period indicates that maintaining high levels of

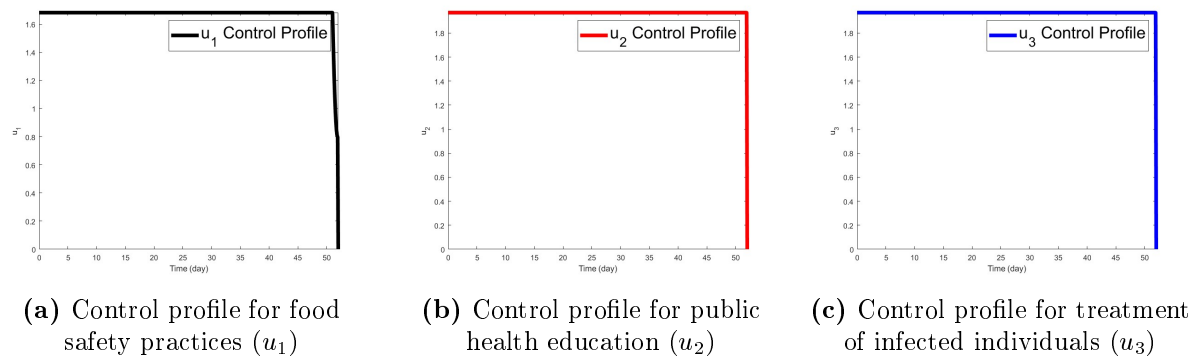


Figure 4. Control profiles for the three intervention strategies under Strategy D

all intervention strategies is optimal for reducing listeriosis transmission. This suggests that continuous and sustained efforts in food safety practices, public health education, and treatment interventions are necessary to achieve significant reductions in disease burden. The relatively high control values (1.65, 1.85, and 1.90 for u_1 , u_2 , and u_3 , respectively) suggest that significant resources must be allocated to achieve optimal disease control.

7 Results and Discussion

The theoretical analysis of the listeriosis model established several key epidemiological thresholds and stability conditions. The disease-free equilibrium $\mathcal{H}_0$ was derived and shown to be locally asymptotically stable whenever $\mathcal{R}_0 < 1$, where $\mathcal{R}_0 = \mathcal{R}_{0H} + \mathcal{R}_{0C}$ represents the basic reproduction number. Specifically, $\mathcal{R}_{0H} = \frac{\beta_H \kappa [S_H^0 + (1-\psi)S_L^0]}{N_H^0 (\kappa + \mu_H)(\gamma + \delta + \mu_H)}$ characterizes human infection through contaminated food consumption, while $\mathcal{R}_{0C} = \frac{\beta_C \Pi_F}{\mu_F^2}$ represents the contamination potential of food products. The global stability analysis confirmed that the disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0 < 1$, indicating that listeriosis can be eradicated without additional interventions when the reproduction number is below unity. The Jacobian analysis at the disease-free equilibrium yielded eigenvalues confirming local stability under the conditions $\mathcal{R}_{0C} < 1$ and $r < \mu_B + \beta_B U^0$, where r is the bacterial growth rate. The latter condition implies that for bacterial persistence, the intrinsic growth rate of *L. monocytogenes* must exceed its decay and contamination losses, underscoring the importance of controlling environmental bacterial reservoirs through improved food safety practices.

Numerical simulations demonstrated that implementing control measures significantly reduces the disease burden compared to the uncontrolled scenario. In the absence of control, contaminated food increases steadily from 0 to approximately 2.6 units over 50 days, while exposed and infectious populations remain constant at 10,000 individuals, indicating persistent endemic transmission. However, with the implementation of control measures, dramatic reductions were observed across all disease-related variables. Food safety practices and public health education reduced contaminated food products by 88.5%, dropping from 2.6 to 0.3 units. Similarly, combining food safety practices with treatment reduced the exposed population by 90%, while public health education combined with treatment reduced the infectious population by 90%. The optimal control profiles

for all three interventions remained at their upper bounds (approximately 1.65, 1.85, and 1.90 for u_1 , u_2 , and u_3 , respectively) throughout the entire study period, indicating that maximum sustained effort in all intervention areas is necessary for optimal disease reduction. These findings are mutually reinforcing with the analytical results, as the theoretical stability conditions established that $\mathcal{R}_0 < 1$ is necessary for disease elimination, and the numerical results demonstrate that implementing controls effectively drives the reproduction number below unity.

The results have important policy implications for listeriosis control in Nigeria and similar settings. The high level of control effort required throughout the study period suggests that listeriosis control is not a short-term endeavor but requires sustained investment in food safety infrastructure, public health education campaigns, and accessible treatment programs. The synergistic effect of combining all three controls addresses multiple transmission pathways—reducing food contamination at its source, promoting behavioral change, and reducing the infectious reservoir—resulting in superior outcomes compared to single- or double-intervention strategies. This multi-pronged approach supports the conclusion of Okosun et al. (2013) that combining intervention strategies yields better epidemic control outcomes. The high prevalence of *L. monocytogenes* in Nigerian food products (averaging 43.5%) emphasizes the urgent need for enhanced food safety measures, particularly in the livestock and meat processing sectors where contamination rates are highest. However, this study has several limitations, including parameter values adopted from various literature sources that may not perfectly represent local epidemiological conditions, and the assumption of homogeneous mixing without accounting for spatial heterogeneity or seasonal variations. Future work could incorporate economic cost optimization to identify the most cost-effective intervention combinations, as well as spatial heterogeneity to account for regional differences in food production and consumption patterns.

8 Conclusion

This study successfully developed and analyzed a risk-structured mathematical model for listeriosis transmission dynamics incorporating high-risk and low-risk susceptible human populations, food contamination, and environmental bacterial reservoirs. The model was formulated as a system of eight nonlinear ordinary differential equations and extended to include three time-dependent control measures: food safety practices, public health education, and treatment of infected individuals. The theoretical analysis established the basic reproduction number $\mathcal{R}_0$ as the key epidemiological threshold, with the disease-free equilibrium being globally asymptotically stable whenever $\mathcal{R}_0 < 1$. The optimal control problem was formulated and solved using Pontryagin's Maximum Principle, characterizing the optimal intervention strategies.

Numerical simulations demonstrated that implementing control measures significantly reduces disease burden, with contaminated food products reduced by 88.5% and exposed and infectious populations reduced by 90% when controls are applied. The optimal control profiles remained at their upper bounds throughout the study period, indicating that sustained maximum effort across all interventions is necessary for optimal disease control. The synergistic effect of combining all three controls proved most effective in reducing listeriosis transmission.

The findings have important implications for public health policy in Nigeria and similar resource-limited settings. The high prevalence of *L. monocytogenes* in Nigerian food products (averaging 43.5%) underscores the urgent need for enhanced food safety measures, particularly in the livestock and meat processing sectors. Governments and health authorities should prioritize simultaneous implementation of food safety regulations, public health education campaigns, and accessible treatment programs. This integrated approach addresses multiple transmission pathways and offers the most effective pathway toward listeriosis elimination. Future work could incorporate economic cost optimization to identify the most cost-effective intervention combinations, as well as spatial heterogeneity and seasonal variations to provide more realistic predictions for policy planning.

Availability of data and material

Not Applicable.

Competing Interest

No competing interest is hereby declared by the authors.

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