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MATHEMATICAL ANALYSIS OF A RISK STRUCTURED LISTERIOSIS DYNAMICS MODEL

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ABSTRACT

A foodborne disease called listeriosis is brought on by the bacteria Listeria monocytogenes which typically infects people after consuming contaminated food. Listeriosis mostly affects people with weakened immune systems, pregnant women and newborns. In this paper, we developed and analyzed a risk-structured mathematical model describing the dynamics of Listeriosis using ordinary differential equations. Three equilibrium points were obtained, viz; disease free equilibrium point, E_0^d, bacteria free equilibrium point, E_0^b, and endemic equilibrium point, E_0^e. Contaminated food threshold was established as R_L. The disease-free equilibrium and Bacteria-free equilibrium points are found to be locally asymptotically stable whenever the contaminated food threshold is less than unity ($R_L < 1$). Also, the endemic equilibrium point is found to be locally asymptotically stable using the Routh-Hurwitz criterion whenever the food safety index is less than unity ($R_c < 1$). Global stability analysis of the disease-free equilibrium point using Castillo-Chavez method revealed that the disease-free equilibrium point, E_0^d is globally asymptotically stable.

Keywords: Listeria, Bacteria, Equilibria, Stability

INTRODUCTION

Consuming contaminated food products can infect humans. On the other hand, though to a lesser degree, contact with infected humans or animals can also result in transmission. These illnesses can also be passed on to fetuses from infected pregnant women or female animals. The elderly, pregnant women, newborns, and those with weaker immune systems due to illnesses including HIV/AIDS, diabetes, cancer, and kidney disease are the groups most at risk of contracting this disease. The primary symptoms of listeriosis are fever, flu-like symptoms, vomiting, nausea, and diarrhea. Listeriosis can be prevented and treated, like other bacterial diseases, by properly preparing meat and poultry, avoiding keeping products in the refrigerator past their expiration date, and preventing raw food from coming into contact with other foods and equipment (Iwu & Okoh, 2020).

The Nigerian meat industry has been linked to the spread of Listeria throughout West Africa.

(Odu & Okonko, 2017L. monocytogenes was found to be 7% prevalent in raw meat samples in Rivers State, South-South Nigeria, and 91.8% prevalent in chicken flocks and meat in Oyo State, Nigeria.

(Ishola *et al.*, 2016). The prevalence of Listeria spp. in beef and chevon was found to be 58.2% (78/134) and 41.8% (56/134) in Lafia, Nigeria, respectively. It was determined that 64.4% (67/104) of these isolates were L. monocytogenes (Chukwu *et al.*, 2020).

The prevalence of Listeria spp. isolated from samples of beef, pork, and chicken meat in Enugu state, Nigeria, was 45.8%, 27.1%, and 13.2%, respectively (Odu & Okonko, 2017). L. monocytogenes was found in 4.0% of raw meat and meat products in Zaria, Nigeria (Ndahi *et al.*, 2014). According to other research conducted in Nigeria, the prevalence of L. monocytogenes in vegetables, such as tomatoes, cucumber, cabbage, carrots, and lettuce, was 28.28, 9.02, 23.36, 19.67, and 19.67%, respectively (Ajayeoba *et al.*, 2016). Furthermore, 78% of locally produced soft cheeses (wara) had Listeria spp. 12.4% of which were found to be L. monocytogenes (Kunadu *et al.*, 2018). According to this review, Nigeria's mean average prevalence of L. monocytogenes is 43.5%.

Osman *et al.*, (2020) examined a compartmental model of listeriosis including three humans and four animals. A qualitative analysis is conducted to determine whether the model's endemic and disease-free equilibria are stable, as well as whether forward and backward bifurcation is possible. Sensitivity analysis was employed to investigate the impact of altering the model parameters on the disease. The model includes treatment, immunization, and education of susceptible (human) populations as time-dependent control variables. In order to control listeriosis, they further employed Pontryagin's Maximum Principle and determined the most effective course of action. The model is simulated numerically, and the outcomes are shown graphically and quantitatively.

Osman *et al.*, (2018) studied the listeria epidemics in humans and animals with a focus on stability analysis. Chukwu *et al.*, (2020) developed a deterministic model of co-infection between listeriosis and meningitis. We look at the sub-models of meningitis exclusively and listeriosis solely. Every co-infection model and sub-model is examined mathematically. The severity parameters of infection co-dynamics are determined by Latin hypercube sampling. According to numerical models, co-infections between meningitis and listeriosis are decreased when ambient listeria bacteria are reduced and meningitis recovery rates are raised.

Cui *et al.*, (2007) developed a Susceptible Exposed Infected (SEI) model to examine how media affects infectious disease control. Numerical simulations conducted on this model demonstrate that while a lack of media notice can result in several disease outbreaks, its presence shortens the duration of the secondary peak in disease transmission.

MATERIALS AND METHODS

Model Formulation

The model consists of three sub-populations, namely; the human population, bacteria population and the food products. The total human population N_H is subdivided into four compartments; individuals at high risk of contracting

Listeriosis, H_S, individuals at low risk of contracting Listeriosis, L_S, infected individuals, I_H, and Recovered individuals (individuals who recover from the disease), R_H. The total human population, N_H, at any time t is thus given by

$N_H = H_S + L_S + I_H + R_H$

Susceptible human population is increased by recruitment at a constant rate Π_H. A portion z of these recruited individuals is at low risk of contracting the disease, while the rest are at high risk. The population of susceptible high-risk individuals decreases due to education/enlightenment efforts, when adopted by these high-risk individuals, reduce their risk and susceptibility to the disease, thereby reducing their population at a rate of χ. Infection with the Listeriosis disease from contaminated food products at a rate of λ_H and natural death at the rate of μ_H further diminish this population. Thus, the disease dynamics for high-risk susceptible individuals is given by

$(1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S$

For low-risk susceptible individuals, L_S, their population increases with education or enlightenment and subsequent behavioral changes due to high-risk susceptibility at a rate χ. It is decreased by natural death, μ_H, and rate at which low-risk individuals come in contact with Listeria disease at the rate $(1-\varphi)\lambda_H$, where φ is a modification parameter representing the behavioral dispositions adopted by these low-risk. This is mathematically expressed as

$z\Pi_H + \chi H_S - (\mu_H + (1-\varphi)\lambda_H)L_S$

The infected human population is increased due to infection with Listeriosis disease from high risk susceptible, low risk susceptible at a rate λ_H and $(1-\varphi)\lambda_H$ respectively. The population is further decreased due to natural death μ_H, death due to Listeriosis infection ϕ_H, and rate at which infected individuals received treatment from Listeria infection, α_H. Thus, it is given as

$\lambda_H H_S + (1-\varphi)\lambda_H L_S - (\mu_H + \phi_H + \alpha_H)I_H$

The recovered human population is increased due to treatment and decreased by natural death at a rate α_H and μ_H respectively. Thus, it is given as

$\alpha_H I_H - \mu_H R_H$

We let L_b to represents L. *monocytogenes* population with a net growth rate c_1, and a carrying capacity $0 \le d_L \le 1$. The Bacteria is assumed to grow logistically at a rate $c_1 L_b\left(1-\frac{L_b}{d_L}\right)$. The Bacteria population is reduced due to decay rate μ_L, so that

$c_1 L_b\left(1-\frac{L_b}{d_L}\right) - \mu_L L_b$

Food products are divided into two: we have uncontaminated food products, U_F, and contaminated food products, C_F; with the total food products $F = U_F + C_F$: production rate into uncontaminated food products is Π_F . Uncontaminated food is thus contaminated at a rate λ_F through contact with bacteria from the environment and contaminated food in the factory's handling as distribution processes and is reduced due to food products removal rate μ_F , so that

$\Pi_F - (\lambda_F + \mu_F)U_F$

Similarly, contaminated food products is increased due to contamination rate λ_F and is reduced by food products removal rate μ_F , so that

$\lambda_F U_F - \mu_F C_F$

With $\lambda_H = \beta_1 C_F$ and $\lambda_F = \beta_2 L_b + \beta_3 C_F$. Where: $\beta_1, \beta_2$and β_3 been the effective contact rates of contaminated foods with humans, uncontaminated foods with bacteria and contaminations of uncontaminated food caused by contaminated food products respectively. In general, the model formulated for this research is represented by equation (1), and the parameters and variables are listed in Table 1. A schematic diagram of the proposed model is shown in Figure 1.

$$\left.\begin{aligned}
\frac{dH_S}{dt} &= (1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S \\
\frac{dL_S}{dt} &= z\Pi_H + \chi H_S - (\mu_H + (1-\varphi)\lambda_H)L_S \\
\frac{dI_H}{dt} &= \lambda_H H_S + (1-\varphi)\lambda_H L_S - (\mu_H + \phi_H + \alpha_H)I_H \\
\frac{dR_H}{dt} &= \alpha_H I_H - \mu_H R_H \\
\frac{dL_b}{dt} &= c_1 L_b\left(1-\frac{L_b}{d_L}\right) - \mu_L L_b \\
\frac{dU_F}{dt} &= \Pi_F - (\lambda_F + \mu_F)U_F \\
\frac{dC_F}{dt} &= \lambda_F U_F - \mu_F C_F
\end{aligned}\right\} \quad (1)$$

where:

$\lambda_H = \beta_1 C_F$ and $\lambda_F = \beta_2 L_b + \beta_3 C_F$

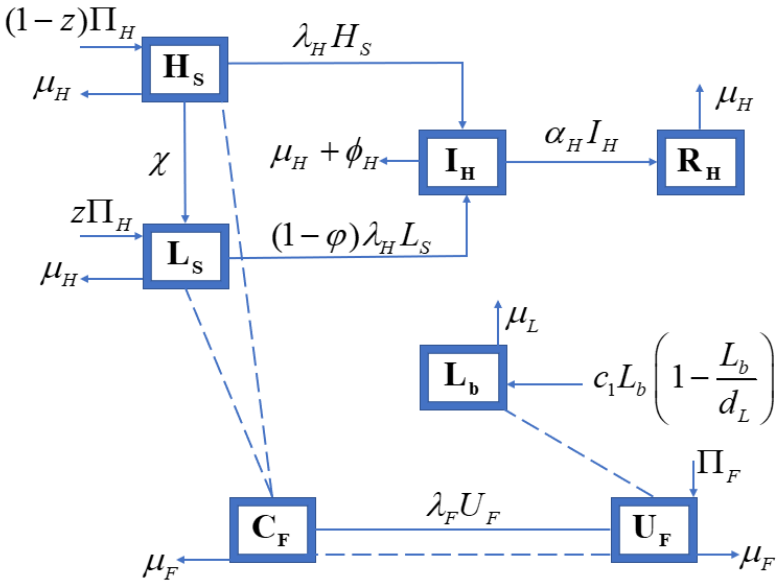

Figure 1: A Risk-Structured Listeriosis Model Diagram

Table 1: Description of the model variables and parameters

Variable/Parameters	Description
H_S	High-risk susceptible human population
L_S	Low-risk susceptible human population
I_H	Infected human population
R_H	Recovered human population
L_b	Bacteria *Monocytogenes* population
U_F	Uncontaminated food products
C_F	Contaminated food product
β_1	effective contact rate of contaminated foods with humans
β_2	effective contact rate of contaminated foods with bacteria
β_3	effective contact rate of uncontaminated foods with contaminated food products
Π_H	Human recruitment rate
Π_F	Food products production rate
z	Fraction of humans categorized to be low-risk individuals
χ	Education/ Enlightment campaign efforts
φ	Modification parameter for behavioral change of low-risk Susceptibles
$\mu_i (i = H, F, L)$	Human natural death/food product removal rate/bacteria decay rate
c_1	Bacteria growth rate
d_L	Bacteria population carrying capacity
α_H	Recovery rate for humans
ϕ_H	Listeria induced death rate for humans

Basic Properties of the Model

Boundedness of solutions

In this section, we proved that the solutions to model system (1) exists, 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 (1) contained in $\mathbb{R}_+^7$. The positivity of the solutions is governed by the following theorem.

Theorem 1: The closed set $\Omega = \Omega_H \times \Omega_F \times \Omega_L$, with $\Omega_H = \left\{H_S, L_S, I_H, R_H \in \mathbb{R}_+^4 : N_H \leq \frac{\Pi_H}{\mu_H}\right\}$, $\Omega_F = \left\{U_F, C_F \in \mathbb{R}_+^2 : F \leq \frac{\Pi_F}{\mu_F}\right\}$ and $\Omega_L = \{L_b \in \mathbb{R}_+^1\}$ is positively invariant with respect to model equations (1).

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

$N_H(t) = H_S(t) + L_S(t) + I_H(t) + R_H(t)$

and is differentiated and summed together to have

$\frac{N_H(t)}{dt} = \Pi_H - \mu_H N_H - \phi_H I_H$ (2)

in absence of mortality due to Listeria (i.e. $\phi_H = 0$) then by standard comparison theorem and rearranging equation (2), we obtain

$\frac{N_H(t)}{dt} + \mu_H N_H \leq \Pi_H$ (3)

solving equation (4) by integral factor method, we have

$N_H(t) \leq \frac{\Pi_H}{\mu_H}[1 - exp(-\mu_H t)] + N_H(0)\, exp(-\mu_H t)$ (4)

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

$N_H(t) \leq \frac{\Pi_H}{\mu_H}$ (5)

following a similar approach yields similar result for food products.

$F(t) \leq \frac{\Pi_F}{\mu_F}$ (6)

Furthermore, for the bacteria population, we have that

$L_b(t) = \left[\frac{c_1}{d_L(c_1 - \mu_L)} + D\, exp(-(\mu_L - c_1)t)\right]^{-1}$ (7)

thus, as $t \to \infty$, $L_b(t) \to d_L\left(1 - \frac{\mu_L}{c_1}\right)$. So, we note that for human Listeria to exists, its decay rate, μ_L must be less than its growth rate, c_1. Implying that $0 \leq L_b \leq d_L$. Thus, we have shown that Ω is positively invariant and attracts all solutions of model equation (1) in finite time. This guarantees that our investigation and analyses will be carried out in a feasible region and that every solution of our model having initial conditions in Ω will always remain in Ω for all time $t > 0$.

Non-negativity of solutions

Next, we establish that every solution of the model equation (1) will be non-negative for all time t.

Theorem 2: Let $(H_S(0), L_S(0), I_H(0), R_H(0), L_b(0), U_F(0), C_F(0))$ be the initial states of the model equation (1). Then every solution $(H_S(t), L_S(t), I_H(t), R_H(t), L_b(t), U_F(t), C_F(t)) \geq 0$ for all time $t > 0$.

Proof: From the first equation of our model equations (1), we have

$\frac{dH_S}{dt} = (1 - z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S$

So, by collecting like terms, we have

$\frac{dH_S}{dt} + (\lambda_H + \mu_H + \chi)H_S \geq (1 - z)\Pi_H$ (8)

solving equation (8) using integral factor method gives

$\frac{d}{dt}\left\{H_S\, exp\left[\mu_H t + \chi t + \int_0^t \lambda_H(\tau)d\tau\right]\right\}$

$\geq (1 - z)\Pi_H\, exp\left[\mu_H t + \chi t + \int_0^t \lambda_H(\tau)d\tau\right]$

integrating both sides, we have

$H_S(t)\, exp\left[\mu_H t + \chi t + \int_0^t \lambda_H(\tau)d\tau\right] - H_S(0)$

$\geq (1 - z)\Pi_H \int_0^t \left[exp\{(\mu_H + \chi)g + \int_0^g \lambda_H(\tau)d\tau\}\right] dg$

so that

$H_S(t) \geq H_S(0)\, exp\left[-\left\{(\mu_H + \chi)t + \int_0^t \lambda_H(\tau)d\tau\right\}\right] +$

$exp\left[-\left\{(\mu_H + \chi)t + \int_0^t \lambda_H(\tau)d\tau\right\}\right] \times$

$(1 - z)\Pi_H \int_0^t \left[exp\{(\mu_H + \chi)g + \int_0^g \lambda_H(\tau)d\tau\}\right] dg$

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

Consider the second equation of system (1)

$\frac{dL_S}{dt} = z\Pi_H + \chi H_S - \left((1 - \varphi)\lambda_H + \mu_H\right)L_S$ (9)

suppose $H_S > 0$, then (9) becomes

$\frac{dL_S}{dt} + \left((1 - \varphi)\lambda_H + \mu_H\right)L_S \geq z\Pi_H$

solving using integral factor method, we obtain

$\frac{d}{dt}\left\{L_S\, exp\left[\mu_H t + \int_0^t (1-\varphi)\lambda_H(\tau)d\tau\right]\right\}$
$\geq (1-z)\Pi_H\, exp\left[\mu_H t + \int_0^t (1-\varphi)\lambda_H(\tau)d\tau\right]$
Integrating both sides, we have
$L_S(t)\, exp\left[\mu_H t + \int_0^t (1-\varphi)\lambda_H(\tau)d\tau\right] - L_S(0) \geq$
$z\Pi_H \int_0^t \left[exp\{\mu_H g + \int_0^g (1-\varphi)\lambda_H(\tau)d\tau\}\right] dg$
so that
$L_S(t) \geq L_S(0)\, exp\left[-\left\{\mu_H t + \int_0^t (1-\varphi)\lambda_H(\tau)d\tau\right\}\right] +$
$exp\left[-\left\{\mu_H t + \int_0^t (1-\varphi)\lambda_H(\tau)d\tau\right\}\right] \times z\Pi_H \int_0^t \left[exp\{\mu_H g +$
$\int_0^g (1-\varphi)\lambda_H(\tau)d\tau\}\right] dg$
Thus, $L_S(t) > 0$ for $t > 0$, therefore, $L_S(t) > 0$ for $t > 0$. In the same manner, it can be shown that $I_H(t) > 0, R_H(t) > 0, L_b(t) > 0, U_F(t) > 0, C_F(t) > 0$.

Existence and uniqueness of solutions
Theorem 3: The model equation (1) defined by $\mathbb{R}_+^7 \in \Omega$ has a solution that exists, and the solution is unique in the region Ω if $\forall t \geq 0$.
$\left|\frac{\partial f_i}{\partial X}\right| < \infty, i = 1(1)7$ and $X = \{H_S, L_S, I_H, R_H, L_b, U_F, C_F\}$;
$|f(t, X^2) - f(t, X^1)| \leq M|X^2 - X^1|$, where M is Lipchitz constant.
Proof: We write the model equation (1) as
$f_1 = (1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S$
$f_2 = z\Pi_H + \chi H_S - \left((1-\varphi)\lambda_H + \mu_H\right)L_S$
$f_3 = \lambda_H H_S + (1-\varphi)\lambda_H L_S - (\alpha_H + \mu_H + \phi_H)I_H$
$f_4 = \alpha_H I_H - \mu_H R_H$
$f_5 = c_1 L_b\left(1 - \frac{L_b}{d_L}\right) - \mu_L L_b$
$f_6 = \Pi_F - (\lambda_F + \mu_F)U_F$
$f_7 = \lambda_F U_F - \mu_F C_F$
With
$\lambda_H = \beta_1 C_F$ and $\lambda_F = \beta_2 C_F + \beta_3 L_b$
taking the partial derivative of f_1 with respect to each state variable defined by X, we have
$\left|\frac{\partial f_1}{\partial H_S}\right| = |\lambda_H + \mu_H + \chi| < \infty,\ \left|\frac{\partial f_1}{\partial C_F}\right| = |\beta_1| < \infty$
and
$\left|\frac{\partial f_1}{\partial L_S}\right| = \left|\frac{\partial f_1}{\partial I_H}\right| = \left|\frac{\partial f_1}{\partial R_H}\right| = \left|\frac{\partial f_1}{\partial L_b}\right| = \left|\frac{\partial f_1}{\partial U_F}\right| = 0 < \infty$
repeating the same for other functions $f_2, f_3, \ldots, f_7$, 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 (1), we check each variable of X at these points to see if the system satisfies the Lipchitz condition, i.e. Consider;
$f_1 = (1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S$
this function at any two points of H_S is
$f_1 = (1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S^2$
$f_1 = (1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S^1$
therefore,
$\left|f_1(t, H_S^2) - f_1(t, H_S^1)\right| = \left|(1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S^2 - \left((1-z)\Pi_H - (\lambda_H + \mu_H + \chi)H_S^1\right)\right| \leq |-(\lambda_H + \mu_H + \chi)|\left|H_S^2 - H_S^1\right|$
where $M = (\lambda_H + \mu_H + \chi)$ and is Lipchitz constant. The process is repeated for other variables of $X \in f_1$ and functions, which establishes the Lipchitz condition. Hence, this completes the proof.

Model Steady States and Stability Analysis
Model Steady States
To solve for the steady states of model equation (1), we equate the RHS to zero as follows and obtain;

$$\left.\begin{aligned}
0 &= (1-z)\Pi_H - \left(\beta_1 C_F^* + \mu_H + \chi\right)H_S^* \\
0 &= z\Pi_H + \chi H_S^* - \left((1-\varphi)\beta_1 C_F^* + \mu_H\right)L_S^* \\
0 &= \beta_1 C_F^* H_S^* + (1-\varphi)\beta_1 C_F^* L_S^* - (\alpha_H + \mu_H + \phi_H)I_H^* \\
0 &= \alpha_H I_H^* - \mu_H R_H^* \\
0 &= c_1 L_b^*\left(1 - \tfrac{L_b^*}{d_L}\right) - \mu_L L_b^* \\
0 &= \Pi_F - \left(\beta_2 L_b^* + \beta_3 C_F^* + \mu_F\right)U_F^* \\
0 &= \left(\beta_2 L_b^* + \beta_3 C_F^*\right)U_F^* - \mu_F C_F^*
\end{aligned}\right\} \quad (10)$$

from the last three equations of (10), we can solve for the values of L_b^*, U_F^* and C_F^* as follows: From the fifth equation of (10), we have that;
$L_b^* = 0$ or $L_b^* = d_L\left(1 - \frac{\mu_L}{c_1}\right)$ (11)
Now, If $L_b^* = 0$, we can solve for U_F^* from second to the last equation of (10) as
$U_F^* = \frac{\Pi_F}{\beta_3 C_F^* + \mu_F}$ (12)
we substitute (12) into the last equation of (10) to obtain
$\beta_3 \Pi_F C_F^* - \beta_3 \mu_F C_F^{*2} - \mu_F^2 C_F^* = 0$ (13)
Simplifying (13), we obtain
$C_F^{*0} = 0$ or $C_F^{*1} = \frac{\mu_F}{\beta_3}(R_L - 1)$
where $R_L = \frac{\Pi_F \beta_3}{\mu_F^2}$ denotes the contaminated food threshold.

Disease-free Equilibrium state
The case when $L_b^* = 0$, and $C_F^{*0} = 0$ reduces system (10) to
$0 = (1-z)\Pi_H - (\mu_H + \chi)H_S^0$
$0 = z\Pi_H + \chi H_S^0 - \mu_H L_S^0$
$0 = \Pi_F - \mu_F U_F^0$
which gives the disease-free equilibrium state given as
$E_0^d = \left[\frac{(1-z)\Pi_H}{\mu_H + \chi}, \frac{\Pi_H(z\mu_H + \chi)}{\mu_H(\mu_H + \chi)}, 0, 0, 0, \frac{\Pi_F}{\mu_F}, 0\right]$

Bacteria-free Equilibrium state
The case when $L_b^* = 0$, $C_F^{*1} = \frac{\mu_F}{\beta_3}(R_L - 1)$ and $R_L > 1$, system (10) reduces to
$0 = (1-z)\Pi_H - \left(\frac{\beta_1 \mu_F}{\beta_3}(R_L - 1) + \mu_H + \chi\right)H_S^*$
$0 = z\Pi_H + \chi H_S^* - \left((1-\varphi)\frac{\beta_1 \mu_F}{\beta_3}(R_L - 1) + \mu_H\right)L_S^*$
$0 = \frac{\beta_1 \mu_F}{\beta_3}(R_L - 1)H_S^* + (1-\varphi)\frac{\beta_1 \mu_F}{\beta_3}(R_L - 1)L_S^* - (\alpha_H + \mu_H + \phi_H)I_H^*$
$0 = \alpha_H I_H^* - \mu_H R_H^*$
$0 = \Pi_F - (\mu_F(R_L - 1) + \mu_F)U_F^*$

this gives the bacteria-free equilibrium state given as
$E_0^b = \left[H_S^*, L_S^*, I_H^*, R_H^*, 0, \frac{\mu_F}{\beta_3}, \frac{\mu_F}{\beta_3}(R_L - 1)\right]$
where:
$H_S^* = \frac{(1-z)\beta_3 \Pi_H}{\beta_1 \mu_F (R_L - 1) + \beta_3(\mu_H + \chi)}$,
$L_S^* = \frac{\beta_3 z\Pi_H + \beta_3 \chi H_S^*}{\beta_1 \mu_F (1-\varphi)(R_L - 1) + \beta_3 \mu_H}$
$I_H^* = \frac{(\beta_1 \mu_F H_S^* + \mu_F(1-\varphi)L_S^*)(R_L - 1)}{\beta_3(\alpha_H + \mu_H + \phi_H)}$,
$R_H^* = \frac{(\beta_1 \mu_F \alpha_H H_S^* + \alpha_H \mu_F (1-\varphi)L_S^*)(R_L - 1)}{\beta_3 \mu_H(\alpha_H + \mu_H + \phi_H)}$

Endemic Equilibrium state
If $L_b^* = d_L\left(1 - \frac{\mu_L}{c_1}\right)$ then we obtain the Listeria endemic equilibrium express as
$E_0^e = \left[H_S^{**}, L_S^{**}, I_H^{**}, R_H^{**}, d_L\left(1 - \frac{\mu_L}{c_1}\right), U_F^{**}, C_F^{**}\right]$

where:

$H_S^{**} = \frac{(1-z)\Pi_H c_1(\mu_F-\beta_2 U_F^{**})}{\beta_1\beta_3 d_L(c_1-\mu_L)U_F^{**}+c_1(\mu_H+\chi)(\mu_F-\beta_2 U_F^{**})}$,

$L_S^{**} = \frac{z\Pi_H(\beta_1 C_F^{**}+\mu_H+\chi)+\Pi_H\chi(1-z)}{\left((1-\varphi)\beta_1 C_F^{**}+\mu_H\right)(\beta_1 C_F^{**}+\mu_H+\chi)}$

$I_H^{**} = \frac{\beta_1\beta_3 d_L(c_1-\mu_L)(H_S^{**}+(1-\varphi)L_S^{**})U_F^{**}}{c_1(\mu_F-\beta_2 U_F^{**})(\alpha_H+\mu_H+\phi_H)}$,

$R_H^{**} = \frac{\alpha_H\beta_1\beta_3 d_L(c_1-\mu_L)(H_S^{**}+(1-\varphi)L_S^{**})U_F^{**}}{\mu_H c_1(\mu_F-\beta_2 U_F^{**})(\alpha_H+\mu_H+\phi_H)}$,

$U_F^{**} = \frac{\Pi_H c_1}{c_1\beta_2 C_F^{**}+\beta_3 d_L(c_1-\mu_L)+c_1\mu_L}$,

$C_F^{**} = \frac{\beta_3 d_L(c_1-\mu_L)U_F^{**}}{c_1(\mu_F-\beta_2 U_F^{**})}$

Local Stability of Disease-free equilibrium state

The Disease-free equilibrium point E_0^d is locally asymptotically stable whenever $c_1 < \mu_L$, $R_L < 1$ and unstable otherwise.

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

$$J(E_0^d) = \begin{bmatrix} -A_1 & 0 & 0 & 0 & 0 & 0 & -\beta_1 H_S^0 \\ \chi & -\mu_H & 0 & 0 & 0 & 0 & -(1-\varphi)\beta_1 L_S^0 \\ 0 & 0 & -A_2 & 0 & 0 & 0 & \beta_1 H_S^0 + (1-\varphi)\beta_1 L_S^0 \\ 0 & 0 & \alpha_H & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & c_1-\mu_L & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_3 U_F^0 & -\mu_F & -\beta_2 U_F^0 \\ 0 & 0 & 0 & 0 & \beta_3 U_F^0 & 0 & \beta_2 U_F^0 - \mu_F \end{bmatrix} \quad (14)$$

where:

$A_1 = (\chi + \mu_H)$ and $A_2 = (\alpha_H + \mu_H + \phi_H)$

The eigenvalues of (14) is given as:

$\lambda_1 = -(\chi + \mu_H)$,

$\lambda_2 = -\mu_H$,

$\lambda_3 = -(\alpha_H + \mu_H + \phi_H)$,

$\lambda_4 = -\mu_H$,

$\lambda_5 = c_1 - \mu_L$,

$\lambda_6 = -\mu_F$ and

$\lambda_7 = \mu_F(R_L - 1)$

Hence, the eigenvalues of (14) have negative real parts whenever $c_1 < \mu_L$ and $R_L < 1$.

Local Stability of Bacteria-free equilibrium state

The Bacteria-free equilibrium point E_0^b is locally asymptotically stable whenever $R_L < 1, c_1 < \mu_L$ and unstable otherwise.

Proof: The Jacobian of system of equation (1) evaluated at bacteria-free equilibrium is given as

$$J(E_0^b) = \begin{bmatrix} Q_0 & 0 & 0 & 0 & 0 & 0 & -\beta_1 H_S^* \\ \chi & Q_1 & 0 & 0 & 0 & 0 & -(1-\varphi)\beta_1 L_S^* \\ Q_0 & Q_1 & -Q_2 & 0 & 0 & 0 & \beta_1 H_S^* + (1-\varphi)\beta_1 L_S^* \\ 0 & 0 & \alpha_H & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & c_1-\mu_L & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_3 U_F^* & -Q_3 & -\beta_2 U_F^* \\ 0 & 0 & 0 & 0 & \beta_3 U_F^* & Q_2 & \beta_2 U_F^* - \mu_F \end{bmatrix} \quad (15)$$

where:

$Q_0 = \frac{\beta_1\mu_F}{\beta_3}(R_L - 1) - (\chi + \mu_H)$,

$Q_1 = \frac{(1-\varphi)\beta_1\mu_F}{\beta_3}(R_L - 1) - \mu_H$

$Q_2 = (\alpha_H + \mu_H + \phi_H)$

$Q_3 = \frac{\beta_2\mu_F}{\beta_3}(R_L - 1) - \mu_F$,

$Q_4 = \frac{\beta_2\mu_F}{\beta_3} - \mu_F$ (16)

The eigenvalues of (15) are:

$\lambda_1 = -\mu_H$,

$\lambda_2 = c_1 - \mu_L$,

$\lambda_3 = -(\alpha_H + \mu_H + \phi_H)$,

$\lambda_4 = \frac{(1-\varphi)\beta_1\mu_F}{\beta_3}(R_L - 1) - \mu_H$ and

$\lambda_5 = \frac{\beta_1\mu_F}{\beta_3}(R_L - 1) - (\chi + \mu_H)$

The remaining eigenvalues can be obtained from the solutions to the characteristic's polynomial of the sub-matrix

$$J_1(E_0^b) = \begin{bmatrix} -Q_3 & -\beta_2 U_F^* \\ Q_2 & \beta_2 U_F^* - \mu_F \end{bmatrix}$$

given by

$\lambda^2 + \theta_0\lambda + \theta_1 = 0$ (17)

in which

$\theta_0 = \frac{\beta_2\mu_F}{\beta_3}(R_L - 1) + 2\mu_F - \frac{\beta_2\mu_F}{\beta_3}$ and

$\theta_1 = \frac{\beta_2\mu_F^2}{\beta_3}(R_L - 1) - \frac{\mu_F^2\beta_2}{\beta_3} + \mu_F^2$

We have that θ_1 and θ_0 are positive if $R_L > 1$. Hence, by Routh-Hurwitz stability criterion, we have that all the eigenvalues of the polynomial (17) have negative real parts. Thus, E_0^b is locally asymptotically stable if and only if $c_1 < \mu_L$ and unstable otherwise.

Local Stability of Endemic equilibrium state

The Endemic equilibrium point E_0^e is locally asymptotically stable whenever contamination control ratio (food safety index) is less than unity ($R_c < 1$) and $c_1 > \mu_L$, and unstable otherwise.

Proof: We define the contamination control ratio (food safety index), $R_c = \frac{U_F^{**}}{C_F^{**}}$ and thus the Jacobian of system of equation (1) evaluated at endemic equilibrium is given as

$$J(E_0^e) = \begin{bmatrix} -Q_5 & 0 & 0 & 0 & 0 & 0 & -\beta_1 H_S^* \\ \chi & -Q_6 & 0 & 0 & 0 & 0 & -B_1 \\ \beta_1 C_F^{**} & (1-\varphi)\beta_1 C_F^{**} & -Q_7 & 0 & 0 & 0 & B_2 \\ 0 & 0 & \alpha_H & -\mu_H & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_L - c_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\beta_3 U_F^{**} & -Q_8 - \mu_F & -\beta_2 U_F^{**} \\ 0 & 0 & 0 & 0 & \beta_3 U_F^{**} & Q_8 & \beta_2 U_F^{**} - \mu_F \end{bmatrix} \quad (18)$$

where:

$Q_5 = -(\beta_1 C_F^{**} + \chi + \mu_H)$,

$Q_6 = -(1-\varphi)\beta_1 C_F^{**} - \mu_H$,

$Q_7 = -(\alpha_H + \mu_H + \phi_H)$,

$Q_8 = \beta_2 C_F^{**} + d_L\beta_3\left(1 - \frac{\mu_L}{c_1}\right)$

The eigenvalues of equation (18) are $\lambda_1 = \mu_L - c_1, \lambda_2 = -\mu_H, \lambda_3 = -(\alpha_H + \mu_H + \phi_H)$and the solutions to the characteristic polynomial obtained from the sub-matrix

$$J_1(E_0^e) = \begin{bmatrix} -(\beta_1 C_F^{**} + \chi + \mu_H) & 0 \\ \chi & -(1-\varphi)\beta_1 C_F^{**} - \mu_H \end{bmatrix}$$

given as

$\lambda^2 + \theta_2\lambda + \theta_3 = 0$ (19)

where

$\theta_2 = (1-\varphi)\beta_1 C_F^{**} + \beta_1 C_F^{**} + 2\mu_H + \chi > 0$,

$\theta_3 = \beta_1\mu_H C_F^{**} + (1-\varphi)\beta_1^2 C_F^{**2} + \mu_H(\chi + \mu_H) + \beta_1 C_F^{**}(1-\varphi)(\mu_H + \chi) > 0$

The remaining eigenvalues are determined from determinant of the sub-matrix $J_2(E_0^e)$ below

$$J_2(E_0^e) = \begin{bmatrix} -\left(\beta_2 C_F^{**} + \mu_F + \beta_3 d_L\left(1 - \frac{\mu_L}{c_1}\right)\right) & -\beta_2 U_F^{**} \\ \beta_2 C_F^{**} + \beta_3 d_L\left(1 - \frac{\mu_L}{c_1}\right) & \beta_2 U_F^{**} - \mu_H \end{bmatrix}$$

Given by the characteristics polynomial

$$\lambda^2 + \theta_4\lambda + \theta_5 = 0 \quad (20)$$

where:

$\theta_4 = \frac{\beta_2 c_1 C_F^{**}(1-R_c)+\beta_3 d_L(c_1-\mu_L)+2c_1\mu_F}{c_1}$

$\theta_5 = \frac{\beta_2 c_1 \mu_F C_F^{**}(1-R_c)+\beta_3 \mu_F d_L(c_1-\mu_L)+c_1\mu_F^2}{c_1}$

Clearly, $\theta_4 > 0$, and $\theta_5 > 0$ if and only if $c_1 > \mu_L$ and $R_c < 1$. This implies that, there must be more contaminated food products than uncontaminated for Listeriosis to be endemic. Hence, by the principle of Routh-Hurwitz stability condition, all the eigenvalues of (19) and (20) have negative real parts.

Global Stability of Disease-free equilibrium state

To prove the global asymptotic stability (GAS) of the disease-free equilibrium E_0^d for the model equations (1) using the conditions of Castillo-Chavez et al., (2002) as used in Danjuma *et al.*, (2024). First, our model system (1) is written in the form

$$\left.\begin{aligned}\frac{dQ}{dt} &= F(Q,W),\\ \frac{dW}{dt} &= G(Q,W), G(Q,0) = 0\end{aligned}\right\} \quad (21)$$

Where $Q = (H_S, L_S, R_H, U_F) \in \mathbb{R}^m$ represents the number of uninfected individuals $W = (I_H, L_b, C_F) \in \mathbb{R}^n$ denotes the number of infected population and $E_0^d = (Q^*, 0)$ represents the disease-free equilibrium. The following assumptions must be satisfied for disease-free equilibrium of system (1) to be globally asymptotically stable:

$H_1 = \frac{dQ}{dt} = F(Q^*, 0), Q^*$ is globally asymptotically stable.

$H_2 = G(Q,W) = AW - \hat{G}(Q,W)$ where $\hat{G}(Q,W) \geq 0$ for $(Q,W) \in \Omega$, and $A = D_w G(Q^*, 0)$ is an M-matrix (the off-diagonal elements are nonnegative).

Theorem 3: The equilibrium point, E_0^d of the system (1) is globally asymptotically stable if $R_L < 1$ (locally asymptotically stable) and the assumptions H_1 and H_2 are satisfied.

Proof: Rewrite the model equations (1) in the form of (21) as

$$F(Q,W) = \begin{bmatrix}(1-z)\Pi_H - (\lambda_H + \mu_H)H_S - \chi H_S\\ z\Pi_H + \chi H_S - ((1-\varphi)\lambda_H + \mu_H)L_S\\ \alpha_H I_H - \mu_H R_H\\ \Pi_F - (\lambda_F + \mu_F)U_F\end{bmatrix}$$

Which gives the reduced system

$$F(Q,0) = \begin{bmatrix}(1-z)\Pi_H - (\chi + \mu_H)H_S\\ z\Pi_H + \chi H_S - \mu_H L_S\\ 0\\ \Pi_F - \mu_F U_F\end{bmatrix} \quad (22)$$

Consider the reduced system (22), solving for H_S we have

$\frac{dH_S}{dt} = (1-z)\Pi_H - (\chi + \mu_H)H_S$

$= -(\chi + \mu_H)\left(H_S - \frac{(1-z)\Pi_H}{\mu_H + \chi}\right)$

which gives that

$ln\,|H_S - \frac{(1-z)\Pi_H}{\mu_H+\chi}| = exp[-(\chi+\mu_H)t] + C$

Simplifying gives

$H_S(t) = \frac{(1-z)\Pi_H}{\mu_H+\chi} + \left(H_S(0) - \frac{(1-z)\Pi_H}{\mu_H+\chi}\right)exp[-(\chi+\mu_H)t]$ (22a)

Similarly, solving for L_S we have

$\frac{dL_S}{dt} = z\Pi_H + \chi H_S - \mu_H L_S$

$= -\mu_H\left(L_S - \frac{z\Pi_H + \chi H_S}{\mu_H}\right)$

which gives that

$ln\,|L_S - \frac{z\Pi_H + \chi H_S}{\mu_H}| = exp[-\mu_H t] + C$

Simplifying gives

$L_S(t) = \frac{z\Pi_H + \chi H_S}{\mu_H} + \left(L_S(0) - \frac{z\Pi_H + \chi H_S}{\mu_H}\right)exp[-\mu_H t]$ (22b)

Also, solving for U_F we have

$\frac{dU_F}{dt} = \Pi_F - \mu_F U_F$

$= -\mu_F\left(U_F - \frac{\Pi_F}{\mu_F}\right)$

which gives that

$ln\,|U_F - \frac{\Pi_F}{\mu_F}| = exp[-\mu_F t] + C$

Simplifying gives

$U_F(t) = \frac{\Pi_F}{\mu_F} + \left(U_F(0) - \frac{\Pi_F}{\mu_F}\right)exp[-\mu_F t]$ (22c)

as $t \to \infty$, $E_0^d = [H_S^0, L_S^0, R_H^0, U_F^0] \to \left[\frac{(1-z)\Pi_H}{\mu_H+\chi}, \frac{\Pi_H(z\mu_H+\chi)}{\mu_H(\mu_H+\chi)}, 0, \frac{\Pi_F}{\mu_F}\right]$ regardless of the initial conditions. Hence, the first condition H_1 holds. Now for the second condition, we compute the matrix A as $A = \frac{\partial G}{\partial Z}[E_0^d, 0]$, now,

$$G = \begin{bmatrix}\beta_1 C_F H_S + (1-\varphi)\beta_1 C_F L_S - (\alpha_H + \mu_H + \phi_H)I_H\\ c_1 L_b\left(1 - \frac{L_b}{d_L}\right) - \mu_L L_b\\ (\beta_2 L_b + \beta_3 C_F)U_F - \mu_F C_F\end{bmatrix}$$

So that

$$A = \frac{\partial G}{\partial Z}[E_0^d, 0] = \begin{bmatrix}-(\alpha_H + \mu_H + \phi_H) & 0 & \beta_1 H_S^0 + (1-\varphi)\beta_1 L_S^0\\ 0 & c_1 - \mu_L & 0\\ 0 & \beta_2 U_F^0 & \beta_3 U_F^0 - \mu_F\end{bmatrix}$$

We observe that matrix A is an M-matrix since all its off diagonal are non-negative. We compute AZ as

$$AZ = A \times \begin{bmatrix}I_H\\ L_b\\ C_F\end{bmatrix} \Rightarrow AZ =$$

$$\begin{bmatrix}-(\alpha_H + \mu_H + \phi_H) & 0 & \beta_1 H_S^0 + (1-\varphi)\beta_1 L_S^0\\ 0 & c_1 - \mu_L & 0\\ 0 & \beta_2 U_F^0 & \beta_3 U_F^0 - \mu_F\end{bmatrix}\begin{bmatrix}I_H\\ L_b\\ C_F\end{bmatrix}$$

$$= \begin{bmatrix}-(\alpha_H + \mu_H + \phi_H)I_H + \beta_1 C_F H_S^0 + (1-\varphi)\beta_1 C_F L_S^0\\ (c_1 - \mu_L)L_b\\ \beta_2 U_F^0 L_b + (\beta_3 U_F^0 - \mu_F)C_F\end{bmatrix}$$

Now, $G(Q,W) = AW - \hat{G}(Q,W)$, then;

$$\hat{G}(Q,W) = \begin{bmatrix}\beta_1 C_F\left[1 - \frac{H_S}{H_S^0}\right]H_S^0 + (1-\varphi)\beta_1 C_F\left[1 - \frac{L_S}{L_S^0}\right]L_S^0\\ c_1\frac{L_b^2}{d_L}\\ \beta_2 L_b\left[1 - \frac{U_F}{U_F^0}\right]U_F^0 + \beta_3 C_F\left[1 - \frac{U_F}{U_F^0}\right]U_F^0\end{bmatrix}$$

Since, $H_S \leq H_S^0, L_S \leq L_S^0$ and $U_F \leq U_F^0$, then, $\hat{G}(Q,W)$ is non-negative, i.e. the second condition H_2 also holds. Thus, the disease-free equilibrium, E_0^d is GAS.

CONCLUSION

In this work, we adopted an SIR-based deterministic model for the Listeriosis dynamics to study the transmission dynamics of Listeriosis. The model consists of a non-linear ordinary 7-dimensional system of ordinary differential equations. Basic properties of the model are established and showed that the proposed model is mathematically and epidemiologically well-posed. Three equilibrium points were obtained; disease free equilibrium point, E_0^d, bacteria free equilibrium point, E_0^b, and endemic equilibrium point, E_0^e. The local stability analyses of the disease-free and Bacteria-free equilibrium points were conducted and analyses showed that the disease free and bacteria free equilibrium points are locally asymptotically stable whenever the contaminated food threshold is found to be less than unity ($R_L < 1$) and $c_1 < \mu_L$. In a similar way, the endemic equilibrium point, E_0^e, is found to be locally asymptotically stable whenever $R_c < 1$ and $c_1 >$

μ_L. Also, the global stability analyses of the disease-free equilibrium point, E_0^d was conducted using Castillo-Chavez method and result shows that the disease-free equilibrium point is globally asymptotically stable.

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