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Research article

Modelling the potential role of media campaigns on the control of Listeriosis

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Abstract: Human Listeria infection is a food-borne disease caused by the consumption of contaminated food products by the bacterial pathogen, Listeria. In this paper, we propose a mathematical model to analyze the impact of media campaigns on the spread and control of Listeriosis. The model exhibited three equilibria namely; disease-free, Listeria-free and endemic equilibria. The food contamination threshold is determined and the local stability analyses of the model is discussed. Sensitivity analysis is done to determine the model parameters that most affect the severity of the disease. Numerical simulations were carried out to assess the role of media campaigns on the Listeriosis spread. The results show that; an increase in the intensity of the media awareness campaigns, the removal rate of contaminated food products, a decrease in the contact rate of Listeria by humans results in fewer humans getting infected, thus leading to the disease eradication. An increase in the depletion of media awareness campaigns results in more humans being infected with Listeriosis. These findings may significantly impact policy and decision-making in the control of Listeriosis disease.

Keywords: media campaigns; infectious disease; Listeria; numerical simulations; food contamination threshold

1. Introduction

Listeriosis is a serious and severe food-borne disease that affects the human population globally. The disease is caused by a bacteria called *Listeria monocytogenes* which exists in the environment as its primary host (soil, water, ready-to-eat (RTE) foods and contaminated food products) [1, 2]. Human beings contract Listeriosis through the ingestion of contaminated RTE food products such as

cantaloupes, meat, Ricotta Salata cheese, vegetables, polony, bean sprouts, ham, or directly from the environment [3, 4]. The epidemiology of Listeriosis is clearly articulated in [1, 4].

Before the 2017 outbreak in South Africa, an average of 60 to 80 confirmed Listeriosis disease cases were recorded annually (i.e., approximately 1 case per week). The recent outbreak in South Africa, which occurred from 1 January 2017 to 17 July 2018, had 1060 confirmed cases, with 216 (26.8%) deaths. This was the world's largest-ever documented Listeriosis outbreak [5]. The source of the disease was traced to be contaminated RTE processed meat products. Also, Listeriosis outbreaks resulting from human consumption of different kinds of contaminated RTE food products occur commonly in the United States of America, Canada and Europe [3].

The media campaign is a series of advertisement messages that share a single idea, beliefs, concepts, and theme, which make up an integrated marketing communication over a particular time frame and target identified audiences [6]. In addition, media campaigns and media-driven awareness programs such as print media, social media, internet, television, radio, and advertisements play an essential role in the disseminating of information about the spread of infectious disease outbreaks [7]. Dissemination of information educates people and helps them take preventive measures such as; practicing better hygiene; factory workers wearing clean gloves to avoid cross-contamination of food products during food production/manufacturing. Further, when a disease breaks out in a human population, changes in behavior in response to the outbreak can alter the progression of the infectious agent. In particular, people aware of a disease in their proximity can take precautionary measures to reduce their susceptibility to infections by isolating a portion of the susceptible population from the infected ones [8].

In recent times, researchers have used mathematical models to model infectious and non-infectious diseases/describe the effect and impact of media campaigns on the dynamics of infectious such as Ebola [9], HIV/AIDS [10, 11], Avian Influenza [12], Listeriosis [13], and vector-host disease [14, 15]. Misra et al. [16] used a non-linear mathematical model which assumed that due to awareness programs by media, once the population becomes aware of the disease spread, they avoid contact with the infectives and therefore form a new class of individuals called the aware class, who become susceptible again if their awareness wanes over time. The model analysis revealed that the number of infectives decreases with an increase in media campaigns. Kaur [17] extended the work by Misra by assuming that aware susceptibles do not lose awareness but can also interact with infected individuals and get infected, albeit at a lower rate. Their study suggested that with the increase in the rate of implementation of awareness programs via media, there is a subsequent decline in the number of infected in any targeted population under consideration. Authors in [9] used a mathematical model to describe the transmission dynamics of Ebola in the presence of asymptomatic cases and the impact of media campaigns on the disease transmission was represented by a linearly decreasing function. Their results showed that messages sent through media have a more significant effect on reducing Ebola cases if they are more effective and spaced out. The $SIRS$ model was proposed in [18] to investigate the impact of awareness programs by considering private and public awareness, which reduces the contact rate between unaware and aware populations and the effect/impact of public information campaigns on disease prevalence. It was shown that both private and public awareness could reduce the size of epidemic outbreaks. A smoking cessation model with media campaigns was presented [19]. The results showed that the reproduction number was suppressed when media campaigns that focus on smoking cessation were increased. Thus, spreading information to encourage smokers to quit smoking was an effective intervention. According to [20], the $SIRS$ model was used to analyze the role of information and limited optimal treatment on disease

prevalence. The model considered the growth rate of information proportional to a saturated function of infected individuals. The results from the mathematical analysis showed that the combined effects of information and treatment is more effective and economical in the control of the infection. Exponential functions have also been used to model the impact of media awareness campaigns on people's behavior, which affects the evolution of infectious diseases. In particular, the effects of Twitter messages on reducing the transmission rate of the influenza virus was studied in [21]. The result revealed that Twitter messages had a substantial influence on the dynamics of influenza disease spread.

To date, there are very few mathematical models on the dynamics of Listeriosis (see, for instance, [22–25]), let alone those investigating the potential role of media awareness campaigns on the dynamics of Listeriosis. This paper is motivated by the work done in [16]. We formulate a mathematical model to study the impact and effects of media campaigns on the dynamics of Listeriosis disease resulting from the consumption of contaminated RTE in the human population. We describe the model in detail in the following section.

The outline of this paper is as follows; Section 1 introduces the research paper followed by the model described in Section 2. The model basic properties and analyses are presented in Section 3. Numerical simulations were done and presented in Section 4. Section 5 concludes the paper.

2. Model description

The human population is divided into four sub-classes, viz: Susceptibles $S_h(t)$, aware susceptibles $S_a(t)$, the infected $I_h(t)$ and the recovered $R_h(t)$. Individuals are recruited at a rate proportional to the size of the human population $N(t)$ where

$$N(t) = S_h(t) + S_a(t) + I_h(t) + R_h(t).$$

The recruitment rate is given by $\mu_h N(t)$ where μ_h is natural birth/mortality rate. Upon infection with Listeria from contaminated food, the susceptibles move into the infectious class $I_h(t)$ with a force of infection $\lambda_h(t)$, where $\lambda_h(t) = \beta_{f_1} F_c$ with β_{f_1} being the rate at which humans gets infected and $F_c(t)$ the contaminated food products. Here, $\lambda_h(t)$ describes the force of infection by the consumption of contaminated food products. The susceptible individuals can also move to the awareness class at a rate ρ as a result of the interaction with the media campaigns. We assume that media campaigns wane over time and the aware individuals can revert to being susceptible again at a rate ω_1. The infected individuals recover at a rate γ with immunity after treatment. These individuals who recover after some time can also lose their immunity and become susceptible again at a rate δ_h. We assume a constant human population $N(t)$, which consists of individuals who do not work in the factory over the modelling time. Further, we assume that aware individuals cannot be infected as their awareness protects them from contracting the disease. Given that the bacteria survive even at 4^0C, it can die or grow in its host or the environment at significantly low temperatures. Let r_l and ξ denote the growth and removal rate of the of Listeria, $L(t)$. Our model assumes a logistic growth of Listeria with carrying capacity K_L. The non-contaminated food products $F_n(t)$ can be contaminated as a result of interaction with the bacteria from the environment that comes, via the workers, exchange of gloves or utensils during food manufacturing and also through the contact with contaminated food $F_c(t)$ with a force of infection $\lambda_f(t)$, where

$$\lambda_f(t) = \beta_L L(t) + \beta_{f_2} F_c(t).$$

The parameters β_L and β_{f_2} are the contact rate of Listeria and the contamination rate of non-contaminated food by contaminated food products, respectively. The contaminated food products are then responsible for transmitting Listeriosis disease to the human population through ingestion of the contaminated food products. The total amount of food products, $F(t)$, at any given time is given by

$$F(t) = F_n(t) + F_c(t),$$

where μ_f is the rate of removal of food products through consumption. Let M_a be the cumulative density of media campaigns with maximum intensity, M, at which media awareness campaigns are implemented, π_0 the rate of implementation of the media awareness campaigns and μ_0 the rate of depletion of media awareness.

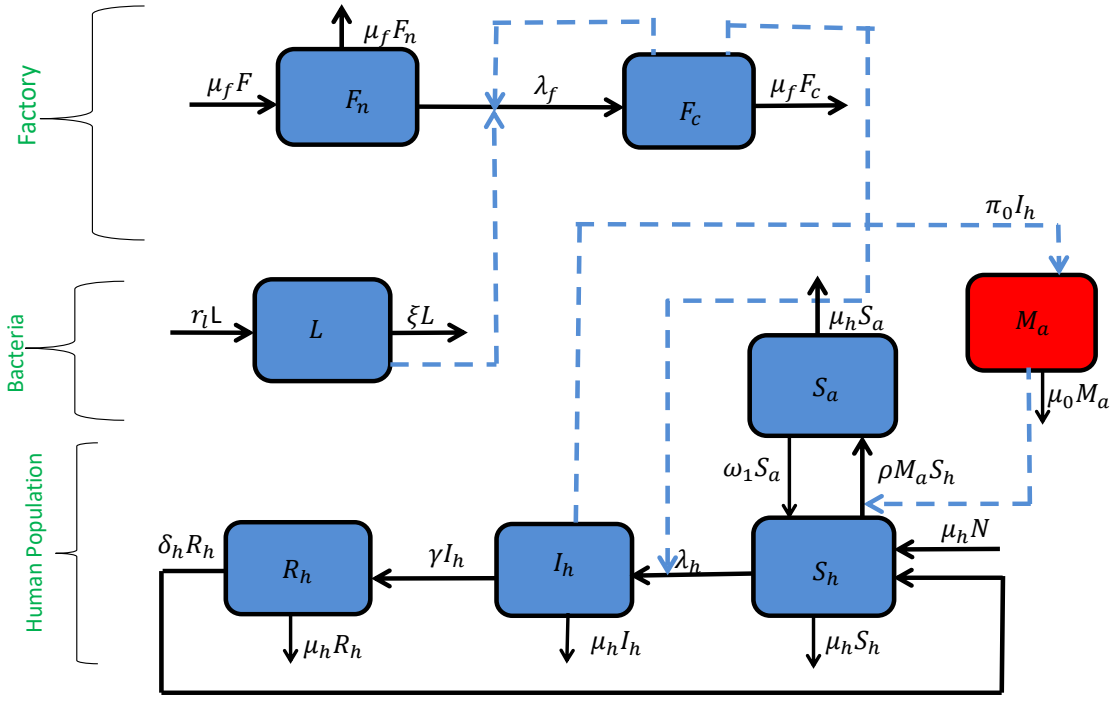


Figure 1. Shows the model flow diagram. The solid arrows represent the transitions between the compartments while the dotted lines represent the influences on the solid arrows and the compartments.

The above model descriptions and Figure 1 gives the following systems of non-linear ordinary

differential equations:

$$\begin{cases} \dfrac{dS_h}{dt} = \mu_h N + \delta_h R_h + \omega_1 S_a - \lambda_h S_h - \mu_h S_h - \rho M_a S_h, \\ \dfrac{dS_a}{dt} = \rho M_a S_h - (\omega_1 + \mu_h) S_a, \\ \dfrac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \gamma) I_h, \\ \dfrac{dR_h}{dt} = \gamma I_h - (\mu_h + \delta_h) R_h, \\ \dfrac{dL}{dt} = r_l L\left(1 - \dfrac{L}{K_L}\right) - \xi L, \\ \dfrac{dM_a}{dt} = \pi_0 I_h - \mu_0 M_a, \\ \dfrac{dF_n}{dt} = \mu_f F - \lambda_f F_n - \mu_f F_n, \\ \dfrac{dF_c}{dt} = \lambda_f F_n - \mu_f F_c. \end{cases} \tag{2.1}$$

All parameters for the model system (2.1) are assumed to be non-negative for all time $t > 0$. By setting

$$s_h = \frac{S_h}{N}, \quad s_a = \frac{S_a}{N}, \quad i_h = \frac{I_h}{N}, \quad r_h = \frac{R_h}{N}, \quad l = \frac{L}{K_L}, \quad m_a = \frac{M_a}{M}, \quad f_n = \frac{F_n}{F}, \quad f_c = \frac{F_c}{F}$$

and given that $r_h(t) = 1 - s_h(t) - s_a(t) - i_h(t)$ we have the following rescaled system

$$\begin{cases} \dfrac{ds_h}{dt} = \mu_h + \delta_h(1 - s_h - s_a - i_h) + \omega_1 s_a - (\tilde{\lambda}_h + \mu_h + \tilde{\rho} m_a) s_h, \\ \dfrac{ds_a}{dt} = \tilde{\rho} m_a s_h - (\omega_1 + \mu_h) s_a, \\ \dfrac{di_h}{dt} = \tilde{\lambda}_h s_h - (\mu_h + \gamma) i_h, \\ \dfrac{dl}{dt} = r_l l(1 - l) - \xi l, \\ \dfrac{dm_a}{dt} = \pi i_h - \mu_0 m_a, \\ \dfrac{df_n}{dt} = \mu_f - (\tilde{\lambda}_f + \mu_f) f_n, \\ \dfrac{df_c}{dt} = \tilde{\lambda}_f f_n - \mu_f f_c, \end{cases} \tag{2.2}$$

where

$$\tilde{\lambda}_h = \beta_1 f_c, \quad \tilde{\lambda}_f = \beta_2 l + \beta_3 f_c, \quad \alpha = \frac{N}{M} \quad \tilde{\rho} = \rho M, \quad \pi = \pi_0 \alpha,$$

with $\beta_1 = \beta_{f_1} F$, $\beta_2 = \beta_l K_L$, $\beta_3 = \beta_{f_2} F$ and initial conditions

$$\begin{aligned} &s_h(0) = s_{h0} > 0, s_a(0) = s_{a0} > 0, i_h(0) = i_{h0} \geq 0, l(0) = l_0 \geq 0, \\ &m_a(0) = m_{a0} \geq 0, f_n(0) = f_{n0} \geq 0, f_c(0) = f_{c0} \geq 0. \end{aligned} \tag{2.3}$$

3. Model properties and analysis

3.1. Positivity of solutions

We prove the positivity of the solutions of model system (2.2) with initial conditions (2.3). First, we state the following Lemma as given in [26].

Lemma 1. *Suppose* $\Omega \subset \mathbb{R} \times C^n$ *is open,* $f_i \in C(\Omega, \mathbb{R}), i = 1, 2, \ldots, n$. *If*

$$f_i|_{x_i(t)=0, X_t \in C^n_{0+}} \geq 0, \;\; X_t = (x_{1(t)}, \cdots, x_{n(t)})^T, \;\; i = 1, 2, \cdots, n,$$

then C^n_{+0} *is the invariant domain of the following equations*

$$\dot{x}_i(t) = f_i(t, X_t), \;\; t \geq 0, \; i = 1, 2, \cdots, n. \tag{3.1}$$

If $f_i|_{x_i(t)=0, X_t \in C^n_{-0}} \leq 0, \;\; X_t = (x_{1(t)}, \cdots, x_{n(t)})^T \;\; i = 1, 2, \cdots, n$, *then* C^n_{-0} *is the invariant domain of Eq (3.1).*

We have the following Theorem on the invariance of system (2.2).

Theorem 1. *Each solution* $(s_h(t), s_a(t), I_h(t), l(t), m_a(t), f_n(t), f_c(t))$ *of the model system (2.2) with the non-negative initial conditions (2.3) is non-negative for all* $t > 0$.

Proof. Let $X = (s_h, s_a, I_h, l, m_a, f_n, f_c)^T$ and

$$g(X) = (g_1(X), g_2(X), g_3(X), g_4(X), g_5(X), g_6(X), g_7(X))^T,$$

then we can re-write the model system (2.2) as follows:

$$\dot{X} = g(X)$$

where

$$g(X) = \begin{pmatrix} g_1(X) \\ g_2(X) \\ g_3(X) \\ g_4(X) \\ g_5(X) \\ g_6(X) \\ g_7(X) \end{pmatrix} = \begin{pmatrix} \mu_h + \delta_h(1 - s_h - s_a - i_h) + \omega_1 s_a - (\tilde{\lambda}_h + \mu_h + \tilde{\rho} m_a) s_h, \\ \tilde{\rho} m_a s_h - (\omega_1 + \mu_h) s_a, \\ \tilde{\lambda}_h s_h - (\mu_h + \gamma) i_h, \\ r_l l(1 - l) - \xi l, \\ \pi_0 \alpha i_h - \mu_0 m_a, \\ \mu_f - (\tilde{\lambda}_f + \mu_f) f_n, \\ \tilde{\lambda}_f f_n - \mu_f f_c, \end{pmatrix}. \tag{3.2}$$

From (3.2), setting all the classes to zero, we have that

$$\begin{array}{rcl} \frac{ds_h(t)}{dt}|_{s_h=0} & = & (\mu_h + \omega_1 s_a) > 0, \;\; \frac{ds_a(t)}{dt}|_{s_a=0} = \rho M m_a s_h > 0, \\ \frac{di_h(t)}{dt}|_{i_h=0} & = & \tilde{\lambda}_h s_h > 0, \;\; \frac{dl(t)}{dt}|_{l=0} = 0, \;\; \frac{dm_a(t)}{dt}|_{m_a=0} = \pi_0 \alpha i_h > 0, \\ \frac{df_n(t)}{dt}|_{f_n=0} & = & \mu_f > 0, \;\; \frac{df_c(t)}{dt}|_{f_c=0} = \tilde{\lambda}_f f_n > 0. \end{array}$$

Thus, it follows that from Lemma 1 that $\mathbb{R}^7_+$ is an invariant set and positive. □

3.2. Existence and uniqueness of solutions

We now show that the solutions of systems (2.2) are bounded. We thus have the following result.

Theorem 2. *The solutions of model system (2.2) are contained in the region* $\Omega \in \mathbb{R}^7_+$, *which is given by* $\Omega = \{(s_h, s_a, i_h, l, m_a, f_n, f_c) \in \mathbb{R}^7_+ : 0 \leq s_h + s_a + i_h \leq 1, 0 \leq l \leq 1, 0 \leq m_a \leq \frac{\pi}{\mu_0}, 0 \leq f_n + f_c \leq 1\}$ *for the initial conditions (2.3) in* Ω.

Proof. Considering the total change in the human population from the model system (2.2) given by

$$\frac{dn}{dt} = \mu_h(1 - s_h - s_a - i_h) + \delta_h(1 - s_h - s_a - i_h) - \gamma i_h, \tag{3.3}$$

for $n = s_h + s_a + i_h \leq 1$ we obtain

$$\begin{aligned} \frac{dn}{dt} &= \mu_h(1 - n) + \delta_h(1 - n) - \gamma i_h, \\ &\leq (\mu_h + \delta_h)(1 - n), \end{aligned}$$

whose solution is

$$n(t) \leq 1 - n(0)\exp[-(\mu_h + \delta_h)t],$$

where $n(0) = s_h(0) + s_a(0) + i_h(0)$ is the initial condition. We note that $0 \leq n \leq 1 - n(0)e^{-(\mu_h+\delta_h)t}$, so that $n(t)$ is bounded provided that $n(0) \geq 0$.

The equation

$$\frac{dl}{dt} = r_l l(1 - l)$$

for the Listeria compartment has a standard solution for a logistic equation

$$l(t) = \frac{1}{1 + \Theta_1 \exp[-r_l t]},$$

which is bounded with $\Theta_1 = \exp[-c]$, where c is a constant.

On the other hand, the total change in the amount of food products resulting from summing the last two equations of (2.2) is given by

$$\begin{aligned} \frac{df}{dt} &= \mu_f - \mu_f f, \\ &\leq \mu_f(1 - f), \end{aligned}$$

whose solution is

$$f(t) = 1 - f(0)\exp[-\mu_f t],$$

where $f(0) = f_n(0) + f_c(0)$ Here, $f_c(t) \leq 1$ as $t \to \infty$ and hence it is bounded above. We thus, conclude that all the solutions of system (2.2) are bounded, biologically feasible and remains in Ω for all $t \in [0, \infty)$. This completes the proof. □

3.3. Steady states and its analyses

The steady states of the model system (2.2) are obtained by equating the right side of Eq (2.2) to zero, so that

$$\begin{cases} \mu_h + \delta_h(1 - s_h^* - s_a^* - i_h^*) + \omega_1 s_a^* - (\beta_1 f_c^* + \mu_h + \tilde{\rho} m_a^*) s_h^* & = 0, \\ \tilde{\rho} m_a^* s_h^* - (\omega_1 + \mu_h) s_a^* & = 0, \\ \beta_1 f_c^* s_h^* - (\mu_h + \gamma) i_h^* & = 0, \\ r_l l^*(1 - l^*) - \xi l^* & = 0, \\ \pi i_h^* - \mu_0 m_a^* & = 0, \\ \mu_f - (\beta_2 l^* + \beta_3 f_c^* + \mu_f) f_n^* & = 0, \\ (\beta_2 l^* + \beta_3 f_c^*) f_n^* - \mu_f f_c^* & = 0. \end{cases} \tag{3.4}$$

From the fourth equation of system (3.4), we have $l^* = 0$ or $l^* = \dfrac{q}{r_l}$, where $q = (r_l - \xi)$ and $r_l > \xi$. We consider the two cases separately.

CASE A: If $l^* = 0$, (i.e., if there is no Listeria in the environment) then from the second last equation of (3.4) we have that

$$f_n^* = \frac{\mu_f}{\beta_3 f_c^* + \mu_f}. \tag{3.5}$$

Substituting (3.5) into the last equation of (3.4) we obtain

$$\beta_3 \mu_f f_c^* - \mu_f f_c^* (\beta_3 f_c^* + \mu_f) \quad = 0,$$

and upon simplification we have

$$f_c^* = 0 \ \text{ or } \ f_c^* = \frac{\beta_3 - \mu_f}{\beta_3}. \tag{3.6}$$

Thus, if $l^* = 0$ then $f_c^* = 0, \tilde{\lambda}_h = \tilde{\lambda}_f = 0$, $f_n^* = 1$ and $i_h^* = 0, m_a^* = 0, s_a^* = 0$. Also, from the first equation of (3.4)

$$s_h^* = 1.$$

This results in the disease-free steady states (DFS) given by

$$E_0^* = (1, 0, 0, 0, 0, 1, 0).$$

On the other hand, from (3.6), we have

$$f_c^* = \frac{\mu_f}{\beta_3}\Big(\mathcal{R}_f - 1\Big),$$

where

$$\mathcal{R}_f = \frac{\beta_3}{\mu_f}.$$

We thus have the following result on the existence of f_c^*.

Lemma 2. *The existence of f_c^* is subject to $\mathcal{R}_f > 1$.*

However, we note that β_3 is the contamination rate contributed by contaminated food products and $\frac{1}{\mu_f}$ is the duration of food contamination. So, $\mathcal{R}_f$ can be defined as the "food contamination threshold" that measures the growth of contaminated food due to the contamination of uncontaminated food products by contaminated food products. This is equivalent to the basic reproduction number ($\mathcal{R}_0$) in disease modelling, see [27].

If $f_c^* = \frac{\mu_f}{\beta_3}(\mathcal{R}_f - 1)$, then expressing the second, third, and fifth equations of (3.4) in terms of i_h^* we obtain the following equation

$$\begin{aligned} m_a^* &= \phi_0 i_h^*, \\ s_h^* &= \phi_1 i_h^*, \\ s_a^* &= \phi_2 i_h^{*2}, \end{aligned} \tag{3.7}$$

where $\phi_0 = \dfrac{\pi}{\mu_0}$, $\phi_1 = \dfrac{\beta_3(\mu_h+\gamma)}{\beta_1\mu_f\left(\mathcal{R}_f-1\right)}$ and $\phi_2 = \dfrac{\tilde{\rho}\phi_0\phi_1}{(\mu_h+\omega_1)}$. Substituting all the expressions in Eq (3.7) into the first equation of (3.4) and after some algebraic simplifications we obtain the following quadratic equation

$$\xi_2 i_h^{*2} + \xi_1 i_h^* + \xi_0 = 0, \tag{3.8}$$

where

$$\begin{aligned} \xi_0 &= -\beta_3(\mu_h+\delta_h) < 0, \\ \xi_1 &= \gamma + \mu_h + \delta_h + \frac{\beta_3(\mu_h+\gamma)(\mu_h+\delta_h)}{\beta_1\mu_f\left(\mathcal{R}_f-1\right)} > 0 \text{ if } \mathcal{R}_f > 1, \\ \xi_2 &= \frac{\pi\tilde{\rho}\beta_3(\mu_h+\gamma)(\mu_h+\delta_h)}{\beta_1\mu_0\mu_f(\mu_h+\omega_1)\left(\mathcal{R}_f-1\right)} > 0 \text{ if } \mathcal{R}_f > 1. \end{aligned}$$

However, we note that the solutions of the quadratic equation (3.8) are given by

$$i_h^* = \frac{-\xi_1 \pm \sqrt{\xi_1^2 - 4\xi_2\xi_0}}{2\xi_2}.$$

The solutions to (3.8) has one positive root when $\mathcal{R}_f > 1$. Biologically, this implies that the disease will persist and eventually invade the human population. This results in the Listeria steady state (LFS)

$$E_1^* = \left(\phi_1 i_h^*,\ \phi_2 i_h^*,\ 0,\ \phi_0 i_h^*,\ \frac{1}{\mathcal{R}_f},\ \tfrac{\mu_f}{\beta_3}(\mathcal{R}_f-1)\right).$$

We note that at Listeria disease free steady state, there are contaminated food products which may result in Listeriosis infection in the human population.

Remark 1. *We note that, when $l^* = 0$, we have two steady states E_0^* and E_1^*. The existence of E_1^* is subject to the contaminated food generation number ($\mathcal{R}_f$) been greater than 1. As long as $\mathcal{R}_f > 1$, even without Listeria in the environment, we will have the disease in the human population.*

CASE B: If $l^* = \dfrac{q}{r_l}$, then from last equation of (3.4) solving for new f_n^*, we have

$$f_n^{*+} = \frac{\mu_f f_c^*}{\beta_3 f_c^* + \dfrac{\beta_2 q}{r_l}}. \tag{3.9}$$

Substituting (3.9), into second last equation of (3.4) we obtain the following expression in terms of f_c^* after some algebraic simplifications

$$\nu_2 f_c^{*2} + \nu_1 f_c^* + \nu_0 = 0, \tag{3.10}$$

where

$$\begin{aligned} \nu_0 &= -\beta_2 q \mu_f < 0, \\ \nu_1 &= \beta_2 \mu_f q + \mu_f^2 r_l (1 - \mathcal{R}_f), \\ \nu_2 &= \beta_3 \mu_f r_l > 0. \end{aligned}$$

The solutions of the quadratic equation (3.10) given by

$$f_c^* = \frac{-\nu_1 \pm \sqrt{\nu_1^2 - 4\nu_2\nu_0}}{2\nu_2},$$

has one positive root irrespective of the signs of ν_1. The solutions of f_c^* say f_c^{*+} exists, but cannot be determined due to its intractability. Hence, as long as $l^* = 1$ we have a positive f_c^{*+}.

Now, we express the second, third and fifth equation of (3.4) in terms of i_h^* and obtain the following expressions

$$\begin{aligned} m_a^* &= \Psi_0 i_h^*, \\ s_h^* &= \Psi_1 i_h^*, \\ s_a^* &= \Psi_2 i_h^{*2}, \end{aligned} \tag{3.11}$$

respectively, where $\Psi_0 = \dfrac{\pi}{\mu_0}$, $\Psi_1 = \dfrac{(\gamma + \mu_h)}{\beta_1 f_c^+}$ and $\Psi_2 = \dfrac{\tilde{\rho}\Psi_0\Psi_1}{\mu_h + \omega_1}$. Similarly, substituting all the expressions from Eq (3.11) into the first equation of (3.4) and after some algebraic manipulations we obtain the following quadratic equation in terms of i_h^*

$$\xi_5 i_h^{*2} + \xi_4 i_h^* + \xi_3 = 0, \tag{3.12}$$

where

$$\begin{aligned} \xi_3 &= -(\mu_h + \delta_h) < 0, \\ \xi_4 &= \frac{(\mu_h + \gamma)(\mu_h + \delta_h) + \beta_1 f_c^{*+}(\delta_h + \mu_h + \gamma)}{\beta_1 f_c^{*+}} > 0, \\ \xi_5 &= \frac{\pi\rho(\gamma + \mu_h)(\delta_h + \mu_h)}{\beta_1 \mu_0 f_c^{*+}(\mu_h + \omega_1)} > 0. \end{aligned}$$

The solutions (i_h^*) of the quadratic equation (3.12) given by

$$i_h^* = \frac{-\xi_4 \pm \sqrt{\xi_4^2 - 4\xi_5\xi_3}}{2\xi_5},$$

exists and has one positive root. We thus have the following result on the existence of new i_h^* say i_h^{*+}.

Lemma 3. *The steady state i_h^{*+} exists whenever f_c^{*+} exists.*

This results in the endemic steady states (ESS) given by

$$E_2^* = (s_h^{*+}, s_a^{*+}, l_1^*, m_a^{*+}, f_n^{*+}) = \left(\Psi_1 i_h^{*+}, \Psi_2 i_h^{*+}, 1, \Psi_0 i_h^{*+}, \frac{\mu_f f_c^{*+}}{\beta_3 f_c^{*+} + \beta_2}\right).$$

Hence, at endemic steady state, there are contaminated food products which result in the persistence of the Listeria infections in the human population.

3.3.1. Local stability of the disease-free steady state

To analyse the local stability of the DFS, we show that the eigenvalues of the Jacobian matrix at DFS have negative real parts. We now state the following theorem for the DFS.

Theorem 3. *The disease-free steady state (E_0^*) is always stable whenever $\mathcal{R}_f < 1$ and $r_l < \xi$.*

Proof. The Jacobian of system (2.2) is given by the block matrix

$$J = \left(\begin{array}{c|c} A_1 & A_2 \\ \hline A_3 & A_4 \end{array}\right), \tag{3.13}$$

where

$$A_1 = \begin{pmatrix} -(\delta_h + \tilde{\rho} m_a^*) & -\delta_h + \omega_1 & -\delta_h & 0 \\ \tilde{\rho} m_a^* & -(\omega_1 + \mu_h) & 0 & 0 \\ \beta_1 f_c^* & 0 & -(\mu_h + \gamma) & 0 \\ 0 & 0 & 0 & \mathcal{F}_0 \end{pmatrix}, \; A_2 = \begin{pmatrix} -\tilde{\rho} s_h^* & 0 & -\beta_1 s_h^* \\ \tilde{\rho} s_h^* & 0 & 0 \\ 0 & 0 & \beta_1 s_h^* \\ 0 & 0 & 0 \end{pmatrix}$$

$$A_3 = \begin{pmatrix} 0 & 0 & \pi & 0 \\ 0 & 0 & 0 & -\beta_2 f_n^* \\ 0 & 0 & 0 & \beta_2 f_n^* \end{pmatrix}, \text{ and } A_4 = \begin{pmatrix} -\mu_0 & 0 & 0 \\ 0 & -(\beta_2 l^* + \beta_3 f_c^* + \mu_f) & -\beta_3 f_n^* \\ 0 & \beta_2 l^* + \beta_3 f_c^* & \beta_3 f_n^* - \mu_f \end{pmatrix}$$

in which $\mathcal{F}_0 = r_l - \xi - 2r_l l^*$. Evaluating (3.13) at DFS, we have that

$$J(E_0^*) = \left(\begin{array}{c|c} J_1(E_0^*) & J_2(E_0^*) \\ \hline J_3(E_0^*) & J_4(E_0^*) \end{array}\right),$$

where

$$J_1(E_0^*) = \begin{pmatrix} -\delta_h & \omega_1 - \delta_h) & -\delta_h & 0 \\ 0 & -(\omega_1 + \mu_h) & 0 & 0 \\ 0 & 0 & -(\mu_h + \gamma) & 0 \\ 0 & 0 & 0 & r_l - \xi \end{pmatrix}, \quad J_2(E_0^*) = \begin{pmatrix} -\tilde{\rho} & 0 & -\beta_1 \\ \tilde{\rho} & 0 & 0 \\ 0 & 0 & \beta_1 \\ 0 & 0 & 0 \end{pmatrix},$$

$$J_3(E_0^*) = \begin{pmatrix} 0 & 0 & \pi_0\alpha & 0 \\ 0 & 0 & 0 & -\beta_2 \\ 0 & 0 & 0 & \beta_2 \end{pmatrix} \text{ and } J_4(E_0^*) = \begin{pmatrix} -\mu_0 & 0 & 0 \\ 0 & -\mu_f & -\beta_3 \\ 0 & 0 & \beta_3 - \mu_f \end{pmatrix}.$$

Similar to the approach used in [28], the eigenvalues of $J(E_0^*)$ are: $\lambda_1 = -\delta_h, \lambda_2 = -(\omega_1 + \mu_h), \lambda_3 = -(\mu_h + \gamma)$

$\lambda_4 = (r_l - \xi) < 0$, if $r_l < \xi$, $\lambda_5 = -\mu_0$, $\lambda_6 = -\mu_f$ and $\lambda_7 = \mu_f(\mathcal{R}_f - 1) < 0$ when $\mathcal{R}_f < 1$. We note that all the eigenvalues are negatives. Hence E_0^* is locally asymptotically stable. □

3.3.2. Local Stability of the Listeria-free steady state

We state the following theorem for the local stability of Listeria-free steady state.

Theorem 4. *The Listeria-free steady state* (E_1^*) *is always stable whenever* $\mathcal{R}_f > 1$ *and* $r_l < \xi$.

Proof. Evaluating (3.13) at Listeria-free steady state, we have that

$$J(E_1^*) = \left(\begin{array}{c|c} J_1(E_1^*) & J_2(E_1^*) \\ \hline J_3(E_1^*) & J_4(E_1^*) \end{array}\right),$$

where

$$J_1(E_1^*) = \begin{pmatrix} -(\delta_h + \tilde{\rho}\phi_0 i_h^*) & -\delta_h + \omega_1 & -\delta_h & 0 \\ \tilde{\rho}\phi_0 i_h^* & -(\omega_1 + \mu_h) & 0 & 0 \\ \frac{\beta_1\mu_f}{\beta_3}(\mathcal{R}_f - 1) & 0 & -(\mu_h + \gamma) & 0 \\ 0 & 0 & 0 & r_l - \xi \end{pmatrix}, \quad J_2(E_1^*) = \begin{pmatrix} -\tilde{\rho}\phi_1 i_h^* & 0 & -\beta_1\phi_1 i_h^* \\ \tilde{\rho}\phi_1 i_h^* & 0 & 0 \\ 0 & 0 & \beta_1\phi_1 i_h^* \\ 0 & 0 & 0 \end{pmatrix},$$

$$J_3(E_1^*) = \begin{pmatrix} 0 & 0 & \pi_0\alpha & 0 \\ 0 & 0 & 0 & \frac{-\beta_2}{\mathcal{R}_f} \\ 0 & 0 & 0 & \frac{\beta_2}{\mathcal{R}_f} \end{pmatrix}, \text{ and } J_4(E_1^*) = \begin{pmatrix} -\mu_0 & 0 & 0 \\ 0 & -\mu_f\mathcal{R}_f & \frac{-\beta_3}{\mathcal{R}_f} \\ 0 & \mu_f(\mathcal{R}_f - 1) & \frac{\beta_3}{\mathcal{R}_f} - \mu_f \end{pmatrix}.$$

The eigenvalues from $J_1(E_1^*)$ are: $\lambda_1 = (r_l - \xi) < 0$ if $r_l < \xi$ and the solutions to the cubic equation

$$\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where

$$a_0 = \frac{1}{\beta_3\mu_0}\Big(\beta_3(\gamma + \mu_h)(\pi\tilde{\rho}\mu_h i_h^* + \delta_h(\pi\tilde{\rho} i_h^* + \mu_0(\mu_h + \omega_1))) + (\beta_1\mu_0\mu_f\delta_h(\mu_h + \omega_1)(\mathcal{R}_f - 1)\Big),$$

$$a_1 = \frac{1}{\beta_3\mu_0}\Big(\beta_3\Big(\pi\tilde{\rho} i_h^*(2\mu_h + \delta_h + \gamma) + \mu_0\Big((\mu_h + \gamma)(\mu_h + \omega_1) + \delta_h(2\mu_h + \omega_1 + \gamma)\Big)\Big) + (\beta_1\mu_0\mu_f\delta_h)(\mathcal{R}_f - 1)\Big),$$

$$a_2 = 2\mu_h + \delta_h + \omega_1 + \gamma + \frac{\pi\tilde{\rho} i_h^*}{\mu_0}.$$

Given that a_0, a_1 and a_2 are positive provided that $\mathcal{R}_f > 1$, we note that

$$a_2a_1 - a_0 = \frac{1}{\beta_3\mu_0^2}\Big(\Upsilon_0 + \beta_1\delta_h\mu_0\mu_f(\pi\tilde{\rho}i_h^* + \mu_0(\gamma + \delta_h + \mu_h))(\mathcal{R}_f - 1)\Big),$$

for

$$\Upsilon_0 = \beta_3(\pi\tilde{\rho}i_h^* + \mu_0(\delta_h + \mu_h + \omega_1))(\pi\tilde{\rho}i_h^*(\gamma + \delta_h + 2\mu_h) + \mu_0(\gamma + \delta_h + \mu_h)\mathcal{F}_1)$$

and $\mathcal{F}_1 = (2\mu_h + \omega_1 + \gamma)$. We also, note that $a_2a_1 - a_0 > 0$ if $\mathcal{R}_f > 1$. Hence, by the Routh-Hurwitz criterion the eigenvalues of $J(E_1^*)$ have negative real parts. The rest of the eigenvalues are determined from $J_4(E_1^*)$, which are; $\lambda_5 = -\mu_0$ and the solutions to the quadratic equation

$$\lambda^2 + a_4\lambda + a_3 = 0,$$

where

$$a_3 = \mu_f\mathcal{R}_f + \mu_f\Big(1 - \frac{1}{\mathcal{R}_f^2}\Big) \text{ and } a_4 = \mu_f\mathcal{R}_f\Big(1 - \frac{1}{\mathcal{R}_f^3}\Big).$$

We thus have that $a_3 > 0$ and $a_4 > 0$ if $\mathcal{R}_f > 1$ and a_4 is always positive. Therefore, the eigenvalues have negative real parts by Routh-Hurwitz criteria. Hence, E_1^* is locally asymptotically stable. □

3.3.3. Local stability of the endemic steady state

We now state the following theorem on the local stability of endemic steady state.

Theorem 5. *The endemic steady state* (E_2^*), *is always locally asymptotically stable if* $\mathcal{R}_f > 1$ *and* $r_l > \xi$.

Proof. Evaluating (3.13) at endemic steady state, we have that

$$J(E_2^*) = \left(\begin{array}{c|c} J_1(E_2^*) & J_2(E_2^*) \\ \hline J_3(E_2^*) & J_4(E_2^*) \end{array}\right),$$

where

$$J_1(E_2^*) = \begin{pmatrix} -(\delta_h + \tilde{\rho}\Psi_0 i_h^{*+}) & -\delta_h + \omega_1 & -\delta_h & 0 \\ \tilde{\rho}\Psi_0 i_h^{*+} & -(\omega_1 + \mu_h) & 0 & 0 \\ \beta_1 f_n^{*+} & 0 & -(\mu_h + \gamma) & 0 \\ 0 & 0 & 0 & -(r_l - \xi) \end{pmatrix}, \quad J_2(E_2^*) = \begin{pmatrix} -\tilde{\rho}\Psi_1 i_h^{*+} & 0 & -\beta_1\Psi_1 i_h^{*+} \\ \tilde{\rho}\Psi_1 i_h^{*+} & 0 & 0 \\ 0 & 0 & \beta_1\Psi_1 i_h^{*+} \\ 0 & 0 & 0 \end{pmatrix},$$

$$J_3(E_2^*) = \begin{pmatrix} 0 & 0 & \pi_0\alpha & 0 \\ 0 & 0 & 0 & -\beta_2 f_n^{*+} \\ 0 & 0 & 0 & \beta_2 f_n^{*+} \end{pmatrix} \text{ and } J_4(E_2^*) = \begin{pmatrix} -\mu_0 & 0 & 0 \\ 0 & -(\beta_2 + \beta_3 f_c^{*+} + \mu_f) & -\beta_3 f_n^{*+} \\ 0 & \beta_2 + \beta_3 f_c^{*+} & \beta_3 f_n^{*+} - \mu_f \end{pmatrix}.$$

The eigenvalues from $J_1(E_2^*)$ are: $\lambda_1 = -(r_l - \xi) < 0$ if $r_l > \xi$ and the solutions to the cubic equation

$$\lambda^3 + b_2\lambda^2 + b_1\lambda + b_0 = 0,$$

where

$$b_0 = \frac{1}{\mu_0}\Big(\pi\tilde{\rho}i_h^{*+}\mu_h(\gamma+\mu_h)+\delta_h(\pi\tilde{\rho}i_h^{*+}(\gamma+\mu_h)+\mu_0\mathcal{D}_0)\Big),$$

$$b_1 = \frac{1}{\mu_0}\Big(\pi\tilde{\rho}i_h^{*+}(\gamma+\delta_h+2\mu_h)+\mu_0((\gamma+\mu_h)(\mu_h+\omega_1)+\delta_h\mathcal{D}_1)\Big),$$

$$b_2 = 2\mu_h+\delta_h+\omega_1+\gamma+\tfrac{\pi\tilde{\rho}i_h^{*+}}{\mu_0},$$

Note that $b_0,\ b_1$ and b_2 are positive and

$$b_2b_1-b_0=\frac{1}{\mu_0^2}\Big(\Upsilon_1+\Upsilon_2\Big)>0,$$

for

$$\Upsilon_1 = \pi^2\tilde{\rho}^2i_h^{*+2}(\gamma+\delta_h+2\mu_h)+\mu_0^2(\gamma+\delta_h+\mu_h)\Big((\mu_h+\omega_1)(\gamma+2\mu_h+\omega_1)+\delta_h(\gamma+\beta_1f_c^{*+}+2\mu_h+\omega_1)\Big) \text{ and}$$

$$\Upsilon_2 = \pi\tilde{\rho}\mu_0i_h^{*+}(\delta_h^2+(\gamma+2\mu_h)^2+(2\gamma+3\mu_h)\omega_1+\delta_h[\beta_1f_c^{*+}+5\mu_h+2(\gamma+\omega_1)]).$$

Thus, by the Routh-Hurwitz criteria, the eigenvalues have negative real parts. The remaining eigenvalues are determined from $J_4(E_2^*)$, which are: $\lambda_5=-\mu_0$ and the solutions to the quadratic equation

$$\lambda^2+b_4\lambda+b_3=0,$$

where

$$b_3 = \frac{\mu_f\left((\beta_2+\beta_3f_c^{*+})^2+\beta_2\mu_f\right)}{\beta_2+\beta_3f_c^{*+}}>0,$$

$$b_4 = \beta_2+\beta_3f_c^{*+}+\frac{(2\beta_2+\beta_3f_c^{*+})\mu_f}{\beta_2+\beta_3f_c^{*+}}>0.$$

Hence, the eigenvalues have negative real parts by Routh-Hurwitz criterion. Therefore, all the eigenvalues of $J(E_2^*)$ have negative real parts. Thus, E_2^* is locally asymptotically stable and otherwise unstable. □

4. Numerical results

4.1. Parameter values

Numerical simulations of the model system (2.2) were done using the set of parameters values given in Table 1. Most of the parameter values were estimated since there are very few mathematical models in literature, that have been done on *L. Monocytogenes* disease dynamics and hence the parameter values are elusive. We used a fourth order Runge-Kutta numerical scheme to perform the simulations with the initial conditions: $s_h(0)=0.42, s_a(0)=0.53, i_h(0)=0.05, l(0)=0.1, m_a(0)=0.25, f_n=0.2$ and $f_c=0.8$. The initial conditions were hypothetically chosen for the numerical simulations presented in section and are thus only for illustrative purpose and do not represent any observed scenario.

Table 1. Parameter values used for numerical simulations.

Parameter description	Symbol	Value (day^{-1})	Source
Mortality rate of humans	μ_h	0.02/365	[24]
Recovery rate of humans	γ	0.02	Assumed
Rate of loss of immunity for humans	δ_h	0.2	[24]
Waning rate of aware susceptibles	ω_1	0.25	[16]
Rate of non-aware susceptibles to aware	$\tilde{\rho}$	0.9	Assumed
Growth rate of Listeria	r_l	0.25	[24]
Death rate of Listeria	ξ	0.056	Assumed
Depletion rate of media campaigns	μ_0	0.03	[17]
Implementation rate of media campaigns	π	0.001	[17]
Contact rate between Listeria and humans	β_1	0.0025	Assumed
Food contamination rate by Listeria	β_2	0.09	Assumed
Food contamination rate	β_3	0.0048	Assumed
Removal rate of food products	μ_f	0.056	Assumed

4.2. Sensitivity analysis

Sobol sensitivity analysis [29] were used to determine the model parameters that are sensitive to changes in some variable of the model system (2.2). We performed simulations for some chosen parameters π, μ_0, $\tilde{\rho}$, ω_1, β_1, β_2, β_3 and μ_f versus some of the model state variables s_a, i_h and f_c to show their respective PRCCs over time. These parameters and state variables were selected because they are the most significant in the Listeria disease transmission and control according to our model formulation relating to the subject under investigation. The simulations were done with 1000 runs over 800 days as depicted in Figures 2 and 3. The scatter plots for parameters with positive and negative PRCCs are also shown in Figure 4. Thus, an increase in the rate at which susceptible individuals move into the aware susceptible class results in a fewer number of humans been infected with the Listeriosis and parameter ω_1 with negative correlation signifying that if aware individuals revert to being susceptible, then they are prone to contracting the disease as depicted in Figure 4 respectively. This highlights the importance of media campaigns in disease control.

4.3. Effects of varying π and μ_0 on Listeriosis spread

This subsection is devoted to numerical simulations that show the effects of increasing and decreasing media campaigns over time as depicted in in Figure 5. Figure 5(a),(b) reveal that the increase in the awareness campaigns result in a decrease in the number of infected and increase the aware susceptible humans, respectively. While the reduction of media campaigns results in more humans being infected with Listeriosis and less aware susceptible humans as shown in Figure 5(c),(d), respectively.

4.4. Contour plot of β_3 and μ_f on $\mathcal{R}_f$

In Figure 6, we present a contour plot of the food contamination rate β_3 and rate of food product removal, μ_f, versus the food contamination constant, $\mathcal{R}_f$. An increase in the contamination of non-contaminated food by contaminated food products results in an increase in the value of $\mathcal{R}_f$. Hence, more

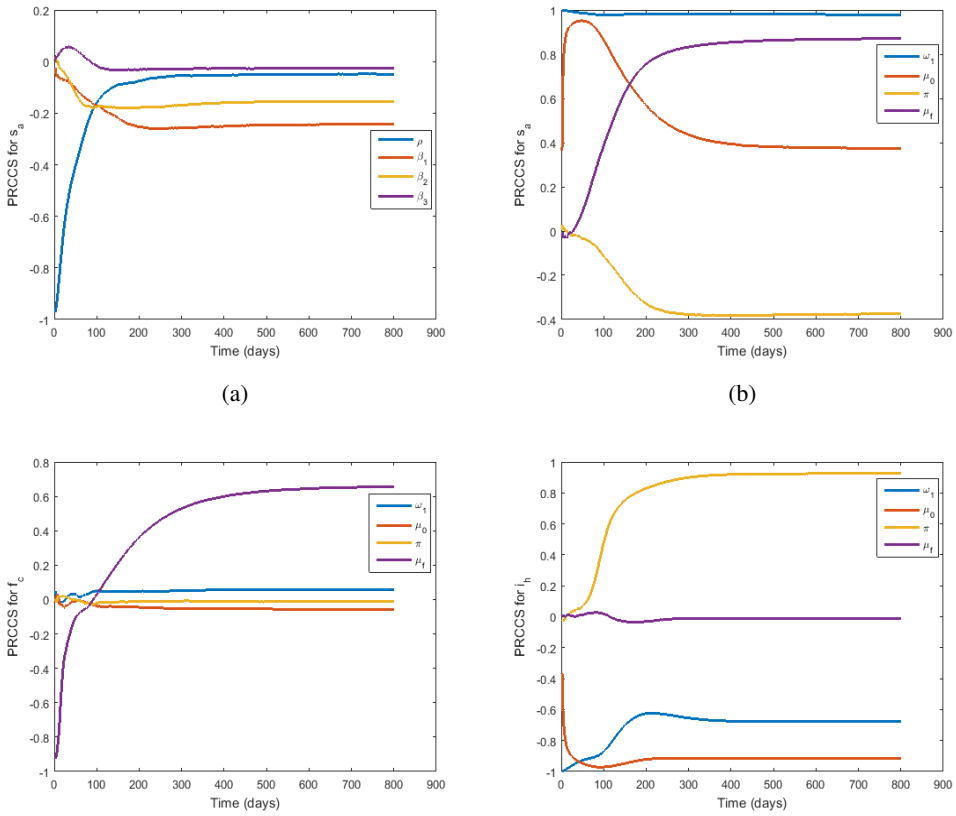


Figure 2. Partial correlation coefficients of parameters $\pi, \mu_0, \tilde{\rho}, \omega_1, \beta_1, \beta_2, \beta_3$ and μ_f over time for state variables s_a, f_c and i_h respectively. Note that, ρ in the legend represents $\tilde{\rho}$.

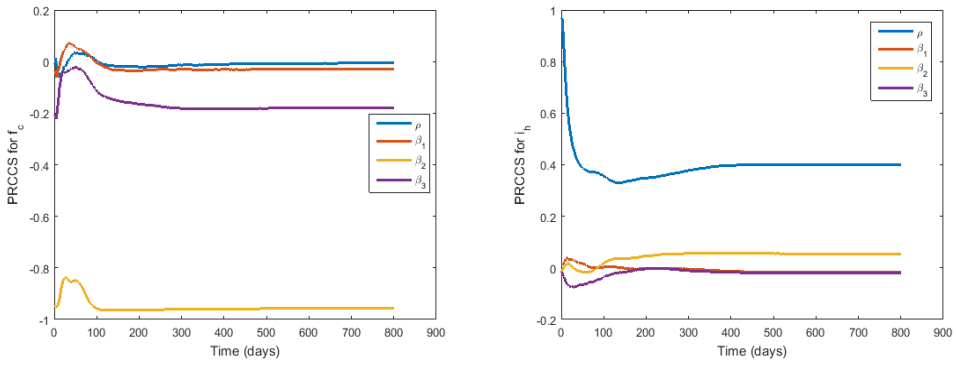
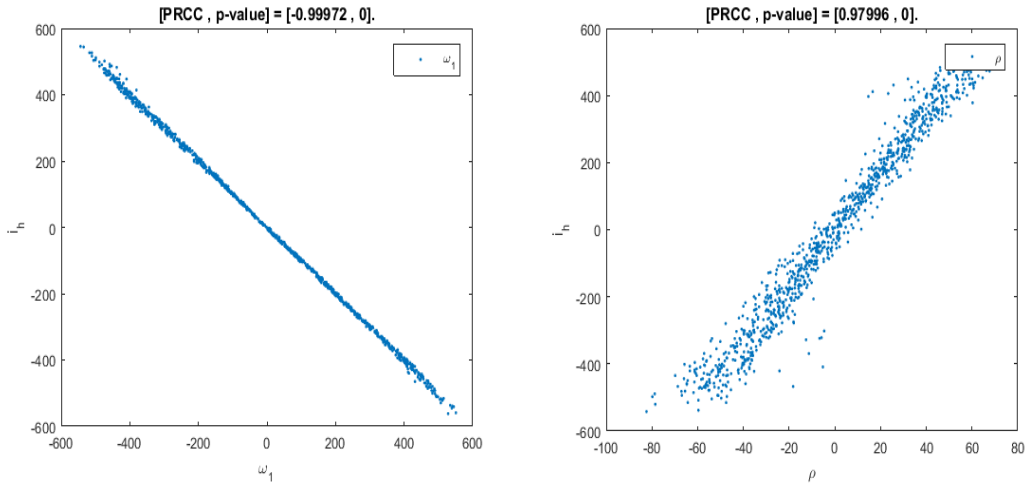
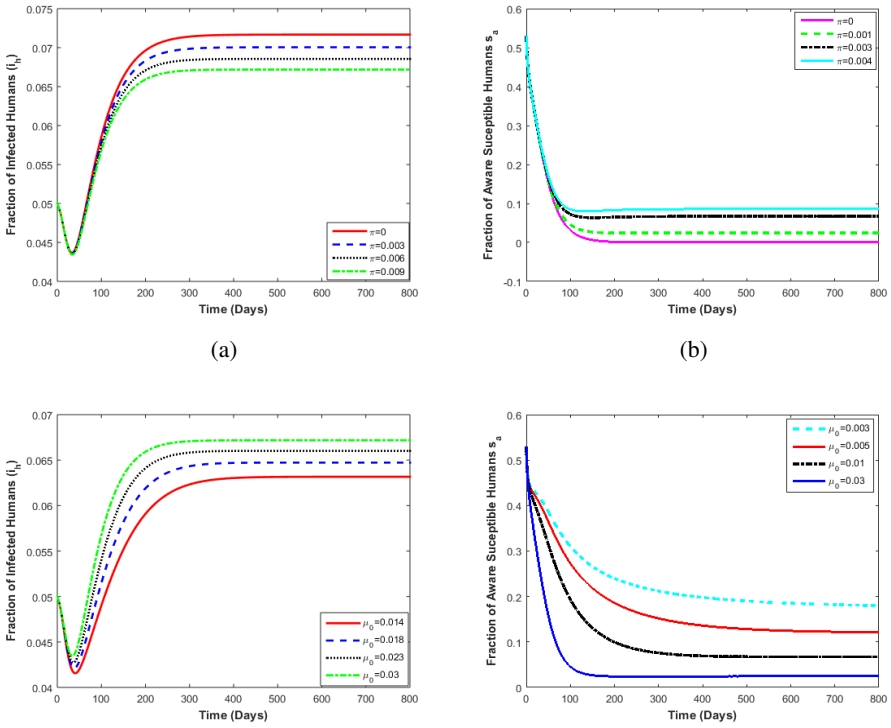


Figure 3. Partial correlation coefficients of parameters $\pi, \mu_0, \tilde{\rho}, \omega_1, \beta_1, \beta_2$ and β_3 over time against f_c and i_h. Note that, ρ in the legend represents $\tilde{\rho}$.



(a) (b)

Figure 4. Scatter plots of parameters with the negative ($\tilde{\rho}$) and positive (ω_1) PRCCs.



(a) (b)

(c) (d)

Figure 5. Effects of implementation of media campaigns (π) on the fraction of; (a) infected humans (b) aware susceptible humans, while the effects of depletion of media campaigns (μ_0) on the fraction of; (c) infected humans (d) aware susceptible humans.

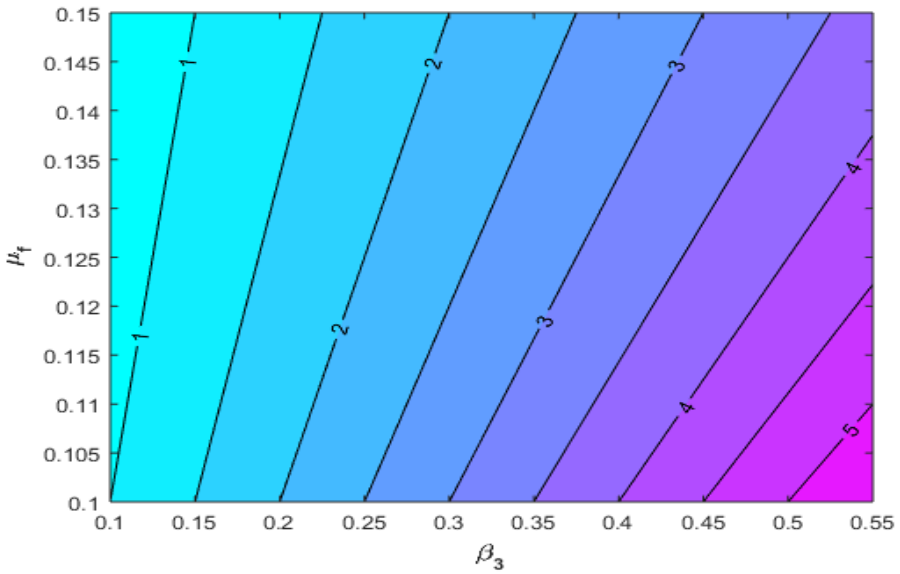


Figure 6. Contour plot of $\mathcal{R}_f$ as a function of β_3 and μ_f.

humans get infected with Listeriosis. Also, an increase in the removal of contaminated food products results in a decrease in the values of $\mathcal{R}_f$ which implies that fewer humans contract the disease. Note that, the changes in the value of μ_f do not significantly impact $\mathcal{R}_f$ when compared to β_3.

4.5. Effects of β_3 and μ_f on $\mathcal{R}_f$

Figure 7 illustrates a mesh plot showing the variation of β_3, μ_f and the contaminated food generation number $\mathcal{R}_f$. It is seen that an increase in the rate of food processing increases $\mathcal{R}_f$, while an increase in the removal of contaminated food results in a decrease in $\mathcal{R}_f$. Note that at the intersection of the plane $\mathcal{R}_f = 1$ and the mesh plot, we obtain all values of β_3 and μ_f necessary for the eradication of the disease.

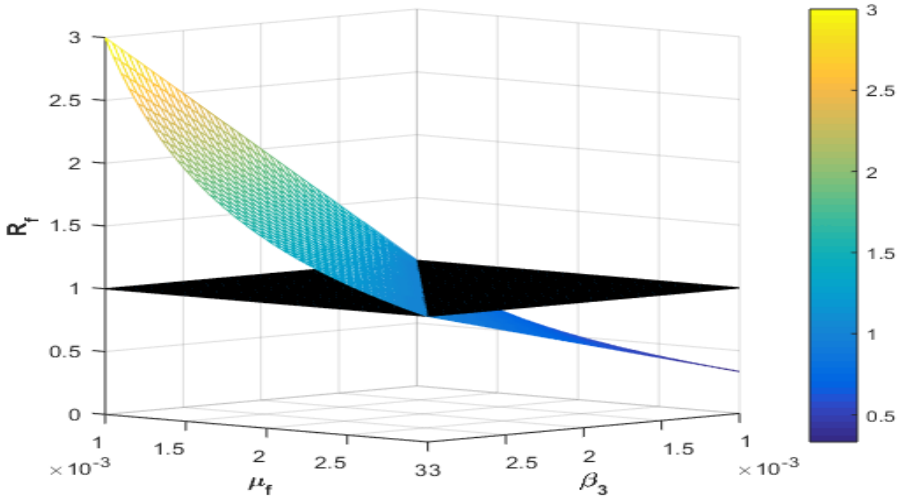
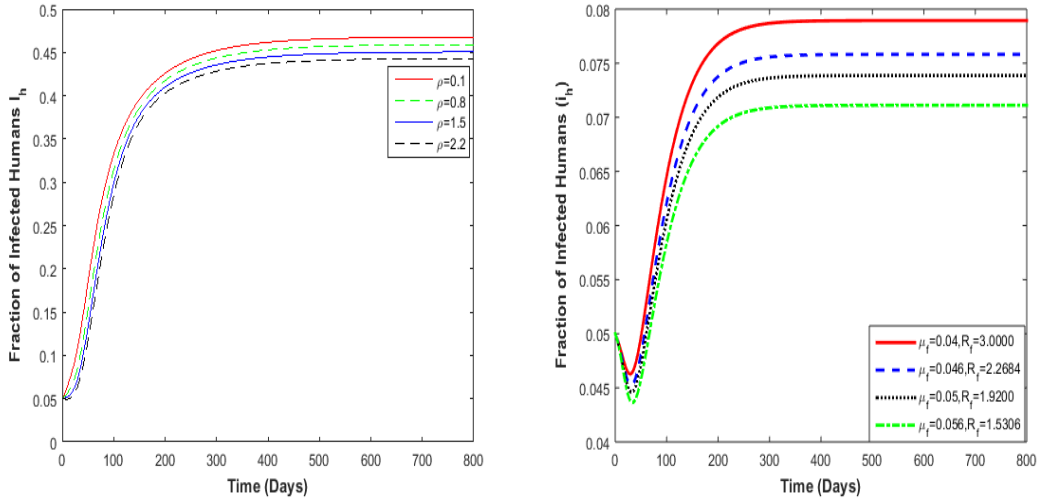


Figure 7. Effects of β_3 and μ_f on the contamination food constant ($\mathcal{R}_f$).

4.6. *Impact of varying $\tilde{\rho}$ and μ_f on infected humans*

Figure 8 depicts the effects of varying parameters $\tilde{\rho}$ and μ_f. In Figure 8(a), we observe that increasing $\tilde{\rho}$ decreases the number of infected humans. This implies that media campaigns have the potential to reduce the number of infectives. On the other hand, an increase in $\tilde{\rho}$ does not impact the value of the contaminated food generation number, $\mathcal{R}_f$. On the other hand, increasing μ_f also reduces the number of infected individuals and $\mathcal{R}_f$ (see Figure 8(b)). So the removal of contaminated food products during an outbreak is an important intervention in the control of Listeriosis.



(a) (b)

Figure 8. (a) Effects varying removal of contaminated food products (μ_f) on the fraction of infected humans. (b) Effects varying the rate of non-aware susceptible becomes aware susceptible ($\tilde{\rho}$) on the fraction of infected humans. The value for the $\mathcal{R}_f$ is 1.5306.

5. Conclusions

A deterministic model on media campaigns' potential role on Listeriosis disease transmissions was developed and analysed in this manuscript. Stability analyses of the model were done in terms of the food contamination constant $\mathcal{R}_f$. The model exhibited three different steady states, which are: disease-free, Listeria-free, and endemic equilibria. The disease-free and the Listeria-free steady states are locally asymptotically stable if the net growth rate $r_l < \xi$, $\mathcal{R}_f < 1$ and $r_l < \xi$, $\mathcal{R}_f > 1$ respectively, while the endemic equilibrium state is locally asymptotically stable if $r_l > \xi$ and $\mathcal{R}_f > 1$. On the other hand from our numerical results, it was established that an increase in the removal of contaminated food products and an increase in the rate at which the susceptible individuals become aware susceptible individuals leads to a decrease in the number of infected humans (see Figure 8). Thus to effectively control Listeria disease spread, policymakers, public health, governments, and global stakeholders are advised to implement media campaigns that do not wane as time progresses. This means that media campaigns need to be effective. Further, these interventions from the campaigns should target mainly susceptible individuals in the danger of contracting Listeriosis. As people adhere to the media campaign effectively, it helps reduce and control the number of infected humans leading to less disease

transmission. The model presented in this paper is not without fallibility. The model was not fitted to epidemiological data and we assumed that the infectives do not interact with the aware susceptibles. During Listeriosis spread, the assumption has a negative impact on the human population since fewer un-aware individuals become aware of the disease. Despite these shortcomings, the results obtained in this paper are still implementable to help manage, control or contain Listeriosis disease transmission in the event of an outbreak.

In future work, a non-standard explicit discretization method can be considered for solving the Listeriosis model which developed in [30] and the result can be compared with classical methods such as Euler, Runge–Kutta, and some other established approaches.

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Conflicts of interest

No conflict of interest.

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