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Research Article

Mathematical Model for the Transmission and Control of Mastitis in Dairy Cows

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Abstract: Mastitis in dairy cows is a complex infectious disease that significantly impacts animal health, milk quality, and farm profitability. This study develops and analyzes a deterministic compartmental mathematical model describing the transmission of mastitis among cows and via environmental contamination. The model is formulated using a system of ordinary differential equations that include key biological and management parameters, such as bacterial shedding, recovery, and culling rates. Dimensional consistency and stability analyses were conducted to ensure the model's validity. The basic reproduction number was calculated, and the equilibrium points were identified and analyzed for both local and global stability. Numerical simulations demonstrate the dynamic behavior of susceptible, infected, and recovered groups, highlighting how infection control depends on changes in shedding and recovery parameters. The model offers a theoretical framework for optimizing mastitis prevention strategies and supports informed decision-making in dairy herd management, as shown through the application of data from a Macedonian dairy farm.

***Keywords*:** mathematical model SIR + P, mastitis, equilibrium points, basic reproduction number

MSC: 37N25, 34C60, 65L05, 92D30

1. Introduction

Interactions among humans, animals, and the environment are central to the emergence of infectious diseases, many of which originate from animal reservoirs [1]. Zoonotic diseases are those diseases or infections that can be transmitted between humans and wild and domestic animals. These diseases account for a large share of recent disease outbreaks, often linked to animal-derived foods [2]. Zoonoses pose a serious global public health threat due to their potential for severe illness and death.

Mastitis in dairy cows is a complex disease that not only affects animal health but also poses substantial public health risks. The development of mastitis in dairy herds results from a complex interaction among the host, the environment, and pathogenic microorganisms. Risk factors for mastitis are typically classified into two major categories: cow-level determinants and environmental determinants. The latter encompasses a range of factors, including herd management practices, hygiene protocols, housing conditions, milking technology, nutritional strategies, calving seasonality, and preventive health programs [3]. This multifactorial condition is primarily associated with bacterial infections and can

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lead to significant economic losses in the dairy industry [4], as well as concerns regarding food safety and public health due to the potential spread of antimicrobial resistance [5]. The implications of mastitis are underpinned by the prevalence of various pathogens, including Staphylococcus aureus, *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus faecalis*, and others, which have been increasingly associated with multidrug resistance due to antimicrobial misuse and poor management practices [4].

The primary contagious pathogens responsible for udder infections in dairy herds are Staphylococcus aureus, Streptococcus agalactiae, and coliform species [4]. In addition, mastitis can be caused by a wide variety of opportunistic environmental pathogens, including *Escherichia coli*, *Klebsiella spp.*, *Enterobacter spp.*, *Serratia spp*, *Pseudomonas spp.*, *Proteus spp.*, *Corynebacterium pyogenes*, *Streptococcus uberis*, *Streptococcus dysgalactiae*, as well as coagulase-negative staphylococci (as in [5, 6]).

Depending on the route of transmission, mastitis can be divided into contagious and non-contagious mastitis caused by the pathogens in the environment [7]. Environmental microorganisms, particularly those found in bedding and manure, are significant sources of mastitis pathogens.

Pathogen-specific studies reveal that different bacterial species exhibit diverse infection modes, ultimately impacting mastitis severity and economic losses. While Staphylococcus aureus is well-documented for causing both clinical and subclinical mastitis, *Streptococcus uberis* is recognized as a leading cause of chronic mastitis, particularly under intensive production conditions [8]. There are pathogen-specific characteristics associated with the bacterial counts in milk and their shedding from infected mammary quarters [9] Such distinctions are vital for developing tailored control measures aimed at specific pathogens prevalent in distinct environments (as in [10, 11]).

The control of mastitis in dairy cows is critical for maintaining animal health, improving milk quality, and enhancing overall dairy farm profitability. Effective mastitis control programs rely on various strategies, including risk factor identification, hygiene management, regular monitoring, and timely interventions [12]. Furthermore, the integration of mathematical modelling and predictive analytics into these programs can enhance the understanding of infection dynamics, improve decision-making, and optimize resource allocation for prevention and treatment [13].

In scientific literature, mathematical models have been extensively developed to study the dynamics of various diseases. Some of these models are closely related to epidemiology, particularly in the description of infectious disease transmission.

Deterministic models have been widely applied to capture the dynamic behavior of disease spread [14–16]. For instance, Greenhalgh and Rozins [15] proposed a generalized model of the differential equations for modeling infectious disease transmission, whereas Van den Driessche [16] investigated the role and implications of the basic reproduction number within such models.

Mathematical models can also be applied in the field of chemistry, as demonstrated in [17].

Susceptible-Exposed-Infectious-Recovered (SEIR)-type mathematical models and their extensions have been developed to describe the transmission dynamics of infectious diseases [18, 19], such as measles [20], influenza [21], tuberculosis [22], and COVID-19 [23].

A similar modeling framework has been applied in the veterinary context, focusing on the prevalence dynamics of mastitis in dairy herds [13].

Several studies have also integrated optimization and control strategies, such as those used in mastitis management [24, 25]. Additionally, Sisk and Fefferman introduced methods for studying infectious disease spread [26].

Understanding pathogen dynamics in dairy herds is also pivotal. The efficacy of mastitis control efforts can be enhanced further through mathematical modeling. Research indicates that mathematical models can simulate mastitis dynamics, accounting for factors such as pathogen transmission, environmental conditions, and herd management practices [27]. For instance, models that utilize Somatic Cell Counts (SCC) as indicators of mastitis prevalence can provide early warnings to dairy producers about potential outbreaks, enabling timely intervention measures [28]. A study by Hyde et al. emphasized the role of machine learning techniques in predicting mastitis infection patterns based on historical data related to herd health and environmental conditions [29].

Mathematical models of differential equations are applied in many other areas of human life, and provide sustainable predictions for the deepening or suppression (as much as possible) of a certain problem that is the subject of observation

(as in [30]). From the mathematical side, these models are most often subjected to dynamic analysis (as in the papers [31–34]) and then treated with some mathematical software (as in [32, 35–37]).

Therefore, the main aim of this paper was to develop mathematical models based on differential equations that can help reduce the negative effects of mastitis in real-world dairy environments (as an example in the scientific literature [13, 24, 25]).

2. Materials and methods

A dynamic system of differential equations was developed to describe the spread and persistence of mastitis in a dairy cow population. The model incorporated four key processes: (i) transmission of the disease from cow to cow, (ii) acquisition of infection from pathogens in the environment, (iii) loss of immunity after recovery, and (iv) recurrence of the disease, which may occur up to four times. The model was adapted from the epidemiological framework of cholera proposed in [16], with modifications to account for the biological and environmental characteristics of cow's mastitis.

For the model, the dynamic analysis was carried out through several theorems supported by proofs. A limited region was identified, following the approaches in [21, 22, 24, 25, 30]. Within this region, the positivity of the solutions of the system was proven (as in [24, 25, 30, 38]). The corresponding theorem used to prove the positivity of the solutions followed the method described in [38]. The equilibrium points were determined using a procedure like that in [18, 21, 22, 24, 25, 30, 31], as this step is of exceptional importance for such models. The local stability of the model, which describes the behavior of the system near equilibrium points, was analyzed using the Routh-Hurwitz stability criterion [39], following the approaches in [18, 21, 22, 24, 25, 30]. The local stability of the model was related to the basic reproduction number. This number was calculated as the largest eigenvalue of the next generation matrix developed by Van den Driessche and Watmough [16, 40]. Therefore, the basic reproduction number for this model was obtained in line with [16, 21, 22, 24–26, 30, 41]. In addition to local stability, the global stability of the model was analyzed using the Castillo-Chavez conditions and the construction of a Lyapunov function, following the methods described in [24, 25, 42].

To evaluate the practical relevance of the model, a case study was conducted using the data on mastitis prevalence from a dairy farm in the Republic of North Macedonia. Farm records and herd information were used to estimate input values for the mathematical model. The analysis was carried out under two scenarios:

1. Ideal scenario: The farm is assumed to maintain impeccable hygienic conditions, resulting in a negligible concentration of pathogens in the environment, such that the risk of environmental transmission is minimal at the initial stage.

2. Equilibrium scenario: The environment is assumed to be approximately in an equilibrium state at the initial stage, with bacterial levels sufficient to contribute to the persistence of mastitis.

For each scenario:

- The basic reproduction number ($\Re_0$) was calculated;
- The coordinates and stability of equilibrium points were determined;
- Numerical simulations were performed in Mathematica, providing graphical visualizations of model dynamics;
- Predicted values of model functions over several years were tabulated.

The outcomes of the model analysis were used to predict the future dynamics of mastitis under the two scenarios. Based on these results, recommendations for potential management interventions were developed, aiming to improve herd health, ensure animal welfare, and increase economic profitability on the farm.

3. Mathematical model

The mathematical model will be presented in several parts. The formulation is through a system of ordinary differential equations, and its sustainability is considered. The two equilibrium points are identified: the equilibrium point presenting a cow population without mastitis, and the equilibrium point presenting a cow population with mastitis

manifested as an epidemic. For this model, the basic reproduction number will also be found, based on which the local and global stability of the system will be considered.

3.1 *Formulation of the mathematical model*

The so-called mathematical model SIRS + P will be used for solving our problem. This model is based on the epidemiological model of Cholera [16], which belongs to the group of classical SIR epidemiological models. Its visualization is given in Figure 1.

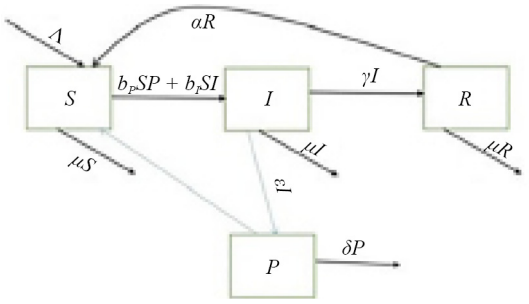

Figure 1. Mathematical model SIRS + P

According to Figure 1, the population of cows on the farm is divided into three groups: susceptible $S(t)$, infected $I(t)$, and recovered $R(t)$ at the time t, so that the cow population

$$N(t) = S(t) + I(t) + R(t)$$

Since the disease can also arise from bacteria present in the environment where the cows live, the influence of the environment is considered by $P(t)$-pathogen concentration (mean: measuring the concentration of bacteria in the environment). On the other hand, cows do not develop solid immunity to this disease, so it can recur multiple times, which implies that this aspect must be considered in constructing the model. The differential equations of the model are

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - b_P SP - b_I SI - \mu S + \alpha R \\
\frac{dI}{dt} &= b_P SP + b_I SI - (\mu + \gamma) I \\
\frac{dR}{dt} &= \gamma I - (\mu + \alpha) R \\
\frac{dP}{dt} &= \varepsilon I - \delta P
\end{aligned} \tag{1}$$

with the initial values $S(0) = S_0 > 0$, $I(0) = I_0 > 0$, $R(0) = R_0 > 0$, $P(0) = P_0 > 0$.

Interpretation of equations of the model (1). The model (1) describes how mastitis spreads within a dairy herd through interactions between cows and environmental bacteria. The formulation relies on the following biological and epidemiological assumptions:

• The recruitment of new susceptible cows into the herd occurs at a constant rate Λ;

• The susceptible cows may become infected by direct contact with infected cows at a rate proportional to $b_I SI$ or through exposure to environmental bacteria at a rate proportional to $b_P SP$;

• The infected cows recover at a constant rate γ;

• The recovered cows can relapse into the susceptible group at a mastitis recurrence rate α;

• All cows, regardless of their infection status, are subject to culling or natural death at a culling rate μ;

• The concentration of bacteria in the environment $P(t)$ increases through shedding from infected cows at a rate εI, but decreases naturally at a rate δP.

Thus, the model construction is grounded in biologically justified premises, and each process in the equations corresponds directly to a real phenomenon observed in the epidemiology of cow mastitis.

Dimensional homogeneous of the model (1) variables and parameters. Let the basic dimensions be defined as:

• $[c]$-concentration of bacteria (e.g., $CFU\dot{m}l^{-1}$);

• $[N]$-number of cows;

• $[T]$-time.

The main parameters have the following descriptions and dimensions:

• The parameter Λ is the recruited rate $\left[\frac{N}{T}\right]$;

• The parameter b_P is the transmission rate of mastitis to cows from bacteria in the environment $\left[\frac{1}{T \cdot c}\right]$;

• The parameter b_I is the transmission rate of mastitis from cow to cow $\frac{1}{T \cdot N}$;

• The parameter γ is the recovery rate $\left[\frac{1}{T}\right]$;

• The parameter ε is the shedding rate of the bacteria from the infected mammary gland quarters, linking the number of infected cows to the increase in bacterial concentration in the environment $\left[\frac{c}{T \cdot N}\right]$;

• The parameter δ is the removal rate of the shed bacteria $\left[\frac{1}{T}\right]$;

• The parameter μ is the culling rate of cows from the herd (when animals are removed from the herd entirely, often due to poor production, health, fertility problems, or natural death) $\left[\frac{1}{T}\right]$;

• The parameter α is the mastitis recurrence rate $\left[\frac{1}{T}\right]$.

Each equation in the system (1) is dimensionally homogeneous. Therefore, the model (1) is dimensionally consistent and physically interpretable.

3.2 *Sustainability of the mathematical model*

The sustainability of the mathematical model (1) will be given by two theorems, the first of which relates to the bounded region, and the second to the positivity of the solutions within it.

Theorem 1 Assume the system of differential Equation holds. Then, a feasible solution set for initial conditions $S(0) = S_0 > 0,\ I(0) = I_0 > 0,\ R(0) = R_0 > 0,\ P(0) = P_0 \geq 0$

$$\Omega = \left\{ (S,\ I,\ R,\ P) \in \mathbb{R}^4 \,|\, 0 < N \leq \frac{\Lambda}{\mu} \right\}$$

is a bounded region.

Proof. For the model, the total population is given by $N(t) = S(t) + I(t) + R(t)$; then:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda - \mu(S+I+R) = \Lambda - \mu N$$

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

The general solution of this differential equation is $N = \frac{\Lambda}{\mu} + C_0 e^{-\mu t}$. The solution with initial condition $N(0) = N_0$ is $N = \frac{\Lambda}{\mu} + (N_0 - 1)e^{-\mu t}$. For $t \to \infty$, it is obtained $N \to \frac{\Lambda}{\mu}$. So, the set $\Omega = \left\{ (S,\ I,\ R,\ P) \in \mathbb{R}^4 \,|\, 0 < N \leq \frac{\Lambda}{\mu} \right\}$ is a bounded region. □

Theorem 2 For any finite, nonnegative initial values $(S_0,\ I_0,\ R_0,\ P_0)$, the trajectories of the solution $(S(t),\ I(t),\ R(t),\ R(t))$ of the system (1) satisfy $S(t) \geq 0,\ I(t) \geq 0,\ R(t) \geq 0,\ R(t) \geq 0$ for all $t \geq 0$.

Proof. Firstly, it is established that all compartments $(S(t),\ I(t),\ R(t),\ R(t))$ defined by the system of Equations are continuously differentiable. As such, if all compartments have nonnegative initial conditions, and if any of the compartments are zero at a given time $t = t_i \geq 0$, its derivative is nonnegative by inspection. Assume that $S_0 = S(0),\ S(t_1) = 0,\ N(t_1) \neq 0$ and $R(t_1) > 0$ at time instant $t = t_1$. Then, the first equation of the model (1) can be rewritten as:

$$\frac{dS(t_1)}{dt} = \Lambda - b_P S(t_1)P(t_1) - b_I S(t_1)I(t_1) - \mu S(t_1) + \alpha R(t_1) = \Lambda + \alpha R(t_1) > 0,$$

then $S(t) \geq S(t_1) = 0$ for t close to $t_1,\ t \geq t_1 > 0$. From the arbitrary of the point t_1 it is following that $S(t) > 0,\ \forall t \geq 0$.

Assume that $I_0 = I(0) \geq 0,\ I(t_2) = 0$ and $S(t_2) > 0,\ P(t_2) > 0,\ N(t_2) > 0$ at time instant $t = t_2$. The second equation from the model (1) can be rewritten as:

$$\frac{dI(t_2)}{dt} = b_P S(t_2)P(t_2) + b_I S(t_2)I(t_2) - (\mu + \gamma)I(t_2) = b_P S(t_2)P(t_2) > 0.$$

From the arbitrary of the t_2 it is following that $I(t) > 0$ for all $t \geq 0$.

Assume that $R_0 = R(0) \geq 0,\ R(t_3) = 0$ and $I(t_3) > 0$ at time instant $t = t_3$. The third equation of the model (1) can be rewritten as:

$$\frac{dR(t_3)}{dt} = \gamma I(t_3) - (\mu + \alpha)R(t_3) = \gamma I(t_3) > 0.$$

From the arbitrary of the t_3 it is following that $R(t) > 0$ for all $t \geq 0$.

Assume that $P_0 = P(0) \geq 0,\ P(t_4) = 0$ and $I(t_4) > 0$ at time instant $t = t_4$. The fifth equation of the model (1) can be rewritten as follows:

$$\frac{dP(t_4)}{dt} = \varepsilon I(t_4) - \delta P(t_4) = \varepsilon I(t_4) > 0.$$

From the arbitrary of the t_4 it is following that $R(t) > 0$, for all $t \geq 0$.

Since none of the compartments would have a negative derivative at any time instant $t = t_i$, when all other compartments are positive, we can conclude that all compartments are nonnegative at any given time instant $t \geq 0$. Consequently, considering that $N(t) = S(t) + I(t) + R(t)$, the total population $N(t)$ is also positive for time instant $t \geq 0$. Hence, the proof is complete. □

From both theorems, we conclude that the model (1) is mathematically and epidemiologically sound.

3.3 *Equilibrium point for a cow population without mastitis*

The equilibrium point of the model (1), which manifests a cow population without mastitis, is given by Theorem 3.

Theorem 3 The equilibrium point of model (1), which manifests a cow population without mastitis, is

$$X^0 = \left(\frac{\Lambda}{\mu},\ 0,\ 0,\ 0\right) \tag{2}$$

Proof. The equilibrium points of models such as the model (1) are determined by setting the right-hand sides of the system's equations equal to zero. Thus, each equation in the system (1) is equated to zero, yielding a new algebraic system. By expressing the variables $R = \dfrac{\gamma}{\mu+\alpha} I$ and $P = \dfrac{\varepsilon}{\delta} I$ from two of the resulting equations accordingly and substituting them into the third equation of the new system, we obtain:

$$\left(b_P S \frac{\varepsilon}{\delta} + b_I S - \mu - \gamma\right) I = 0 \tag{3}$$

If $I = 0$ (e.g., $I^0 = 0$) into (3), then $P = P^0 = 0,\ R = R^0 = 0$. For $I = 0$, we obtain

$$S = S^0 = \frac{\Lambda}{\mu}.$$

The equilibrium point of the model (1), which manifests a cow population without mastitis, is (2). □

3.4 *Basic reproduction number*

The basic reproduction number is a fundamental concept in epidemiology that gives the potential for disease spread in some population. It represents the average number of secondary infections in the susceptible population produced by one infected individual. The finding of the basic reproduction number is given by Theorem 4.

Theorem 4 The basic reproduction number of the model (1) is

$$\Re_0 = \frac{\beta_I + \beta_P}{\mu + \gamma} \tag{4}$$

where $\beta_I = b_I \dfrac{\Lambda}{\mu}$ and $\beta_P = \dfrac{\Lambda}{\mu} \cdot \dfrac{b_P \cdot \varepsilon}{\delta}$.

Proof. The next generation matrix is a square matrix M, where each element represents the transmission rate from one component to another, represented as the difference of two matrices W and Y. The matrix $W(X)$ refers to the rates of emergence of new infections, while $Y(X)$ represents the rates of progression of individuals into and out of the components. The model (1) , according to Theorem 3, has an equilibrium point (2). Let $X = (S,\ I,\ R,\ P)^T$. Then the model (1) can be written as

$$\frac{dX}{dt} = W(X) - Y(X)$$

where

$$W(X) = [0 \quad b_P SP + b_I SI \quad 0 \quad 0]^T$$

$$Y(X) = [b_P SP + b_I SI + \mu S - \alpha R \quad (\mu+\gamma)I \quad (\mu+\alpha)R - \gamma I \quad \delta P - \varepsilon I]^T.$$

For the new generations, it is obtained:

$$F = \begin{bmatrix} b_P SP + b_I SI \\ \\ 0 \end{bmatrix}, \; J(F(X^0)) = \begin{bmatrix} b_I \dfrac{\Lambda}{\mu} & b_P \dfrac{\Lambda}{\mu} \\ \\ 0 & 0 \end{bmatrix}.$$

For the old generations, it is obtained:

$$V = \begin{bmatrix} (\mu+\gamma)I \\ \\ \delta P - \varepsilon I \end{bmatrix}, \; J(V(X^0)) = \begin{bmatrix} \mu+\gamma & 0 \\ \\ -\varepsilon & \delta \end{bmatrix}.$$

The square matrix M has the form:

$$M = J(F(X^0)) \cdot (J(V(X^0)))^{-1} = \frac{1}{\delta(\mu+\gamma)} \cdot \begin{bmatrix} b_I \dfrac{\Lambda}{\mu}\delta + b_P \dfrac{\Lambda}{\mu}\varepsilon & b_P \dfrac{\Lambda}{\mu}(\mu+\gamma) \\ \\ 0 & 0 \end{bmatrix}.$$

The basic reproduction number is

$$\Re_0 = \frac{\Lambda}{\mu} \cdot \frac{b_I \delta + b_P \varepsilon}{\delta(\mu+\gamma)}.$$

If we replace $\beta_I = b_I \dfrac{\Lambda}{\mu}$ and $\beta_P = \dfrac{\Lambda}{\mu} \cdot \dfrac{b_P \cdot \varepsilon}{\delta}$, then the basic reproduction number will have the form (4).

It is known that the basic reproduction number $\Re_0$ represents the average number of secondary infections generated by one infectious individual in a completely susceptible population. According to our model, $\Re_0$ depends on the transmission rates through direct contact (β_I) and indirect contact (β_P), as well as on the recovery rate (γ) and natural death rate (μ).

The significance of the basic reproduction number is illustrated in Figure 2.

Figure 2a presents the behavior of $S(t)$ and $I(t)$ in a model similar to our model (1) when $\Re_0 < 1$, whereas Figure 1b corresponds to the case when $\Re_0 > 1$.

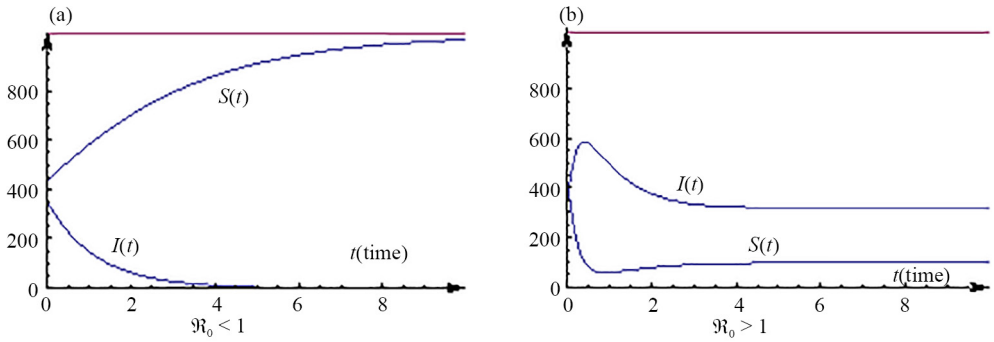

Figure 2. Significance of the basic reproduction number in the mathematical model (1)

It is evident that for $\Re_0 < 1$, $I(t)$ tends to zero and the disease eventually dies out, while for $\Re_0 > 1$, $I(t) > 0$ and the disease becomes endemic within the population.

Remark The red line on the graphs is the number of units in the considered population.

3.5 *Epidemic equilibrium*

The determination of the epidemic equilibrium for the model (1) is provided by Theorem 5.

Theorem 5 The epidemic equilibrium for the model (1) is

$$X^\star = (S^\star, I^\star, R^\star, P^\star)$$

$$= \left(\frac{\Lambda}{\mu \Re_0}, \frac{\Lambda(\mu+\alpha)}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma} \cdot \left(1 - \frac{1}{\Re_0}\right), \frac{\Lambda\gamma}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma} \cdot \left(1 - \frac{1}{\Re_0}\right), \right. \tag{5}$$

$$\left. \frac{\Lambda(\mu+\alpha)\varepsilon}{\delta((\mu+\alpha)(\mu+\gamma)-\alpha\gamma)} \cdot \left(1 - \frac{1}{\Re_0}\right) \right)$$

Proof. If $I \neq 0$ into (4), then $b_P S \frac{\varepsilon}{\delta} + b_I S - \mu - \gamma = 0$. This equation implies

$$S = S^\star = \frac{\Lambda}{\mu \Re_0}.$$

From the first equation, it obtained:

$$I = I^\star = \frac{\Lambda(\mu+\alpha)}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma} \cdot \left(1 - \frac{1}{\Re_0}\right).$$

Then:

$$R = R^{\star} = \frac{\Lambda\gamma}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma} \cdot \left(1 - \frac{1}{\Re_0}\right),$$

$$P = P^{\star} = \frac{\Lambda(\mu+\alpha)\varepsilon}{\delta((\mu+\alpha)(\mu+\gamma)-\alpha\gamma)} \cdot \left(1 - \frac{1}{\Re_0}\right).$$

The epidemic equilibrium is (5). □

Remark It can be observed that for $\Re_0 < 1(\Lambda,\ \alpha,\ \varepsilon,\ \gamma,\ \delta,\ \mu > 0)$, we obtained that $I^{\star},\ R^{\star},\ P^{\star} < 0$. Since we are discussing a real case, this equilibrium point $X^{\star}$ would be impossible under these conditions. Therefore, when we refer to the existence of this equilibrium point $X^{\star}$, we mean in the case of $\Re_0 > 1$.

3.6 *Local stability of equilibrium points*

The local stability of the model (1) is closely related to the basic reproduction number. To analyze the local stability of the model (1), we will use the Routh-Hurwitz stability criterion. The analysis is conducted through the roots of the characteristic equation of the Jacobian matrix of the system (1). If all roots are negative real numbers, then the model (1) is locally stable. Otherwise, it is locally unstable.

Theorem 6 presents the conditions for the local stability of the equilibrium points that manifest a cow population without mastitis.

Theorem 6 (a) The equilibrium point X^0 is locally stable for $\Re_0 < 1$;

(b) If $\Re_0 = 1$, then the equilibrium point X^0 is at the stability limit, i.e. the system at the point X^0 is critically stable.

Proof. The Jacobian matrix of the model (1) at the equilibrium point X^0 is:

$$J(X^0) = \begin{bmatrix} -\mu & -b_I\dfrac{\Lambda}{\mu} & \alpha & -b_P\dfrac{\Lambda}{\mu} \\ 0 & b_I\dfrac{\Lambda}{\mu} - (\mu+\gamma) & 0 & b_P\dfrac{\Lambda}{\mu} \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & \varepsilon & 0 & -\delta \end{bmatrix}.$$

The characteristic polynomial D^0 is

$$D^0 = (\mu+\lambda)(\mu+\alpha+\lambda)\left(\lambda^2 + \lambda\left(\mu+\gamma+\delta - \frac{\Lambda}{\mu}b_I\right) + \delta(\mu+\delta)(1-\Re_0)\right).$$

The clear that $-\mu < 0,\ -\mu-\alpha < 0$ for $\alpha,\ \mu > 0$.

(a) By using Routh-Hurwitz criterion for stability for equation

$$\lambda^2 + \lambda\left(\mu+\gamma+\delta - \frac{\Lambda}{\mu}b_I\right) + \delta(\mu+\delta)(1-\Re_0) = 0,$$

it is true:

$$a_0 = \delta(\mu+\delta)(1-\Re_0) > 0,$$

$$a_1 = (\mu+\gamma)(1-\Re_0) - \frac{\Lambda}{\mu}\frac{b_P\varepsilon}{\delta} + \delta > 0,$$

for $\Re_0 < 1$ and $\Lambda,\ \alpha,\ \varepsilon,\ \gamma,\ \delta,\ \mu,\ b_I,\ b_P > 0$. The signs of the roots of the characteristic equation $D^0 = 0$ are

$$\lambda_1 = -\mu < 0,\ \lambda_2 = -\mu - \alpha < 0,\ \lambda_{3/4} < 0.$$

Therefore, the equilibrium point X^0 is locally stable for $\Re_0 < 1$.

(b) If $\Re_0 = 1$, then the equation

$$\lambda^2 + \lambda\left(\mu+\gamma+\delta-\frac{\Lambda}{\mu}b_I\right) + \delta(\mu+\delta)(1-\Re_0) = 0$$

takes the following form

$$\lambda^2 + \lambda\left(\mu+\gamma+\delta-\frac{\Lambda}{\mu}b_I\right) = 0 \quad i.e., \quad \lambda\left(\lambda+\mu+\gamma+\delta-\frac{\Lambda}{\mu}b_I\right) = 0.$$

The coefficient a_1 is $a_1 = \mu+\gamma+\delta-\frac{\Lambda}{\mu}b_I = \frac{\Lambda}{\mu}\cdot\frac{b_P\varepsilon}{\delta} + \delta > 0$ for $\Lambda,\ \varepsilon,\ \delta,\ \mu,\ b_P > 0$. The signs of the roots of the characteristic equation $D^0 = 0$ are

$$\lambda_1 = -\mu < 0,\ \lambda_2 = -\mu - \alpha < 0,\ \lambda_3 = 0,\ \lambda_4 < 0.$$

We get a root $\lambda_3 = 0$. This main that the system (1) is marginally stable (at the very limit). In control theory, point X^0 is called the stability limit and indicates that the system is "getting ready" to change stability. □

Remark 1 From Theorem 6, it is clear that for $\Re_0 > 1$, all characteristic values of the characteristic equation will not be negative, and therefore the equilibrium point X^0 will be unstable.

Theorem 7 gives the conditions for locally stability of the equilibrium for a mastitis epidemic.

Theorem 7 The equilibrium point $X^\star$ is the local stable for $\Re_0 > 1$.

Proof. The Jacobian matrix of the model (1) at the equilibrium point $X^\star$ is:

$$J(X^\star)=\begin{bmatrix} -b_P\frac{\varepsilon}{\delta}A\left(1-\frac{1}{\Re_0}\right)-b_IA\left(1-\frac{1}{\Re_0}\right)-\mu & -b_I\frac{\Lambda}{\mu\Re_0} & \alpha & -b_P\frac{\Lambda}{\mu\Re_0} \\ b_P\frac{\varepsilon}{\delta}A\left(1-\frac{1}{\Re_0}\right)+b_IA\left(1-\frac{1}{\Re_0}\right) & b_I\frac{\Lambda}{\mu\Re_0}-(\mu+\gamma) & 0 & b_P\frac{\Lambda}{\mu\Re_0} \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & \varepsilon & 0 & -\delta \end{bmatrix}.$$

where $K=\dfrac{\Lambda(\mu+\alpha)}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma}$. The characteristic polynomial $D^\star$ is

$$D^\star=(\mu+\lambda)^2(\lambda^2+\lambda(u-w+\alpha+\gamma+\delta+\mu)+u\alpha-w\alpha+u\gamma+\alpha\gamma+u\delta-w\delta+\alpha\delta+\delta\gamma-C\varepsilon+\delta\mu)$$

where $C=b_P\dfrac{\Lambda}{\mu\Re_0}$, $u=\dfrac{K\mu(\mu+\gamma)}{\Lambda}(\Re_0-1)$, $w=\mu+\gamma-C\dfrac{\varepsilon}{\delta}$. It is clear that: $K,\ C,\ u>0,\ -\mu<0$ for $\Lambda,\ \alpha,\ \varepsilon,\ \gamma,\ \delta,\ \mu,\ b_I,\ b_P>0$ and $\Re_0>1$.

By using Routh-Hurwitz criterion for stability for equation

$$\lambda^2+\lambda(u-w+\alpha+\gamma+\delta+\mu)+u\alpha-w\alpha+u\gamma+\alpha\gamma+u\delta-w\delta+\alpha\delta+\delta\gamma-C\varepsilon+\delta\mu=0,$$

it is true:

$$a_1=\frac{K\mu(\mu+\gamma)}{\Lambda}(\Re_0-1)+C\frac{\varepsilon}{\delta}+\alpha+\gamma+\delta+\mu>0,$$

for $\Lambda,\ \alpha,\ \varepsilon,\ \gamma,\ \delta,\ \mu,\ b_I,\ b_P>0$ and $\Re_0>1$.

Lemma For $\Re_0>1$, it is true

$$a_0=\alpha(u-\mu)+C\frac{\alpha\varepsilon}{\delta}+u\gamma+u\delta+\alpha\delta>0.$$

Proof. Let $\Re_0>1$. Then

$$a_0=\alpha(u-\mu)+C\frac{\alpha\varepsilon}{\delta}+u\gamma+u\delta+\alpha\delta>\frac{\mu(\mu+\gamma)(\mu+\alpha)(\delta+\gamma)+\alpha^2\mu\gamma}{(\mu+\alpha)(\mu+\gamma)-\alpha\gamma}+C\frac{\alpha\varepsilon}{\delta}+\alpha>0,$$

for $C,\ \alpha,\ \varepsilon,\ \gamma,\ \delta,\ \mu>0$. □

The signs of the roots of the characteristic equation $D^\star=0$ are

$$\lambda_{1/2}=-\mu<0,\ \lambda_{3/4}<0.$$

Therefore, the equilibrium point $X^\star$ is locally stable for $\Re_0 > 1$. □

Remark 2 From Theorem 7, it is clear that for $\Re_0 < 1$, all characteristic values of the characteristic equation will not be negative, and therefore the equilibrium point $X^\star$ will be unstable.

3.7 *Global stability of equilibrium points*

In addition to local stability, we will also analyze the global stability of the equilibrium points. To examine the global stability of the equilibrium point X^0, we will use the Castillo-Chavez criterion. We will write the model (1) in the form

$$\begin{aligned} \frac{dX}{dt} &= F(X,\, Y) \\ \frac{dY}{dt} &= G(X,\, Y),\; G(X,\, Y) = 0 \end{aligned} \tag{6}$$

where $X = (S,\, R) \in \mathbb{R}^4$, $Y = (I,\, P) \in \mathbb{R}^4$ are the ineffective and infected states of the system (6), with $I = 0$ and $P = 0$. Regarding the model (1), we will take the reduced system $\frac{dX}{dt} = F(X,\, 0)$ from the system (6), i.e.,

$$\frac{dS}{dt} = \Lambda - \mu S + \alpha R$$

$$\frac{dR}{dt} = -(\mu + \alpha)R.$$

The equilibrium point of the reduced system is $\hat{X} = \left(\frac{\Lambda}{\mu},\, 0\right)$.

The equilibrium point X^0 is globally stable for the model (1) for $\Re < 1$, if the conditions of the Castillo-Chavez criterion are satisfied:

H_1 : For $\frac{dX}{dt} = F(\hat{X},\, 0)$, X^0 is globally stable.

$H_2 : G(X,\, Y) = D_Y G(\hat{X},\, 0)Y - \hat{G}(X,\, Y)$, $\hat{G}(X,\, Y) \geq 0$ for all $(X,\, Y) \in \Omega$, where $D_Y G(\hat{X},\, 0)$ is the Jacobian matrix of $G(X,\, Y)$ at $(I,\, P)$, but it rated at the equilibrium point $X^0 = (\hat{X},\, 0,\, 0)$.

If the system (6) satisfies the conditions of the Castillo-Chavez criterion, then the following theorem will be true.

Theorem 8 The equilibrium point $X^0 = (\hat{X},\, 0,\, 0)$ is globally stable for the model (1) for $\Re < 1$, if the conditions of the Castillo-Chavez criterion are satisfied.

Proof. From the system (1), we make two subsystems $X = (S,\, R) \in \mathbb{R}^4$, $Y = (I,\, P) \in \mathbb{R}^4$ according to (6):

$$F(X,\, Y) = \begin{bmatrix} \Lambda - b_P SP - b_I SI - \mu S + \alpha R \\ \\ \gamma I - (\mu + \alpha)R \end{bmatrix}$$

and

$$G(X,\ Y) = \begin{bmatrix} b_P SP + b_I SI - (\mu+\gamma)I \\ \\ \varepsilon I - \delta P \end{bmatrix}.$$

About the reduced system of the hypothesis H_1, the functions $S(t) \to \dfrac{\Lambda}{\mu}$ and $R(t) \to 0$ are independent of $S(0) = S_0$ and $R(0) = R_0$ accordingly, when $t \to \infty$. We can conclude that the dynamics of the system do not depend on the initial conditions, i.e., every solution of the system with initial conditions in Ω converges to $\widehat{X}$ when $t \to \infty$. Therefore, the equilibrium point $\widehat{X}$ is globally stable in Ω. This implies that the condition H_1 is satisfied.

About the model (1), we have

$$D_Y G(\hat{X},\ 0) = \begin{bmatrix} b_I \dfrac{\Lambda}{\mu} - (\mu+\gamma) & b_P \dfrac{\Lambda}{\mu} \\ \\ \varepsilon & -\delta \end{bmatrix}.$$

Then

$$D_Y G(\hat{X},\ 0)Y = \begin{bmatrix} b_I \dfrac{\Lambda}{\mu} I + b_P \dfrac{\Lambda}{\mu} P - (\mu+\gamma)I \\ \\ \varepsilon I - \delta P \end{bmatrix}.$$

Next

$$\hat{G}(X,\ Y) = \begin{bmatrix} b_I(\dfrac{\Lambda}{\mu} - S)I + b_P(\dfrac{\Lambda}{\mu} - S)P \\ \\ 0 \end{bmatrix} = \begin{bmatrix} \hat{G}_1(X,\ Y) \\ \\ \hat{G}_2(X,\ Y) \end{bmatrix}.$$

Therefore, $\hat{G}_1(X,\ Y) \geq 0$ for $S \leq \dfrac{\Lambda}{\mu}$ at any time t, as shown in H_1. This implies that the condition H_2 is satisfied. It follows that the equilibrium point X^0 is globally stable for $\Re_0 < 1$. It is unstable otherwise. □

About analyzing the global stability of the equilibrium point $X^\star$, we will present Theorem 9.

Theorem 9 If $\Re_0 > 1$, then the equilibrium point $X^\star$ is globally stable in Ω.

Proof. We will analyze the global stability of the equilibrium point $X^\star$ by constructing the following Lyapunov function:

$$V(t) = \left(S - S^\star - S^\star ln\frac{S}{S^\star}\right) + \left(I - I^\star - I^\star ln\frac{I}{I^\star}\right) + \left(R - R^\star - R^\star ln\frac{R}{R^\star}\right) + \frac{b_P S^\star P^\star}{\varepsilon I^\star}\left(P - P^\star - P^\star ln\frac{P}{P^\star}\right).$$

It is clear that $V(t) \geq 0$ for all $S,\ I,\ R,\ P \geq 0$ and that $V(t) = 0$ if and only if $X = X^\star = (S^\star,\ I^\star,\ R^\star,\ P^\star)$.

Differentiating the $V(t)$ and using the equilibrium relations

$$\Lambda = b_P S^\star P^\star + b_I S^\star I^\star + \mu S^\star - \alpha R^\star, \quad (\mu+\gamma) I^\star = b_P S^\star P^\star + b_I S^\star I^\star, \quad \gamma I^\star = (\mu+\alpha) R^\star, \quad \varepsilon I^\star = \delta P^\star,$$

we obtain

$$\dot{V} = -\mu \frac{(S-S^\star)^2}{S} - (\mu+\gamma)\frac{(I-I^\star)^2}{I} - (\mu+\alpha)\frac{(R-R^\star)^2}{R} - \frac{b_P \delta S^\star P^\star}{\varepsilon I^\star}\frac{(P-P^\star)^2}{P} + (residue),$$

where $(residue)$ represents the mixed terms:

$$(residue) = \left(\frac{S^\star}{S} - \frac{I^\star}{I}\right) b_P SP + \left(\frac{S^\star}{S} - \frac{I^\star}{I}\right) b_I SI - \left(\frac{S^\star}{S} - \frac{I^\star}{I}\right)(\mu+\gamma) I^\star + \left(1 - \frac{S^\star}{S}\right)\alpha(R-R^\star) + \left(1 - \frac{R^\star}{R}(I-I^\star)\right)$$

$$+ b_P S^\star P^\star \left(1 - \frac{P^\star}{P}\right)(I-I^\star)$$

By substituting the equilibrium relation, the mixed terms in $(residue)$ cancel out, leaving only negative terms, and thus $\dot{V} \leq 0$ with equality if and only if

$$S = S^\star, \quad I = I^\star, \quad R = R^\star, \quad P = P^\star.$$

Therefore, for the largest compact invariant set Ω, the equilibrium point $X^\star$ is the unique equilibrium point. Thus, LaSalle's invariance principle, all solutions starting in the positive orthant tend to the equilibrium point $X^\star$ as $t \leftrightarrow \infty$. Hence, $X^\star$ is globally asymptotically stable in Ω. □

4. Case study

Given the devastating nature of cow mastitis and its impact on the economy, in this study, we will examine the dynamics of the disease based on real parameters obtained from real data from dairy farm located on the territory of the RSM. The aim is to analyze the spread of mastitis and its impact on the cow productivity and culling from reproduction, to develop effective strategies for mastitis control in dairy herds.

In our real-world farm case, we have a constant number of cows over time, and that number is $N = 1,031$ cows. Maintaining this population relies on the given recruitment rate $\Lambda = \mu N$ of new cows entering the farm.

The initial values of the differential equation system for our case are $S_0 = 439$, $I_0 = 352$, $R_0 = 240$. The time on which the functions in the differential equation system depend will be given in weeks.

During the survey, the mastitis recurred in some cows on the farm, up to four or more times. The recurrence rates were $\alpha_1 = 0.0761$ for once, $\alpha_2 = 0.0393$ for twice, $\alpha_3 = 0.0101$ for three times, and $\alpha_4 = 0.0052$ for four or more times. The risk of having at least one recurrent case of mastitis is given by $\alpha = 0.1307$. This means that 13.07% of cows will be infected at least once during a year.

Based on the explanation for this real situation for mastitis prevalence on dairy farm, in Table 1 the real values of the parameters are given along with their description.

Table 1. Description of the parameters

Parameters	Meaning	Value	Reference
$\Lambda = \mu N$	Recruited rate	231.975	Estimated
b_P	Transmission rate from bacteria in the environment	0.003041	Estimated
b_I	Transmission rate from cow to cow	0.008964	Estimated
γ	Recovery rate	0.6824	Estimated
ε	Shedding rate	0.031746	Assumed [9]
δ	Removal rate of the shed bacteria	0.222	Assumed [9]
μ	Culling rate	0.225	Estimated
α	Mastitis recurrence rate	0.1307	Estimated

With these given parameters, a reproduction number is obtained $\mathfrak{R}_0 \approx 10.6791 > 1$. This means that there are two equilibrium points

$$X^0 = (1{,}031,\ 0,\ 0,\ 0) \quad \text{and} \quad X^\star = (96.5436,\ 320.187,\ 614.269,\ 45.7867).$$

The equilibrium point X^0 is unstable, and $X^\star$ is stable. So, if this situation continues (the parameter values remain approximately the same), we do not have the destruction of the disease over time, but it is smoldering further, and the trajectories of the functions should approach $X^\star$ over time.

This is also seen with the graphical visualization of the model in the next two scenarios:

1. An ideal case in which we assume that impeccable hygienic conditions are maintained on the farm, due to which we have a negligible number of bacteria in the environment that initially do not affect the cause of mastitis in cows. However, over time, some mastitic cows (brought to the farm) shed bacteria into the environment, which contributes to an increase in the number of bacteria, which exceeds the critical number of bacteria for infecting healthy cows. Therefore, in this case, we have the initial value $P_0 = 0$.

2. We assume that the environment is initially approximately in an equilibrium state. For this purpose, we use the last equation of the system. This means that the initial level puts the environment in quasi-equilibrium with $P_0 = \dfrac{\varepsilon \cdot I_0}{\delta} \approx 50.34$ infected cows.

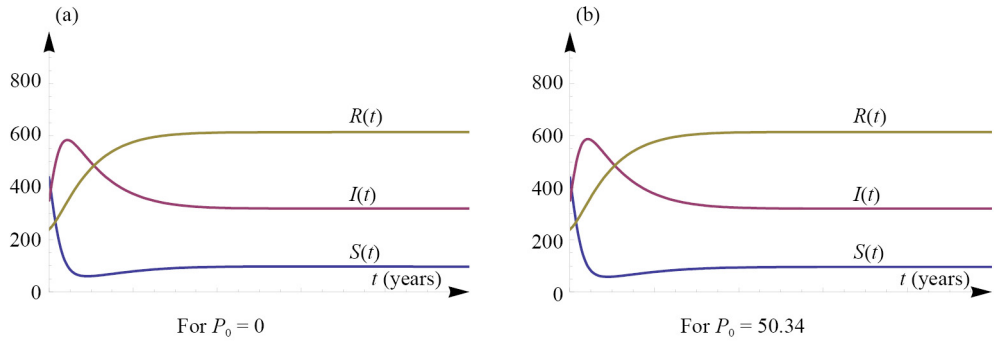

Figure 3. Graphical visualization of the model (1)

Figure 3 shows a prediction of the behavior of bovine mastitis for the next 10 years under existing farm conditions based on the graphical visualization of the mathematical model (1). Clearly, the disease will not disappear in the coming years; instead, it will smolder, i.e., the number of suspects, sick, and recovered cows will tend towards certain values.

A rapid decline in suspected cows is observed, but there is a situation on the farm where an increase in the number of diseased and recovered cows exists. In the first year, the number of diseased cows is greater than the number of suspected and recovered cows, but then that number decreases and becomes smaller than the number of recovered cows. Then, in the following years, the number of diseased cows will be smaller in relation to the number of recovered cows, but the number of suspected cows will decrease significantly in relation to the rest. As the basic reproduction number indicates, the graphical visualization of the model (1) predicts the presence of the mastitis in subsequent years and the convergence of the numbers towards the equilibrium point $X^{\star}$.

Figure 4 shows the graphical visualization of $S(t)$, $I(t)$, and $R(t)$ for the mathematical model (1) with different initial values of P_0. For $P_0 = 0$, the curve is shown in blue, while for $P_0 = 50.34$, it is shown in red. In Figure 4a, the graph of $S(t)$ is displayed. In Figure 4b, the graph of $I(t)$ is shown. In Figure 4c, the graph of $R(t)$ is presented. The graphs not only confirm the previous conclusion regarding the disease's behavior in the cow population but also show that different initial conditions of P_0 lead to the same behavior of susceptible, infected, and recovered cows. This suggests that, for these initial amounts of bacteria in the environment where the cow population lives, we do not have a significant impact on predicting the dynamics of the disease in the years to come.

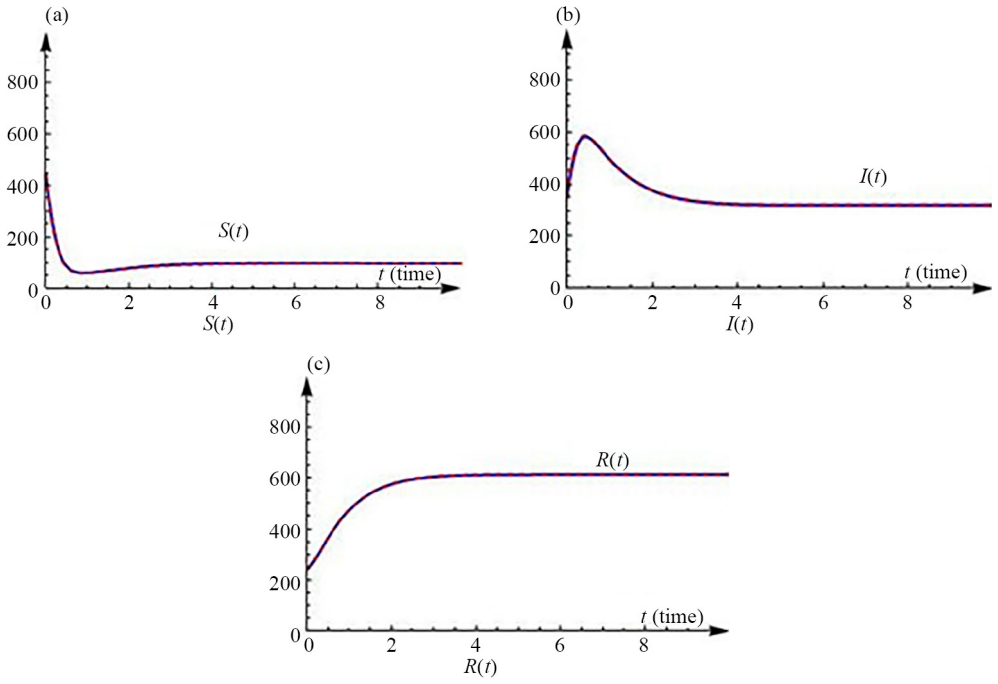

Figure 4. Graphical visualization of $S(t)$, $I(t)$ and $R(t)$ for the model (1)

Table 2 and Table 3 show that the number of suspects, sick, and recovered cows will tend towards the corresponding coordinates of the equilibrium point $X^{\star} = (96.5436,\ 320.187,\ 614.269,\ 45.7867)$.

Table 2. The values of the functions of the model (1) for $P_0 = 0$ per year

Time t (years)	$S(t)$	$I(t)$	$R(t)$
1	60.4891	496.362	474.149
2	78.8632	376.612	575.525
3	91.3637	335.02	604.616
4	96.0778	323.233	611.689
5	97.2217	320.574	613.205
10	96.8799	320.132	613.988

Table 3. The values of the functions of the model (1) for $P_0 = 50.34$ per year

Time t (years)	$S(t)$	$I(t)$	$R(t)$
1	58.6855	496.501	475.813
2	76.8817	377.245	576.873
3	89.0933	335.88	606.026
4	94.0146	323.873	613.113
5	95.5391	320.97	614.491
10	96.3361	320.221	614.443

These tables are in addition to the graphical visualization of the model (1) and the conclusions reached above.

5. Conclusion

Mathematical modeling of mastitis transmission in dairy herds offers valuable insights into understanding, predicting, and controlling this persistent disease. The developed compartmental model, based on ordinary differential equations, effectively captures the key epidemiological processes governing mastitis dynamics, including transmission, recovery, and bacterial shedding. Analytical results verify the model's dimensional consistency and stability, while numerical simulations show how infection levels depend on critical biological parameters such as shedding and recovery rates.

This study successfully develops and analyzes a mathematical model describing the epidemiological behavior of mastitis in dairy cows. The model demonstrates stability and dimensional consistency, offering a theoretical basis for interpreting disease persistence and control within herds. By combining epidemiological concepts with mathematical rigor, the research contributes to a better understanding of mastitis dynamics and establishes a foundation for future applied modeling in veterinary epidemiology.

Using real data from a Macedonian dairy farm, the model predicts that mastitis may persist at a low but steady level if preventive measures are not improved. Employing such mathematical approaches, including those within the SIR framework, emphasizes the importance of herd-specific, risk-based control strategies.

Overall, the study provides a well-founded and practically useful model that can help veterinarians and farm managers optimize mastitis prevention and treatment strategies. Future research should aim to refine the model through better parameter estimation, ongoing data validation, and the integration of additional factors like pathogen diversity and management practices, thereby boosting its predictive power and applicability in real farm conditions.

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Conflict of interest

The authors declare no competing financial interest.

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