

# Qualitative Analysis of the Effect of Minimising Listeriosis in Cow Population on the Eradication of Human Listeriosis

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**Abstract** Listeriosis is a bacterial infection and one of the key zoonotic foodborne diseases, accounting for about 28% of yearly food-related mortality in the US. Listeriosis spreads into human populations mostly through the ingestion of contaminated milk and meat. Since cows are a major source of milk and meat to humans, a nonlinear epidemic model is designed to analyse the transmission dynamics of listeriosis in cow populations under management conditions of treatment and disinfection. The model is studied qualitatively by deriving its equilibria and reproductive ratio ( $R_0$ ), and by analysing its stability using stability theory for differential equations. The analysis reveals the necessary and sufficient conditions for listeriosis persistence and eradication in cow populations. The results indicate that listeriosis spread in cow populations could be prevented or eradicated if the rates of application of treatment and disinfection exceed certain critical values.

**Keywords:** Listeriosis; model; equilibria; reproductive ratio; stability

## 1 Introduction

Listeriosis is a contagious infection that instigates stillbirths in mammals. The agent of listeriosis is *Listeria monocytogenes*, which survives in soil and water and attacks the intestinal track through the ingestion of contaminated food materials [1]. Listeriosis persists as a major issue in veterinary medicine and as one of the key zoonotic food-borne diseases, accounting for about 28% of food-related mortality in the US each year [2]. It is a serious foodborne disease, and studies have established a link between ingestion of contaminated silage and animal rhombencephalitis [3].

While cattle play a crucial role in spreading and amplifying the listeria bacteria, *L. monocytogenes* is a saprophyte, meaning it thrives and naturally survives as a free-living organism in soil, mud, and water [4, 5]. The bacteria are highly resilient and can persist in dry soil, manure, and on straw for long periods, continuously seeding the infection into animal feed [6, 7]. While listeriosis is a serious infection in certain individuals, humans do not typically contract it through direct contact with cattle; rather, they are infected through foodborne transmission [8, 9].

The incubation period for listeriosis is between 11–70 days, and the most vulnerable individuals are the elderly, babies, pregnant women and immunosuppressed patients such as individuals who are suffering from AIDS, hemochromatosis, cirrhosis, diabetes, sarcoidosis, renal failure, aplastic anaemia, hematologic malignancies, ulcerative colitis and collagen vascular disease [10–12]. When the infection is fully developed, infected cows may exhibit meningitis, febrile gastroenteritis, sepsis, perinatal infections, etc. [13]. *L. monocytogenes* infections in cows may be treated with antibiotics such as rifampicin, linezolid and meropenem [11].

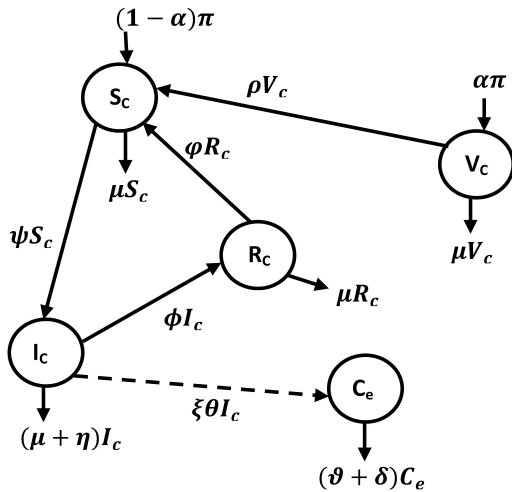
Listeriosis outbreaks have been reported in Europe, Japan, North America, and South Africa, where 209 deaths were attributed to listeriosis infections between 2017 and 2018 [14]. Several mathematical and nonmathematical models have been developed to give insights into the transmission dynamics, growth and survival of *L. monocytogenes*. The majority of the mathematical models in the literature consider listeriosis transmission in the environment and in human and animal populations, without considering any particular animal [2, 11, 13, 14]. Since listeriosis spreads into the human population mostly through the ingestion of contaminated milk and meat [15] and given that cows are a major source of milk and meat to humans, it is reasonable to argue that elimination of *L. monocytogenes* in cow populations may be enough to prevent or eradicate listeriosis in

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the human population. Based on that hypothesis, a nonlinear epidemic model is developed by incorporating factors capable of preventing or eradicating listeriosis in cow populations; in the present analysis, unlike earlier works, the dynamics of listeriosis in the environment and in human and animal populations are considered. The management conditions incorporated into the present model are vaccination of calves, treatment of cows, and disinfection of the environment.

## 2 Model Formulation

The population  $N(t)$  that consists of cows and contaminated environment in Fig. (1) is divided into five compartments: susceptible cows ( $S_c$ ), vaccinated cows ( $V_c$ ), infected cows ( $I_c$ ), recovered cows ( $R_c$ ) and contaminated environment ( $C_e$ ). Cows are recruited at rate  $\pi$ , with some proportion  $\alpha$  vaccinated at birth and moved to the vaccination class  $V_c$  with the quantity  $\alpha\pi$ , while the remaining proportion  $(1 - \alpha)$  moves to the susceptible compartment at birth with the quantity  $(1 - \alpha)\pi$ . The proportion  $\alpha$  of the cows that are vaccinated at birth is immune to listeriosis infection until the immunity wanes. The vaccinated cows move to the susceptible compartment at a rate  $\rho$  after waning of immunity. Infections occur when there is effective contact between susceptible and infected cows, and between susceptible cows and a contaminated environment, at rates  $\beta_1$  and  $\beta_2$ , respectively.



**Fig. 1** Transmission diagram of listeriosis dynamics in cow populations

Infected cows increase *L. monocytogenes* in the soil at rate  $\theta$  and regain susceptibility after recovery at rate  $\phi$ . Treatment and disinfection are introduced to the infected

cows and the environment at rates  $\phi$  and  $\vartheta$ , respectively, and the introduction of the two controls results in reductions in disease spread from the infected cows and the contaminated environment, with reduction factors  $\tau_1$  and  $\tau_2$ , respectively. The reduction factors  $\tau_1$  and  $\tau_2$  are embedded in  $\psi$ . The introduction of treatment  $\phi$  to the infected cows also reduces  $\theta$ , the rate at which the infected cows increase the growth of *L. Monocytogenes* in the environment with reduction factor  $\xi$ . Mortality unrelated to listeriosis in cows occurs at rate  $\mu$  while listeriosis-related mortality in cows occurs at rate  $\eta$ . Also, listeriosis removal from the soil unrelated to disinfection occurs at rate  $\delta$ . Going by the aforementioned assumptions and the transmission diagram, the following nonlinear epidemic model is derived.

$$\frac{dV_c}{dt} = \alpha\pi - (\rho + \mu)V_c, \quad (1)$$

$$\frac{dS_c}{dt} = (1 - \alpha)\pi + \rho V_c + \phi R_c - (\psi + \mu)S_c, \quad (2)$$

$$\frac{dI_c}{dt} = \psi S_c - (\phi + \xi\theta + \eta + \mu)I_c, \quad (3)$$

$$\frac{dR_c}{dt} = \phi I_c - (\phi + \mu)R_c, \quad (4)$$

$$\frac{dC_e}{dt} = \xi\theta I_c - (\vartheta + \delta)C_e, \quad (5)$$

where

$$\psi = \tau_1\beta_1 I_c + \tau_2\beta_2 C_e. \quad (6)$$

### 2.1 Positivity and Boundedness of Solutions

**Theorem 1** The listeriosis model in Eq. (1)-Eq. (5) is bounded in the region

$$\Omega = \left\{ (V_c, S_c, I_c, R_c, C_e) \in \mathbb{R}^5 : 0 < S_c \leq \frac{\pi}{\mu}, \right. \\ \left. 0 \leq V_c < 1, \quad 0 \leq I_c < 1, \right. \\ \left. 0 \leq R_c < 1, \quad 0 \leq C_e \leq \frac{\xi\theta}{\vartheta + \delta} \right\}. \quad (7)$$

if it is established that the solutions of the model are non-negative.

*Proof* Let the solution set of the model be  $\{V_c(t), S_c(t), I_c(t), R_c(t), C_e(t)\}$ . Since the model monitors the populations of living things, the initial conditions to each state variable are nonnegative, i.e.,  $V_c(0) \geq 0$ ,  $S_c(0) \geq 0$ ,  $I_c(0) \geq 0$ ,  $R_c(0) \geq 0$ , and  $C_e(0) \geq 0$ .

From Eq. (1),

$$\frac{dV_c(t)}{dt} = \alpha\pi - (\rho + \mu)V_c, \quad (8)$$

$$\frac{dV_c(t)}{dt} + \sigma V_c(t) = \varepsilon, \quad (9)$$

where  $\sigma = \rho + \mu$  and  $\varepsilon = \alpha\pi$ . Then,

$$\Rightarrow \frac{dV_c(t)}{dt} \exp(\sigma t) + \sigma V_c(t) \exp(\sigma t) = \varepsilon \exp(\sigma t), \quad (10)$$

$$\Rightarrow \frac{d}{dt} [V_c(t) \exp(\sigma t)] = \varepsilon \exp(\sigma t), \quad (11)$$

$$\Rightarrow V_c(t) = V_c(0) \exp(-\sigma t) + \frac{\varepsilon}{\sigma} [1 - \exp(-\sigma t)]. \quad (12)$$

Therefore,  $V_c(t) \geq 0$  in Eq. (12) since  $V_c(0) \geq 0$ . Hence, the solution  $V_c(t)$  is nonnegative. By the same argument, the nonnegativity for other solutions  $S_c(t)$ ,  $I_c(t)$ ,  $R_c(t)$ , and  $C_e(t)$  can be established. Hence, the solution set  $\{V_c(t), S_c(t), I_c(t), R_c(t), C_e(t)\}$  for the model is nonnegative. Therefore, the model is bounded in the region defined in Eq. (7).

### 3 Model Analysis

#### 3.1 Equilibria and Reproductive Ratio

The listeriosis model Eq. (1)-Eq. (5) allows a listeriosis-free equilibrium (LFE) obtained as  $L_0 = (V_c^0, S_c^0, I_c^0, R_c^0, C_e^0)$  =  $\left(\frac{\alpha\pi}{\mu+\rho}, \frac{(\mu+\rho)(1-\alpha)\pi+\rho\alpha\pi}{\mu(\mu+\rho)}, 0, 0, 0\right)$  and a listeriosis-endemic equilibrium (LEE) obtained as  $L^* = (V_c^*, S_c^*, I_c^*, R_c^*, C_e^*)$  with coordinates

$$V_c^* = \frac{\alpha\pi}{\mu+\rho} > 0, \quad (13)$$

$$S_c^* = \frac{(\mu+\rho)(1-\alpha)\pi+\rho\alpha\pi+\varphi(\mu+\rho)R_c^*}{(\mu+\rho)(\psi+\mu)} > 0, \quad (14)$$

$$I_c^* = \frac{\psi S_c^*}{\phi+\xi\theta+\eta+\mu} > 0, \quad (15)$$

$$R_c^* = \frac{\phi I_c^*}{\varphi+\mu} > 0, \quad (16)$$

$$C_e^* = \frac{\xi\theta I_c^*}{\vartheta+\delta} > 0. \quad (17)$$

After deriving the model's equilibria, the reproductive ratio ( $R_0$ ) must be computed to quantify the tendency toward listeriosis outbreaks or eradication in cow populations. It is an epidemiological parameter that measures the transmission potential of infectious diseases. If the numerical value of  $R_0$  is greater than one, the infection will spread, but if  $R_0 < 1$ , the infection will fail to spread or die out. Following the Next Generation Matrix Operator developed by Driessche and Watmough, which has been applied in many epidemic models [16–21] and outlined in [22], the reproductive ratio for the present listeriosis model is derived and is given by

$$R_0 = \frac{(\vartheta+\delta)\tau_1\beta_1 S_c^0 + \xi\theta\tau_2\beta_2 S_c^0}{(\vartheta+\delta)(\phi+\xi\theta+\eta+\mu)}. \quad (18)$$

The result of  $R_0$  in Eq. (18) indicates that listeriosis transmission can be prevented or eradicated in cow populations if the treatment and disinfection parameters,  $\phi$  and  $\vartheta$ , are sufficiently large. Increasing these parameters reduces the effective transmission rates  $\beta_1$  and  $\beta_2$  associated with infected cows and the contaminated environment, respectively, as well as the parameter  $\theta$ , which governs the contribution of infected cows to the growth of *L. monocytogenes* in the environment. Consequently, higher values of  $\phi$  and  $\vartheta$  lead to lower effective values of  $\beta_1$ ,  $\beta_2$ , and  $\theta$ , thereby decreasing the magnitude of  $R_0$ . If  $R_0$  is reduced below unity, as indicated by Eq. (18), listeriosis transmission cannot be sustained, providing a necessary condition for the prevention or eradication of the disease in cow populations.

#### 3.2 Stability Analysis of Listeriosis-Free Equilibrium

**Theorem 2** *The listeriosis-free equilibrium of the model is locally asymptotically stable if the associated  $R_0 < 1$ ; otherwise the model is locally asymptotically unstable.*

*Proof* To investigate the stability of the model about the listeriosis-free equilibrium  $L_0$ , the variational matrix of the model about  $L_0$  is derived and is given as

$$J(L_0) = \begin{pmatrix} -(\mu+\rho) & 0 & 0 & 0 & 0 \\ \rho & -\mu & -\tau_1\beta_1 S_c^0 & \varphi & -\tau_2\beta_2 S_c^0 \\ 0 & 0 & \tau_1\beta_1 S_c^0 - \Gamma & 0 & \tau_2\beta_2 S_c^0 \\ 0 & 0 & \phi & -(\varphi+\mu) & 0 \\ 0 & 0 & \xi\theta & 0 & -(\vartheta+\delta) \end{pmatrix}, \quad (19)$$

where  $\Gamma = \phi + \xi\theta + \eta + \mu$ .

Three of the eigenvalues of the matrix  $J(L_0)$  are negative and are given as  $-\mu$ ,  $-(\mu+\rho)$  and  $-(\varphi+\mu)$ . The remaining two eigenvalues can be computed from the submatrix

$$A = \begin{pmatrix} \tau_1\beta_1 S_c^0 - (\phi + \xi\theta + \eta + \mu) & \tau_2\beta_2 S_c^0 \\ \xi\theta & -(\vartheta + \delta) \end{pmatrix}. \quad (20)$$

$|A - \lambda I| = 0$  is evaluated to

$$\lambda^2 + \left[ \vartheta + \delta + \phi + \xi\theta + \eta + \mu - \tau_1\beta_1 S_c^0 \right] \lambda + (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu) - (\vartheta + \delta)\tau_1\beta_1 S_c^0 - \xi\theta\tau_2\beta_2 S_c^0 = 0. \quad (21)$$

All the roots in Eq. (21) are less than zero if

$$\vartheta + \delta + \phi + \xi\theta + \eta + \mu - \tau_1\beta_1 S_c^0 > 0. \quad (22)$$

and,

$$(\vartheta + \delta)(\phi + \xi\theta + \eta + \mu) - (\vartheta + \delta)\tau_1\beta_1 S_c^0 - \xi\theta\tau_2\beta_2 S_c^0 > 0. \quad (23)$$

From Eq. (23),

$$\begin{aligned} & (\vartheta + \delta)\tau_1\beta_1S_c^0 + \xi\theta\tau_2\beta_2S_c^0 \\ & < (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu) \\ \Rightarrow & \frac{(\vartheta + \delta)\tau_1\beta_1S_c^0 + \xi\theta\tau_2\beta_2S_c^0}{(\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)} \\ & < \frac{(\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)}{(\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)}. \end{aligned} \quad (24)$$

In view of Eq. (18),  $R_0 < 1$ .

Since it has been established that  $R_0 < 1$ , inequality (22) is satisfied and all eigenvalues of Eq. (21) are negative. Therefore, the listeriosis-free equilibrium of the model is locally asymptotically stable. The global stability of the listeriosis model can also be investigated around the disease-free equilibrium  $L_0$  using a Lyapunov function.

**Theorem 3** *The listeriosis-free equilibrium  $L_0$  is globally asymptotically stable if  $R_0 < 1$ .*

*Proof* The model is globally asymptotically stable around  $L_0$  if it can be shown that  $\dot{V}(t) < 0$ , where  $V(t)$  is a Lyapunov function. Define

$$\dot{V}(t) = A_1I'_c + A_2C'_e, \quad (25)$$

$$\begin{aligned} \dot{V}(t) = & -\left[A_1(\phi + \xi\theta + \eta + \mu) - A_1\tau_1\beta_1S_c^0 - A_2\xi\theta\right]I_c \\ & - \left[A_2(\vartheta + \delta) - A_1\tau_2\beta_2S_c^0\right]C_e, \end{aligned} \quad (26)$$

$$\begin{aligned} \dot{V}(t) = & (\vartheta + \delta)\tau_1\beta_1S_c^0C_e + \xi\theta\tau_2\beta_2S_c^0C_e \\ & - (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)C_e, \end{aligned} \quad (27)$$

$$\begin{aligned} \dot{V}(t) = & (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu) \\ & \times \left[\frac{(\vartheta + \delta)\tau_1\beta_1S_c^0 + \xi\theta\tau_2\beta_2S_c^0}{(\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)} - 1\right]C_e, \end{aligned} \quad (28)$$

$$\dot{V}(t) = (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)(R_0 - 1)C_e. \quad (29)$$

In view of Eq. (18),

$$\dot{V}(t) = (\vartheta + \delta)(\phi + \xi\theta + \eta + \mu)(R_0 - 1)C_e. \quad (30)$$

If  $R_0 \leq 1$  in Eq. (30), the Lyapunov function satisfies  $\dot{V}(t) \leq 0$ . Also,  $\dot{V}(t) = 0$  if  $C_e = 0$ . Therefore, the listeriosis-free equilibrium of the model is globally asymptotically stable. However, if  $R_0 \geq 1$ , there exists a unique listeriosis-endemic equilibrium.

### 3.3 Stability Analysis of Listeriosis-Endemic Equilibrium

**Theorem 4** *The listeriosis-free equilibrium becomes unstable and the system moves from  $L_0$  to  $L^*$ , the listeriosis-endemic equilibrium, if  $R_0 > 1$ .*

*Proof* The reproductive ratio is greater than one ( $R_0 > 1$ ) and the listeriosis-endemic equilibrium of the model is locally asymptotically stable if it is established that all the The eigenvalues of the variational matrix of the model about  $L^*$ , the listeriosis-endemic equilibrium, are negative. The variational matrix of the model computed at  $L^*$  is derived as

$$J(L^*) = \begin{pmatrix} -\mu - \rho & 0 & 0 & 0 & 0 \\ \rho & -\psi - \mu & -\tau_1\beta_1S_c^* & \phi & -\tau_2\beta_2S_c^* \\ 0 & \psi & \tau_1\beta_1S_c^* - \Gamma & 0 & \tau_2\beta_2S_c^* \\ 0 & 0 & \phi & -\phi - \mu & 0 \\ 0 & 0 & \xi\theta & 0 & -\vartheta - \delta \end{pmatrix}. \quad (31)$$

One of the eigenvalues of  $J(L^*)$  is  $-(\mu + \rho)$  and the remaining eigenvalues can be derived from submatrix  $M$  defined as

$$J(M) = \begin{pmatrix} -(\psi + \mu) & -\tau_1\beta_1S_c^* & \phi & -\tau_2\beta_2S_c^* \\ \psi & \tau_1\beta_1S_c^* - \Gamma & 0 & \tau_2\beta_2S_c^* \\ 0 & \phi & -(\phi + \mu) & 0 \\ 0 & \xi\theta & 0 & -(\vartheta + \delta) \end{pmatrix}. \quad (32)$$

Since a row reduction transform a matrix into a new one that has the exact same solution set with the original matrix, we row reduce  $J(M)$  in (32) and obtain

$$J(M) = \begin{pmatrix} -(\psi + \mu) - \tau_1\beta_1S_c^* & \phi & -\tau_2\beta_2S_c^* \\ 0 & c_1 & \phi & c_2 \\ 0 & \phi & -(\phi + \mu) & 0 \\ 0 & \xi\theta & 0 & -(\vartheta + \delta) \end{pmatrix}, \quad (33)$$

where

$$c_1 = \frac{(\psi + \mu)[\tau_1\beta_1S_c^* - (\phi + \xi\theta + \eta + \mu)]}{\psi} - \tau_1\beta_1S_c^*, \quad (34)$$

$$c_2 = \frac{(\psi + \mu)\tau_2\beta_2S_c^*}{\psi} - \tau_2\beta_2S_c^*. \quad (35)$$

The matrix  $J(M)$  has the eigenvalue  $-(\psi + \mu)$  while the remaining eigenvalues can be obtained from submatrix  $N$  defined as

$$J(N) = \begin{pmatrix} c_1 & \phi & c_2 \\ \phi & -(\phi + \mu) & 0 \\ \xi\theta & 0 & -(\vartheta + \delta) \end{pmatrix}. \quad (36)$$

Row reduce matrix operation reduces the matrix  $J(N)$  to

$$J(N) = \begin{pmatrix} -\frac{c_1}{\phi}(\phi + \mu) + \phi & 0 & -\frac{c_2}{\phi}(\phi + \mu) \\ \phi & -(\phi + \mu) & 0 \\ \xi\theta & 0 & -(\vartheta + \delta) \end{pmatrix}. \quad (37)$$

The matrix  $J(N)$  has one of the eigenvalues being  $-(\varphi + \mu)$  and the remaining two eigenvalues can be derived from matrix  $P$  given as

$$J(P) = \begin{pmatrix} -\frac{c_1}{\varphi}(\varphi + \mu) + \phi - \frac{c_2}{\varphi}(\varphi + \mu) & \\ \xi\theta & -(\vartheta + \delta) \end{pmatrix}. \quad (38)$$

The characteristic equation of Eq. (38), i.e.,  $|A - \lambda I| = 0$ , simplifies to

$$\lambda^2 + \left[ \frac{c_1}{\varphi}(\varphi + \mu) + \vartheta + \delta - \phi \right] \lambda + \frac{\xi\theta c_2}{\varphi}(\varphi + \mu) + (\vartheta + \delta) \left[ \frac{c_1}{\varphi}(\varphi + \mu) - \phi \right] = 0. \quad (39)$$

The two eigenvalues in Eq. (39) are negative if

$$\begin{aligned} \frac{c_1}{\varphi}(\varphi + \mu) + \vartheta + \delta - \phi > 0, \text{ and} \\ \frac{\xi\theta c_2}{\varphi}(\varphi + \mu) + (\vartheta + \delta) \left[ \frac{c_1}{\varphi}(\varphi + \mu) - \phi \right] > 0. \end{aligned} \quad (40)$$

If inequalities (40) are true, the two eigenvalues in Eq. (39) are negative,  $R_0 > 1$ , and the endemic equilibrium of the listeriosis model is locally asymptotically stable. However, if one or both eigenvalues in Eq. (39) are positive,  $R_0 < 1$ , and the endemic equilibrium of the model is locally asymptotically unstable if one or both inequalities (40) are not true.

From inequalities (40), assuming

$$\frac{c_1}{\varphi}(\varphi + \mu) + \vartheta + \delta - \phi < 0. \quad (41)$$

Then,

$$\phi^* - \vartheta^* > \frac{c_1}{\varphi}(\varphi + \mu) + \delta. \quad (42)$$

Notice in the transmission diagram (Figure 1) as well as in Eq. (4) and Eq. (5) that  $\phi$  results in the transfer of infected cows to the recovered compartment, while  $\vartheta$  results in the elimination of *L. monocytogenes* from the environment. Parameter  $\phi$  acts as an injection (positive contribution), whereas  $\vartheta$  acts as a withdrawal (negative contribution). Therefore, the quantity  $\phi^* - \vartheta^*$  appearing in inequality (42) does not represent a net difference between the two controls, but rather the critical threshold condition that must be satisfied for  $R_0$  to be reduced below unity and for the endemic equilibrium to lose its local asymptotic stability. While  $\phi^*$  denotes the critical treatment level required to transfer infected cows into the recovered compartment,  $\vartheta^*$  denotes the critical disinfection level required to eliminate *L. monocytogenes* from the environment. Consequently, the stability of the endemic equilibrium depends on the magnitude of the treatment and disinfection parameters  $\phi$  and  $\vartheta$ .

If the threshold condition (42) is satisfied, one or both eigenvalues in Eq. (39) become positive and the endemic equilibrium is locally asymptotically unstable. Conversely, if condition (42) is not satisfied, the eigenvalues in Eq. (39) may remain negative and the endemic equilibrium may be negative and the endemic equilibrium of the listeriosis model becomes locally asymptotically stable.

## 4 Results and Discussion

Based on the hypothesis underlying the model, eradication of *L. monocytogenes* in cow populations may be enough to prevent or eradicate listeriosis in the human population. A rigorous analysis was conducted, and the necessary conditions for local and global stability of the model were derived, which depended on the threshold parameter  $R_0$ . The disease-free equilibrium of the model was proved to be locally and globally asymptotically stable whenever  $R_0 < 1$ . The implication of the disease-free equilibrium being locally and globally asymptotically stable is that listeriosis would fail to spread within the model framework in both local and global cattle farms if a listeriosis-infected cow were introduced into a naive cow population. Listeriosis would not break out locally and globally in cow populations if the conditions imposed on the model were maintained. The stability of the disease-free equilibrium therefore depended on the rates of applications of treatment and disinfection parameters whose values must attain the critical level defined in the analysis (i.e.,  $\phi^*$  and  $\vartheta^*$ ). If the values of the control parameters treatment and disinfection fall short of these critical levels, the disease-free equilibrium would become unstable. *L. monocytogenes* might spread in cow populations and consequently get into the human population when the disease-free equilibrium becomes unstable.

## 5 Conclusion

Listeriosis is a widespread zoonotic disease. The main natural reservoirs are soil, water, decaying vegetation and the gastrointestinal tracts of animals. A wide range of animals – both domestic and wild – act as reservoirs and asymptomatic carriers, shedding the bacteria into the environment via faeces, milk, and uterine discharges [1, 3]. Examples of animals (both domestic and wild as well as farm animals) that are carriers for *L. monocytogenes* are sheep, cattle, goats, pigs, dogs, cats, poultry, deer, moose, elk, reindeer, wild boars, mice, rabbits, rats, gulls, crows, pigeons, fish, crustaceans, insects, etc. [3, 10]. While animals rarely transmit listeriosis directly to humans, humans typically contract listeriosis by consuming food or water contaminated by these animal reservoirs. Because cattle are a major reservoir that transmits the bacteria into raw milk, unpasteurised dairy products, and

meat, a compartmental model has been designed to analyse the transmission dynamics of listeriosis in cow populations under management conditions of treatment and disinfection. The solutions of the model had been proved to be positive and bounded. The equilibria and reproductive ratio of the model were derived, and the stability was analysed based on the derived reproductive ratio. The model's stability had been analysed to theoretically derive the minimum level of treatment and disinfection needed to eradicate human listeriosis by preventing or minimising listeriosis in the cow population. The model analysis had been limited to qualitative aspects, while the quantitative and numerical simulations had been excluded to reduce space, as in [20, 23–25]. However, it is anticipated that this analysis has provided the essential knowledge required to prevent listeriosis transmission in the human population by preventing or minimising listeriosis spread in the cow population.

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