

A Dynamical Study of Stability and Bifurcation in an In-Vitro Bovine Mastitis System Incorporating Nonlinear Incidence and Therapeutic Functions



Olusola A. Odebiyi¹, Temitayo O. Oluyo¹, Moses O. Adeyemi², Janet K. Oladejo^{1*}, Olajide Olaoye³, Elizabeth O. Elijah⁴, Olusola W. Ayanrinola¹

¹Department of Pure and Applied Mathematics, Ladoko Akintola University of Technology, Nigeria.

²Department of Mathematics, University of Ilesa, Ilesa, Osun State, Nigeria.

³Department of Applied Mathematics, Silesian University of Technology, Gliwice, Poland.

⁴Department of Mathematics, Federal University of Technology, Minna, Niger state, Nigeria



ABSTRACT: Bovine Mastitis is a common dairy cattle disease characterized by inflammation of the mammary gland typically caused by bacterial infection. It is the leading cause of death and economic damages worldwide and causes reduction in the quality and quantity of milk produced by the infected animals. In fact, it is a potential fatal mammary gland infection that is most common in dairy cattle worldwide. In this work, a mathematical model was developed to study the transmission dynamics and control of Bovine Mastitis. The model incorporated nonlinear transmission and treatment functions, and its epidemiological well-posedness was established by considering its non-negative solutions in feasible regions. Also, Stability analysis revealed that the disease-free equilibrium is stable if the basic reproduction number R_m is less than unity. The sensitivity analysis of the model is performed to show the influential model parameters that have high impact on the R_m . The stability of the model's equilibria implied that the mastitis can be eliminated given that the reproduction number is less than unity. However, the model exhibited backward bifurcation, indicating that $R_m < 1$ is necessary but not sufficient for effective control, and that initial population sizes and disease prevalence also play a crucial role. Sensitivity analysis identified key parameters influencing R_m , including per capita recruitment rate (Λ), transmission rates (β_1) and (β_2), and maximum treatment rate (γ). The numerical results showed that the infectious populations can be effectively controlled thereby reducing the loss and eradication of mastitis in the population with targeted interventions, such as optimizing recruitment, reducing transmission, and maximizing treatment rates.

KEYWORDS: Bovine Mastitis, In-vitro Model, Bifurcation Analysis, Non-linear Incidence, Non-linear Treatment function, Sensitivity Analysis

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I. INTRODUCTION

Bovine mastitis is an inflammatory condition of the udder and remains one of the most challenging veterinary diseases to control. It can be described as an inflammatory reaction in the mammary gland tissue triggered by either physical injury or infection by microorganisms. The disease is regarded as the most common cause of economic loss in the dairy industry due to its impact on milk yield and quality (Wattiaux, 2021). Mastitis is also characterized by persistent inflammation of udder tissue resulting from trauma or microbial invasion, and it continues to be a major source of financial loss in dairy production systems (Ruegg, 2012; Gomes, *et al*, 2016; Wattiaux, 2021; Paramasivam *et al*, 2023; Neculai *et al*, 2022; Lakhani *et al*, 2025). The condition is generally classified into three types based on the severity of inflammation: clinical, subclinical, and chronic mastitis. Clinical mastitis is easily identified because affected cattle exhibit visible symptoms such as swelling and redness of the udder, accompanied by fever. It is typically caused by bacterial infection which leads to inflammation and swelling of the udder, pain and discomfort, reduced milk production and quality, potential

spread to other quarters or animals. The most common microorganisms causing mastitis can be divided into two which are environmental bacteria such as *Streptococcus uberis* spp and *Streptococcus dysgalactive susp* and the second is contagious bacteria such as *Staphylococcus aureus* and *Mycoplasma spp* (Blowey and Edmondson, 1995; NMC, 2011; Ashraf and Imran, 2020; Adeyemi and Oluyo, 2023). In recent years, several researchers have highlighted the ability of many mastitis-causing pathogens to form biofilms, noting that this property may contribute to repeated infections as well as increased resistance to antimicrobial treatments and the host's immune response. Mastitis develops when bacteria often originating from the calf's mouth enter the mammary gland through cracks in the nipple. The affected quarter becomes painful and highly sensitive to touch. When acute mastitis is not properly treated and the inflammation persists, it can progress to chronic mastitis, which damages the milk-secreting tissue and eventually stops milk production. Once these chronic changes occur, they are typically irreversible and permanent (Tewari, 2014; Sharun *et al*, 2021).

*Corresponding author: jkoladejo@lautech.edu.ng

Most likely, other possible sources of infection include long-standing (chronic) mastitis and, in rare cases, an aggressive cancer known as inflammatory carcinoma. Hormonal fluctuations can also contribute by causing the milk ducts to accumulate dead skin cells and debris, leading to blockage. These obstructed ducts create favourable conditions for bacterial invasion, and infections may recur even after antibiotic treatment. Other symptoms of Mastitis are Fatigue, breast enlargement, tenderness and swelling, fever and chills, Abscess, and persistent fever. The main environmental sources of mastitis-causing pathogens are the cow's surroundings. This group commonly includes Streptococcus species and Gram-negative bacteria such as *Escherichia coli* and *Klebsiella*. Fungal organisms can also be involved, including *Aspergillus fumigatus*, *A. niger*, *Candida* spp., *Trichosporon* spp., and others (NMC, 2011; Cobirka, *et al*, 2020; Ashraf and Imran, 2020; Sharun *et al*, 2021; Stanek *et al*, 2024; Abebe *et al*, 2023). Physical injuries to the udder, inadequate hygiene, or trauma can further predispose animals to mastitis. Affected cows often resist udder handling and may kick to avoid being milked. When milk is expressed, it may contain blood clots, foul-smelling brownish discharge, or abnormal clumps. Milk production may cease entirely or become severely reduced, and the animal's body temperature frequently rises. Additional signs include reduced appetite, difficulty moving due to pain and swelling, and a hot, tender mammary gland that becomes painful even with light touch.

Preventing mastitis before it develops into a serious problem is far more effective than treating it afterward. The disease can be identified at an early, symptom-free stage (subclinical mastitis) using the California Mastitis Test, a simple and rapid procedure performed on small milk samples. Regular physical examination of the udder, along with diagnostic screening for early detection, is essential for reducing losses associated with both subclinical and clinical mastitis (Arsenopoulos *et al*, 2018; Cobirka *et al*, 2020; Adeyemi and Oluyo, 2023). Controlling subclinical infections is particularly important because animals with subclinical mastitis serve as reservoirs for pathogens that can spread to other cows, and most clinical cases begin as subclinical infections (Tewari, 2014; Cobirka *et al*, 2020; Abebe, *et al*, 2023). Consequently, reducing subclinical mastitis remains the most effective strategy for lowering the incidence of clinical disease (Tewari, 2014; Wattiaux, 2021; Adeyemi and Oluyo, 2023).

Mastitis is often diagnosed through visible symptoms and clinical examination and typically affects only one quarter of the udder. Treatment usually involves administering antibiotics and ensuring the affected quarter is regularly emptied of milk. In some situations, the infection may progress to form a breast abscess. Such abscesses require drainage either surgically or through needle aspiration, sometimes with ultrasound guidance followed by antibiotic therapy. Early detection is crucial in preventing the progression of the disease to more severe clinical stages that cause substantial economic losses for dairy farmers. For this reason, timely drug therapy is important for maintaining productivity (NMC, 2011; Cobirka *et al*, 2020; Smulski *et al*, 2020; Stanek *et al*, 2021). Administration of antibiotics, Polymeric nanoparticles

treatment and anti-inflammatory therapy is also highly required (Ruegg, 2012; Ashraf and Imran, 2020; Sharun *et al*, 2021; Haider *et al*, 2023). In other to prevent this ugly disease, measures such as providing clean, dry and adequate bedding for cows to lie, clean milking environment after milking and other strict hygiene measures has to be maintained (Fernanda *et al*, 2016; Ashraf and Imran, 2020). Likewise, using of germicidal treat dips and insecticides application such as pyrethroids, organophosphates, methoprene and abamectins are also an excellent measure of mastitis disease prevention and control of mastitis disease. Which is aimed at the prevention of transmission of mastitis from udder through vector (flies) to an infected cow, cattle-cattle transmission as well as cattle to flies transmission (Yeruham *et al*, 1996; Arnold, 2019; Neculai *et al*, 2022). Mastitis may spread through direct contact between infected animals and susceptible ones, which can become infected by inhaling contaminated droplets present in the environment. Transmission can also occur via vectors, such as horn flies and possibly houseflies, which can carry the pathogens from one animal to another (Yeruham, *et al*, 1996; Aligaz and Munganga, 2018; Abebe, *et al*, 2023; Ashraf and Imran, 2020). Clinical researches also showed the transmission through flies to cattle and cattle-cattle. Animals affected by mastitis may occasionally recover without intervention, but drug treatment is typically required to sustain productivity. Management often involves systemic and intramammary antibiotic administration, along with supportive fluids, polymeric nanoparticles treatment and anti-inflammatory therapy (Ashraf and Imran, 2020; Abebe, *et al*, 2023; Haider, *et al*, 2023). Despite many years of applying various control measures, mastitis continues to present significant economic challenges and remains one of the costliest health problems in dairy cattle worldwide (Paramasivam, *et al*, 2023, Stanek, *et al*, 2024). The economic consequences of mastitis and its relevance to public health must be accurately assessed, without overstating its significance. The Farm Animal Welfare Council's report at United Kingdom drew attention and emphasized the importance of addressing mastitis, given its substantial implications for animal welfare (Arnold, 2019; Barrett, 2024; Paramasivam 2023). In this vein, we present a six-compartment mathematical model for the study of mastitis disease dynamics in cattle population with nonlinear incidence and treatment function as no other study have done so.

Mathematical models are widely used to design strategies for controlling disease outbreaks and to assist in selecting the most effective treatments for reducing infection spread. The basic reproduction number R_0 is a key metric that helps healthcare planners and epidemiologists predict whether an infection will die out within a population (Odebiyi, *et al*, 2024). The incidence rate is also a critical component in understanding the dynamics of a disease. Traditionally, epidemic models have employed bilinear incidence rates of the form βuv and standard incidence rates of the form $\frac{\beta xy}{N}$, where β represents the transmission rate per contact, u and v are the susceptible and infected populations, respectively, and N is the total population (Wang and Zhang, 2010). Several studies have explored nonlinear forms of incidence rates (Jin

et al., 2007; Jana *et al.*, 2015; Dubey *et al.*, 2013; 2016). Saturated incidence rates were introduced in the form $\frac{\beta uv}{(1+\gamma v)}$, where βv represents the force of infection and $\frac{1}{(1+\gamma v)}$ measures the inhibition factor arising from crowding among infected individuals and social awareness measures in the population (Jana *et al.*, 2015; Dubey *et al.*, 2016; Saha and Ghosh, 2021, 2023). Non-monotone incidence rates, such as $\frac{\beta uv}{(1+\gamma v^2)}$, have also been proposed to account for psychological or behavioural effects in the population (Saha and Ghosh, 2023; Saha *et al.*, 2023). Additionally, saturated incidence rates of the form $\frac{\beta SI}{(1+\psi I)}$ have been introduced, where βI measures the force of infection in a fully susceptible population and $\frac{1}{(1+\psi I)}$ captures the inhibitory effect due to behavioural changes in susceptible (Zhang, *et al.*, 2008; Odebiyi *et al.*, 2025).

The saturated incidence rate of the form $\frac{1}{(1+\psi S)}$ where ψ is the saturation factor which is positive constant and reflects the impact of epidemic control measures and the reduction in effective contacts between infective and susceptible individuals. It accounts for interventions or protective behaviours that limit disease transmission, emphasizing the importance of incorporating behavioural and social factors into epidemic modelling.

In recent years, several authors have investigated epidemiological models incorporating non-linear incidence rates. A commonly used form is $\beta I^p S^q$ where S and I represent the numbers of susceptible and infective individuals, respectively, and p and q are positive constants. Models using this incidence rate have been explored by (Liu *et al.*, 1987), while other studies have also incorporated it into their frameworks (Wei and Chen, 2008; Zhang, *et al.*, 2008). Independently, (Beddington and De-Angelis, 1975) introduced a non-linear incidence rate $\left(\frac{\psi}{(1+\beta S)}\right)$ known as the Beddington–DeAngelis type, which has since been employed by several researchers to describe epidemiological dynamics. Although Susceptible–Infected–Recovered (SIR) and Susceptible–Infected–Susceptible (SIS) models with saturated incidence rates are well-studied in the literature, relatively few studies have examined saturated treatment functions for the study of Bovine Mastitis in cattle, even within SEIR epidemic models.

Likewise, mathematical models exhibiting various forms of bifurcation and their epidemiological implications have been widely investigated in the literature such as (Wang, 2006; Sharomi *et al.*, 2007; Hu *et al.*, 2008; Zhang and Liu *et al.*, 2008; Gumel, 2012; Hu *et al.*, 2012; Dubey *et al.*, 2013; Upadhyay *et al.*, 2019; Lan *et al.*, 2021; Bentout *et al.*, 2021; Omede *et al.*, 2023; Saha *et al.*, 2023;). These include forward and backward bifurcations, Hopf bifurcations, and transcritical bifurcations, all of which play critical roles in determining disease persistence, oscillatory outbreaks, and control thresholds (Castillo-Chavez and Song, 2004; Dushoff *et al.*, 1998; Hethcote and van den Driessche, 1991; Kribs-Zaleta and Velasco-Hernández, 2000; van den Driessche and Watmough, 2002). Bifurcation in infectious disease models occurs when

small variations in key parameters result in qualitative changes in disease dynamics, such as transitions between disease-free and endemic equilibria. A principal driver of such behaviour is variation in the basic reproduction number R_0 , with bifurcation commonly occurring at the critical threshold $R_0 = 1$, which determines whether infection dies out or persists (van den Driessche and Watmough, 2002). Nonlinear incidence functions, treatment saturation, and imperfect control strategies can introduce multiple equilibria and give rise to backward bifurcation, allowing disease persistence even when $R_0 < 1$ (Castillo-Chavez and Song, 2004). Population heterogeneity including age structure, risk groups, and spatial variation also contributes to bifurcation phenomena by modifying contact patterns and susceptibility levels (Hethcote, 2000). Additionally, the incorporation of latency periods, time delays, or exogenous reinfection can generate complex dynamics such as oscillatory behaviour and multiple endemic equilibria (Castillo-Chavez and Feng, 1996). Overall, bifurcation reflects the combined effects of nonlinear feedback mechanisms and threshold dynamics inherent in infectious disease transmission and control processes.

For example, Castillo-Chavez and Feng (1996) in their paper presents a series of mathematical models, with particular emphasis on antibiotic resistance, latency, reinfection, vaccination strategies, and demographic influences. A two-strain TB model demonstrated that coexistence of strains is rare when resistance is independent but becomes likely when resistance emerges due to poor treatment compliance. Results shows that models incorporating distributed latency showed dynamics similar to those with exponentially distributed latency, with disease outcomes determined primarily by whether the basic reproduction number is above or below unity. Inclusion of exogenous reinfection produced qualitatively different dynamics, allowing subcritical bifurcation and multiple endemic equilibria even when the basic reproduction number is less than one. Additional age-structured and demographic models highlighted the role of vaccination optimization, HIV co-infection, and immigration in shaping TB transmission patterns. Kenneth *et al.* (2014) also addresses the limited analytical work available by performing a bifurcation analysis of an existing mathematical model describing the early stages of type 1 diabetes in diabetes-prone NOD mice, based on available biological data. The analysis establishes positivity of solutions and the existence of a family of compact attracting trapping regions in the positive five-dimensional cone, ensuring practical weak persistence of the system. These trapping regions converge to a smaller invariant set defined by their intersection, providing insight into long-term system behaviour. Based on the model outcomes, four novel treatment strategies are proposed, with the introduction of additional macrophages at the inflammation site emerging as the most promising approach despite its initially counter-intuitive nature.

Likewise, in Shafeeq *et al.* (2022) a time-delayed vaccination model designed to analyse and forecast the progression of COVID-19 infections was introduced. The primary objective is to demonstrate the occurrence of Hopf bifurcation, which provides a mathematical explanation for the

recurrent epidemic waves observed globally. The model incorporates delays associated with vaccine dosing intervals, allowing an assessment of how varying time gaps between doses influence infection dynamics across different vaccine types. Analytical results show that the inclusion of vaccination delays can induce Hopf bifurcation, leading to sustained oscillations and continued disease persistence. Their theoretical findings are supported by numerical simulations and graphical illustrations, offering practical insights into effective vaccination strategies for controlling the spread of the disease. Delay differential equation models have also been applied to Parkinson’s and Huntington’s diseases to investigate the role of synchronization within basal ganglia pathways by Elfouly (2024). Using a delay-based RLC (resistor, inductor, capacitor) circuit framework, their study has shown how the direct, indirect, and hyperdirect pathways respectively excite, inhibit, and regulate motor cortex activity, with Hopf bifurcation analysis revealing transitions between stable and oscillatory dynamics. Model simulations indicate that altered response-time delays in these pathways can reproduce key clinical features, including tremor, bradykinesia, and inhibited motor activity in Parkinson’s disease. Additionally, increased delays or desynchronization among pathways can generate chaotic dynamics, providing a mechanistic explanation for movement disorders such as Huntington’s disease.

Mathematical modelling has been widely used to explore the dynamics of infectious diseases across populations. In Ahmad *et al* (2024), a fractional-order SEIBR model incorporating an ABC operator was developed to investigate the impact of early diagnosis, immune system strengthening, and remedial interventions with and without drugs on cholera transmission. The model’s stability, reproductive number, and sensitivity to key parameters were analysed, and simulations illustrated how different intervention strategies influence the spread of the disease, providing guidance for effective control measures. Kiseleva *et al* (2024) further develops and provides theoretical justification for an original mathematical framework to model the transmission of infectious diseases, using COVID-19 as a case study. The proposed approach employs a system of autonomous differential equations designed to capture complex disease dynamics. Model validity is demonstrated through its application to COVID-19 data from one of Ukraine’s most populous and geographically extensive regions. Its practical relevance lies in its adaptability for real-world use, supporting informed decision-making and public health planning. Additionally, the model enables evaluation of system behaviour and identification of key epidemiological parameters, offering valuable insights for effective disease management and response.

The present study aims to investigate the stability and bifurcation behaviour of an in-vitro model of bovine mastitis incorporating non-linear incidence and treatment functions. The paper is structured as follows: Section 2 presents the model formulation, establishing the existence and uniqueness of its solutions. The disease-free and endemic equilibrium points are derived, and both local and global stability analyses are conducted. Bifurcation analysis is performed using the Center Manifold theorem, demonstrating the presence of backward bifurcation and determining the local stability of the

endemic equilibrium, which is critical for disease eradication. Sensitivity analysis is also conducted to identify the most influential parameters affecting the basic reproduction number. Section 3 provides numerical simulations to support the theoretical results, and Section 4 concludes the study with key findings and implications.

II. FORMULATION AND PROPERTIES OF THE MASTITIS MODEL

A. Mathematical Formulation

In this work, the cattle population is subdivided into the subpopulations of six classes consisting of susceptible class $S(t)$, Exposed Class $E(t)$, Subclinical infective class $A(t)$, Clinical infective class $I(t)$, Chronic infective class $C(t)$, and the Recovered Class $R(t)$. The total population of cattle at any time, t , is: $N(t) = S(t) + E(t) + A(t) + I(t) + C(t) + R(t)$

An SIR model with saturated incidence rate $\left(\frac{\beta_1 A}{1+\alpha_1 A} + \frac{\beta_2 I}{1+\alpha_2 I}\right)$ is explored. Susceptible cattle make effective contact with infected animals in the subclinical or clinical stages, after which they transition from the exposed class to the subclinical class at a rate (ρ) and a fraction of them moved to the clinical class. Some cattle died naturally in all of the classes. At the subclinical infective compartment, some cattle develop into clinical infective (class) at a rate (σ) and some progresses to chronic class at a rate (θ) . Also while still receiving treatment at clinical class, some progresses to chronic class due to delay in treatment or failed treatment while some moves to recovered class at a rate (γ) .

The interactions between the cattle-cattle transmission are thereby depicted by the diagrammatic representation in Figure 1.

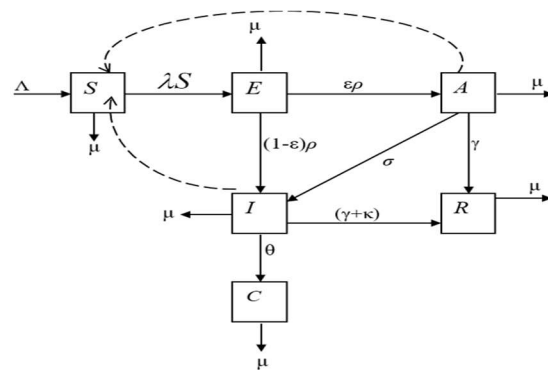


Figure 1: The Mastitis Model’s Schematic Diagram

B. Model’s Assumptions

Other assumptions taken into consideration while formulating the model (1) are

- i. The population is defined as a dynamic system because its size and composition change continually through immigration, emigration, births, and deaths. Biologically, these processes drive natural turnover, shaping age structure, reproductive potential, and genetic variability within the population. Epidemiologically, such demographic shifts influence population density and contact patterns, thereby

affecting disease transmission rates, the distribution of susceptible and immune individuals, and the overall behaviour of infectious or non-infectious health outcomes across time. The model assumes homogeneous mixing, meaning all cattle in the population have an equal chance of contracting mastitis upon effective contact with infected cattle. Transmission of the disease is modelled using a saturated incidence rate.

- ii. Given that the probability of survival to the infectious stage is less than unity. We therefore incorporated an exposed class in to the model to capture the dynamics of cattle that are exposed to mastitis.
- iii. It is assumed that chronically infected cattle do not transmit mastitis to others because of their weakened state, and as a result, they are removed from the herd at a rate d , due to curling (Adeyemi and Oluyo, 2023).
- iv. Spontaneous recovery from mastitis in individual cattle following the administration of antibiotic and anti-inflammatory treatment been administered with (Smulski et al, 2020).
- v. Deaths caused by mastitis occur only during the chronic stage of infection, at a rate δ , in addition to natural mortality in the population (Ashraf and Imran, 2020).

Based on the above assumptions and the interactions illustrated in the schematic diagram in Figure 1, a system of ordinary differential equations is formulated. These equations incorporate a saturated incidence rate and a non-linear treatment function, and serve as the governing equations for the model.

$$\left. \begin{aligned} \frac{dS}{dt} &= \Lambda - \left(\frac{\beta_1 A}{1 + \alpha_1 A} + \frac{\beta_2 I}{1 + \alpha_2 I} \right) S - \mu S \\ \frac{dE}{dt} &= \left(\frac{\beta_1 A}{1 + \alpha_1 A} + \frac{\beta_2 I}{1 + \alpha_2 I} \right) S - (\mu + \rho) E \\ \frac{dA}{dt} &= \rho E - (\mu + \sigma + \gamma) A \\ \frac{dI}{dt} &= (1 - \varepsilon) \rho E + \sigma A - \left(\mu + \theta + \gamma + \frac{\alpha \tau}{1 + b \tau I} \right) I \\ \frac{dC}{dt} &= \theta I - (\mu + \delta + d) C \\ \frac{dR}{dt} &= \gamma A + \left(\gamma + \frac{\alpha \tau}{1 + b \tau I} \right) I - \mu R; \end{aligned} \right\} \quad (1)$$

subject to the initial conditions: $S(0) \geq 0; E(0) \geq 0; A(0) \geq 0; I(0) \geq 0, C(0) \geq 0; R(0) \geq 0$

The variables and parameters of the model are described in Tables 1 and 2.

Table 1: Model's Parameters and description

Parameters	Narrative	Value (day^{-1})	Source
Λ	Per capital recruitment rate	$\frac{1}{5 \times 365}$	(Aligaz, and Munganga, 2018)
β_1	Transmission rates for subclinical infectious cattle	0.00230	(Adeyemi and Oluyo, 2023)
β_2	Transmission rates for clinical infectious cattle	0.00235	(Adeyemi and Oluyo, 2023)
α_1	Inhibition coefficient for $A(t)$	1.005	(Adeyemi and Oluyo, 2023)
α_2	Inhibition coefficient for $I(t)$	1.015	(Adeyemi and Oluyo, 2023)
ρ	Progression rate out from Exposed class	$\frac{1}{6 \times 7}$	(Aligaz, and Munganga, 2018)
σ	Rate of developing clinical symptoms	1.005	(Adeyemi and Oluyo, 2023)
θ	Rate of developing into chronic stage	0.010	(Adeyemi and Oluyo, 2023)
μ	Per capital natural mortality	$\frac{1}{5 \times 365}$	(Aligaz, and Munganga, 2018)
δ	Mastitis induced death rate	0.005	(Aligaz, and Munganga, 2018)
ε	Proportion of exposed cattle that develop subclinical cases	0.333	(Adeyemi and Oluyo, 2023)
γ	Natural recovery rate	0.015	(Adeyemi and Oluyo, 2023)
τ	Maximum rate of treatment	$\frac{1}{28}$	(Aligaz, and Munganga, 2018)
a	Rate of cure of the treatment	6.5	(Aligaz, and Munganga, 2018)
b	Rate of delay in treatment	5.0	(Aligaz, and Munganga, 2018)
d	Rate of curling of chronic infective cattle	0.0025	(Adeyemi and Oluyo, 2023)

Table 2: Model Variables

Variables	Narrative
$S(t)$	Susceptible class
$E(t)$	Exposed class
$A(t)$	Subclinical infective class
$I(t)$	Clinical infective class
$C(t)$	Chronic infective class
$R(t)$	Recovered Class

C. Positivity and Boundedness of Solutions of the Model

From model (2.1), it can be demonstrated that all state variables remain non-negative, and the solutions stay positive for all $t \geq 0$. All model parameters are assumed to be positive.

Theorem 1: Let the initial values of the state variables satisfy $\{(S(0) \geq 0, E(0) \geq 0, A(0) \geq 0, I(0) \geq 0, C(0) \geq 0, R(0) \geq 0) \in \Omega\}$ Then the set $\{S(t), E(t), A(t), I(t), C(t), R(t)\}$ remains non-negative in Ω for all $t \geq 0$.

Proof: To establish the positivity of the state variables, we begin by examining the first equation in system (2.1) and proceed accordingly.

Using the approach of (Adeyemi & Oluyo 2023; Odebiyi et al, 2025)

$$\frac{dS}{dt} \geq -(\lambda + \mu)S$$

$$\int \frac{1}{S} dS \geq - \int (\lambda + \mu) dt$$

$$S(t) \geq S_0 e^{-(\lambda + \mu)t} \geq 0$$

Hence, $S(t) \geq 0$ (2)

Next, considering the second equation in system (1)

$$\frac{dE}{dt} = \left(\frac{\beta_1 A}{1 + \alpha_1 A} + \frac{\beta_2 I}{1 + \alpha_2 I} \right) S - (\mu + \rho)E$$

$$\frac{dE}{dt} \geq -(\mu + \rho)E$$

$$\int \frac{1}{E} dE \geq - \int (\mu + \rho) dt$$

$$E(t) \geq E_0 e^{-(\mu + \rho)t} \geq 0$$

Hence, $E(t) \geq 0$. (3)

Also considering the third equation in (1)

$$\frac{dA}{dt} = \varepsilon \rho E - (\mu + \sigma + \gamma)A$$

$$\frac{dA}{dt} \geq -(\mu + \sigma + \gamma)A$$

$$\int \frac{1}{A} dA \geq \int -(\mu + \sigma + \gamma) dt$$

$$A(t) \geq A_0 e^{-(\mu + \sigma + \gamma)t} \geq 0$$

Hence, $A(t) \geq A_0$. (4)

Also, considering the fourth equation in (1)

$$\frac{dI}{dt} = (1 - \varepsilon)\rho E + \sigma A - \left(\mu + \theta + \gamma + \frac{a\tau}{1 + b\tau I} \right) I$$

$$\frac{dI}{dt} + \left(\mu + \theta + \gamma + \frac{a\tau}{1 + b\tau I} \right) I > 0$$

$$\frac{dI}{dt} e^{\int \left(\mu + \theta + \gamma + \frac{a\tau}{1 + b\tau I(w)} \right) dw} + (\mu + \theta + \gamma) I > 0$$

$$I(t) e^{\int \left(\mu + \theta + \gamma + \frac{a\tau}{1 + b\tau I(w)} \right) dw} + (\mu + \theta + \gamma) I - I(0) > 0$$

$$I(t) > I(0) e^{-\int \left(\mu + \theta + \gamma + \frac{a\tau}{1 + b\tau I(w)} \right) dw} + (\mu + \theta + \gamma) I$$

Hence, $I(t) \geq 0$. (5)

Considering the fifth equation in (1)

$$\frac{dC}{dt} \geq \theta I - (\mu + \delta + d)C$$

$$\int \frac{1}{C} dC \geq - \int (\mu + \delta + \gamma) dt$$

$$C(t) \geq C_0 e^{-(\mu + \delta + \gamma)t} \geq 0$$

Hence, $C(t) \geq 0$. (6)

Considering the sixth equation in (1)

$$\frac{dR}{dt} = \gamma A + \left(\gamma + \frac{a\tau}{1 + b\tau I} \right) I - \mu R$$

$$\frac{dR}{dt} + \mu R \geq 0$$

$$\frac{dR}{dt} e^{\int \mu dt} \geq 0$$

$$R(t) e^{\int \mu dt} - R(0) \geq 0$$

$$R(t) \geq R(0) e^{-\int \mu dt}$$

Hence, $R(t) \geq 0$. (7)

we similarly analyse the terms to ensure that the corresponding state variable remains non-negative for all $t \geq 0$

Theorem 2: Every solution within the region $\Omega = \{(S(t), E(t), I(t), A(t), C(t), R(t)) \in \mathfrak{R}_+^6, N(t) \leq \frac{\Lambda}{\mu}\}$ is

positively invariant with respect to the mastitis model (1) in cattle populations. That is, the solutions of system (1) remain within Ω for all $t \geq 0$.

Proof: Summing all the derivatives of the state variables in model (1) with respect to time t , we obtain

$$\frac{dN(t)}{dt} = \Lambda + \mu(S + E + A + I + C + R) + (\mu + \delta + d)C + \mu R$$

$$\Rightarrow \frac{dN}{dt} \leq \Lambda + \mu N$$

$$\Rightarrow N \leq \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Where N_0 represents the initial population size. Integrating this, it follows that,

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu} \tag{9}$$

This implies that the solutions of the mastitis model (1) are non-negative and bounded within the feasible region Ω . Therefore, all trajectories that start within Ω remain inside Ω or eventually enter it, confirming that the model is epidemiologically well-posed. Consequently, it is valid to study the dynamics of the system within the feasible region Ω .

III. MATHEMATICAL ANALYSIS OF THE MODEL

A. Mastitis-free equilibrium

When mastitis is not present in the herd, $S(t) \neq 0, E(t) = A(t) = I(t) = C(t) = R(t) = 0$ the system settles into a mastitis-free equilibrium state, E_0 , which is determined by solving model equations (1) under the condition

$$\frac{dS}{dt} = 0, \frac{dE}{dt} = 0, \frac{dA}{dt} = 0, \frac{dI}{dt} = 0, \frac{dC}{dt} = 0, \frac{dR}{dt} = 0. \tag{10}$$

The Mastitis free equilibrium is obtained as

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right) \tag{11}$$

B. Reproduction Number R_m

Determining the basic reproduction number is a crucial step. The quantity R_m represents the expected number of new infections generated by a single infectious individual over the

course of their infectious period. This concept applies to models that describe how a disease spreads within a population. In compartmental epidemic models, the population is divided into various disease-related classes, while the remaining groups form the non-infectious compartments. Thus, a general compartmental disease transmission model can be written in terms of F_i representing new infections rate in the i th disease compartment and the V_i stands for the transition terms.

Using the next generation matrix approach described by (Diekman et al, 1990; Hethcote, 2000; Van den Driessche and Watmough, 2002], the basic reproduction number is defined as the spectral radius of FV^{-1} [30], that is, $R_m = \rho(FV^{-1})$,

$$\text{where } F = \begin{bmatrix} 0 & \frac{\beta_1 \Lambda}{\mu} & \frac{\beta_2 \Lambda}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} G_1 & 0 & 0 & 0 \\ -\varepsilon \rho & G_2 & 0 & 0 \\ -(1-\varepsilon)\rho & -\sigma & G_3 + a\tau & 0 \\ 0 & 0 & -\theta & G_4 \end{bmatrix} \tag{12}$$

with $G_1 = \mu + \rho, G_2 = \mu + \sigma + \gamma, G_3 = \mu + \theta + \gamma, G_4 = \mu + \delta + d,$

$$V^{-1} = \frac{1}{G_1 G_2 (G_3 + a\tau)} \begin{bmatrix} G_2 (G_3 + a\tau) G_4 & 0 & 0 & 0 \\ \varepsilon \rho (G_3 + a\tau) G_4 & G_1 (G_3 + a\tau) G_4 & 0 & 0 \\ \varepsilon \rho \sigma G_4 + G_2 (1 - \varepsilon) G_4 & \sigma G_1 G_4 & G_1 G_2 G_4 & 0 \\ \varepsilon \rho \sigma \theta + (1 - \varepsilon) \rho \theta G_2 & G_1 \sigma \theta & G_1 G_2 \theta & G_1 G_2 (G_3 + a\tau) \end{bmatrix}$$

The next generation matrix, G , is $G = FV^{-1}$,

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1 \varepsilon \rho S}{G_1 G_2} + \frac{\beta_2 S (\varepsilon \rho \sigma + (1 - \varepsilon) \rho G_2)}{G_1 G_2 (G_3 + a\tau)} & \frac{\beta_1 S}{G_2} & \frac{\beta_1 S \sigma}{G_2 (G_3 + a\tau)} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{13}$$

Therefore, the effective reproduction number is the leading eigenvalue of the matrix FV^{-1} , computed as the dominant eigenvalue of G obtained by the characteristic equation of FV^{-1} , and the dominant eigenvalue is the basic reproduction number for mastitis. That is,

$$\lambda = \frac{\Lambda \rho (a \varepsilon \tau \beta_1 + \varepsilon \sigma \beta_2 - \varepsilon G_2 \beta_2 + \varepsilon G_3 \beta_1 + G_2 \beta_2)}{\mu G_1 G_2 (a \tau + G_3)}$$

Hence,

$$R_m = \frac{\Lambda \rho (a \varepsilon \tau \beta_1 + \varepsilon \sigma \beta_2 - \varepsilon G_2 \beta_2 + \varepsilon G_3 \beta_1 + G_2 \beta_2)}{\mu G_1 G_2 (a \tau + G_3)} \tag{14}$$

C. Mastitis endemic equilibrium

In the presence of mastitis, $S(t) \neq 0, E(t) \neq 0, A(t) \neq 0, I(t) \neq 0, C(t) \neq 0, R(t) \neq 0$, then we solve from equation (1) for S, E, A, I, C, R simultaneously to obtain

$$C = \frac{\theta I}{G_4}, \text{ where } G_4 = \mu + \delta + d,$$

$$E = \frac{G_2 \{G_3 (1 + b\tau I) + a\tau\} I}{(1 + b\tau I) (1 - \varepsilon) (\rho G_2 + \sigma \varepsilon \rho)}$$

Where $G_2 = \mu + \sigma + \gamma, G_3 = \mu + \theta + \gamma,$

$$A = \left(\frac{\varepsilon \rho}{G_2} \frac{G_2 \{G_3 (1 + b\tau I) + a\tau\} I}{(1 + b\tau I) (1 - \varepsilon) (\rho G_2 + \sigma \varepsilon \rho)} \right)$$

where $G_4 = \mu + \delta + d,$

$$R = \frac{1}{\mu} \left\{ \frac{\gamma \varepsilon \rho [G_3 (1 + b\tau I) + a\tau] I}{(1 + b\tau I) (1 - \varepsilon) (\rho G_2 + \sigma \varepsilon \rho)} + \frac{[\gamma (1 + b\tau I) + a\tau] I}{1 + b\tau I} \right\}$$

$$S = \frac{\Lambda (1 + \alpha_2 I) \left(1 + \alpha_1 \left(\frac{\varepsilon \rho}{G_2} \frac{G_2 \{G_3 (1 + b\tau I) + a\tau\} I}{(1 + b\tau I) (1 - \varepsilon) (\rho G_2 + \sigma \varepsilon \rho)} \right) \right)}{\left(\frac{\varepsilon \rho}{G_2} \frac{G_2 \{G_3 (1 + b\tau I) + a\tau\} I}{(1 + b\tau I) (1 - \varepsilon) (\rho G_2 + \sigma \varepsilon \rho)} \right) (\beta_1 + (\beta_2 I + \mu (1 + \alpha_2 I)) \alpha_1) + \beta_2 I + \mu (1 + \alpha_2 I)}$$

where, I is obtained as the roots of the equation

$$P_1 I^4 + P_2 I^3 + P_3 I^2 + P_4 I + P_5 = 0 \tag{15}$$

with positive constants $P_4, P_2, P_1,$ and P_5 defined as

$$P_1 = G_1 G_2 G_3 b \tau (A_2 + \mu B_2)$$

$$P_2 = G_1 G_2 (G_3 + a \tau) (A_3 + \mu B_3) + G_1 G_2 G_3 b \tau (A_2 + \mu B_2) - A_3 \Lambda b \tau (G_2 (1 - \varepsilon) \rho + \rho \varepsilon \sigma)$$

$$P_3 = G_1 G_2 G_3 b \tau (A_1 + \mu B_1) + G_1 G_2 (G_3 + a \tau) (A_2 + \mu B_2) - \Lambda (G_2 (1 - \varepsilon) \rho + \rho \varepsilon \sigma) (A_3 + b \tau)$$

$$P_4 = G_1 G_2 (G_3 + a \tau) (A_1 + \mu B_1) + G_3 b \tau \mu B_0 - (G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho) \Lambda (A_2 + b \tau)$$

$$P_5 = G_1 G_2 (G_3 + a \tau) \mu B_0 - (G_2 (1 - \varepsilon) \rho + \rho \varepsilon \sigma) \Lambda A_1$$

Now from the constant term, gives

$$G_1 G_2 (G_3 + a \tau) \mu B_0 - (G_2 (1 - \varepsilon) \rho + \rho \varepsilon \sigma) \Lambda A_1$$

Where,

$$B_0 = (G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho)$$

$$A_1 = \beta_1 \rho \varepsilon (G_3 + a \tau) + \beta_2 (G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho)$$

$$(G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho) \left\{ 1 - \frac{\Lambda A_1}{\mu G_1 G_2 (G_3 + a \tau)} \right\}$$

$$\Rightarrow (G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho) \{ 1 - R_0 \} \tag{16}$$

From equation (16), it is evident that $P_1 > 0$, Moreover, $P_5 > 0$ holds whenever $R_m < 0$. Therefore, the number of positive real solutions to equation (16) is determined by the signs of $P_2, P_3,$ and P_4 . This can be examined using Descartes' Rule of Signs applied to the quartic polynomial $f(I) = P_1 I^4 + P_2 I^3 + P_3 I^2 + P_4 I + P_5$. The different possible combinations of $f(I)$ signs and the corresponding number of positive roots are summarized in Table 1. The outcomes associated with each case are outlined in the table below.

Table 2: Number of positive real roots of $f(I)$ for $R_m > 1$ and $R_m < 1$

Case s	P_1, P_2, P_3, P_4, P_5	Sign change count	Basic Reproduction number	Number of non-negative roots
1	+	0	$R_m < 1$	0
2	+	1	$R_m > 1$	1
3	+	2	$R_m < 1$	0,2
4	+	1	$R_m > 1$	1

5	+	2	$R_m < 1$	0,2
6	+	1	$R_m > 1$	1
7	+	4	$R_m < 1$	0,2,4
8	+	3	$R_m > 1$	1,3
9	+	2	$R_m < 1$	0,2
10	+	3	$R_m > 1$	1,3
11	+	2	$R_m < 1$	0,2
12	+	1	$R_m > 1$	1
13	+	2	$R_m < 1$	0,2
14	+	3	$R_m > 1$	1,3
15	+	2	$R_m < 1$	0,2
16	+	3	$R_m > 1$	1,3

Theorem 4: Clearly, there is the possibility of multiple endemic equilibrium when $R_m > 1$ which indicates that a high potential for disease spread and persistence, making it a crucial threshold in infectious disease epidemiology. When we have multiple endemic equilibria, it simply means that a disease can coexist with the population.

Now, when $R_m > 1$, there is no unique endemic equilibrium. The disease is expected to die out eventually, and the population will reach a disease-free equilibrium. In this scenario, there is no stable endemic state when the disease can persist in the population. More so, there is no unique endemic equilibrium because the disease is not able to maintain itself in the population. P is positive when $R_m < 1$ and negative when $R_m > 1$. Also, P_5 is 0 if $R_m = 1$, then a unique non-zero solution exists. It can either die out or become endemic depending on the specific circumstances.

When R_m is greater 1, it means that a disease has the potential to spread and become endemic in a population. A single infected person can infect more than susceptible person on average. In fact, the disease can cause an outbreak or epidemic. The number of new cases will increase over time, at least initially. Finally, the disease will spread and persist in the population unless control measures are

implemented. In some cases, there can be multiple endemic equilibria, which refer to the possibility of multiple stable states where the disease persists in the population.

D. Local Asymptotic Stability of the Mastitis-free equilibrium

Theorem 5: If $R_m < 1$ then the mastitis-free equilibrium point is locally asymptotically stable otherwise, it is unstable.

The linearized Jacobian matrix at the disease-free equilibrium is

$$J_0 = \begin{bmatrix} -\mu & 0 & -\frac{\beta_1 \Lambda}{\mu} & -\frac{\beta_2 \Lambda}{\mu} & 0 & 0 \\ 0 & -G_1 & -\frac{\beta_1 \Lambda}{\mu} & -\frac{\beta_2 \Lambda}{\mu} & 0 & 0 \\ 0 & \varepsilon \rho & -G_2 & 0 & 0 & 0 \\ 0 & (1-\varepsilon)\rho & \sigma & -(G_3 + a\tau) & 0 & 0 \\ 0 & 0 & 0 & \theta & -G_4 & 0 \\ 0 & \gamma & a\tau & 0 & 0 & -\mu \end{bmatrix}$$

The characteristic equation obtained

$$(G_4 + \lambda)(\lambda + \mu)^2(\lambda^3 a_0 + \lambda^2 a_1 + \lambda a_2 + a_3) = 0 \quad (17)$$

Clearly,

$$\lambda_1 = -G_4, \lambda_2 = -\mu, \lambda_3 = -\mu$$

$$a_1 = a\tau + G_1 + G_2 + G_3$$

$$a_2 = [(a\tau + G_1 + G_3)G_2 + G_1(a\tau + G_3)]\mu - \Lambda\rho[\beta_1\varepsilon + \beta_2(1-\varepsilon)]$$

$$a_3 = \mu G_1 G_2 (a\tau + G_3) [1 - R_m] \quad (18)$$

According to Descartes' Rule of Signs, the number of positive real roots of a polynomial with real coefficients is equal to the number of sign changes in the sequence of its coefficients, or less than that number by an even integer.

Therefore, since $a_1 > 0$,

$$a_2 > 0 \text{ if and only if } [(a\tau + G_1 + G_3)G_2 + G_1(a\tau + G_3)]\mu > \Lambda\rho[\beta_1\varepsilon + \beta_2(1-\varepsilon)]$$

and $a_3 > 0$, then the Mastitis-free equilibrium is locally asymptotically stable.

E. Global Asymptotic Stability of the Mastitis free Equilibrium

In other to show the global stability of the Mastitis free equilibrium, the two conditions for the global stability of the Mastitis-free as contained in (Castilo-Chavez et al, 2002; Adeyemi et al, 2020) for $R_m < 1$ were established.

Now, we begin by re-writing the model (1) in the form

$$\frac{dX}{dt} = F(X, Y), \text{ and } \frac{dY}{dt} = G(X, Y), G(X, 0) = 0, \quad (19)$$

where $X = (S(t), R(t))$ and $Y = (E(t), A(t), I(t), C(t))$ are the non-infectious and infectious states of the system. $F(X, Y)$ and $G(X, Y)$ are the right-hand sides of X' and Y' respectively

Now, from the model (2), the reduced system $\frac{dX}{dt} = F(X, 0)$ is given as

$$\frac{dS(t)}{dt} = \Lambda - \mu S$$

$$\frac{dR(t)}{dt} + \mu R = 0 \quad (20)$$

$$\text{Let } X' = E_0 = (S(t), R(t)) = \left(\frac{\Lambda}{\mu}, 0\right)$$

be the equilibrium of the reduced system (19), then the result hold:

Theorem 6: The mastitis free equilibrium, E_0 of the model is globally asymptotically stable if $R_m \leq 1$, provided that the conditions H_1 and H_2 below are satisfied:

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

H_2 : $G(X, Y) = AY - \overset{\Delta}{G}(X, Y)$, $\overset{\Delta}{G}(X, Y) \geq 0$ for $(X, Y) \in \Gamma$,

Where $A = D_Y G(X^*, 0)$ is a Metzler matrix (that means all its off-diagonal entries are non-negative) and Ω denotes the domain in which the model remains biologically meaningful.

Proof:

First, it is demonstrated that X' is globally asymptotically stable by explicitly solving each equation in the reduced system (19) and then evaluating the limit as $t \rightarrow \infty$. To begin, we solve the second equation of system (1) with the initial condition $R(0) = R_0$ to obtain

$$R(t) = R_0 e^{-\mu t}$$

This simply means that the asymptotic dynamics of the system is independent of the initial conditions which implies that every solution in the system with initial conditions in Γ approaches $X^* = E_0$ as $t \rightarrow \infty$, $R(0) \rightarrow 0$ and is independent of R_m

Likewise, for the first equation in (20),

$$\frac{dS(t)}{dt} = \Lambda - \mu S(t)$$

$$\Rightarrow \frac{dS(t)}{dt} + \mu S(t) = \Lambda$$

Solving with integrating factor and subject to initial condition $S(0) = S_0$

$$S \leq \frac{\Lambda}{\mu} + C e^{-\mu t}$$

$$S_0 \leq \frac{\Lambda}{\mu} + C$$

$$C = S_0 - \frac{\Lambda}{\mu}$$

$$S_0 \leq \frac{\Lambda}{\mu} + \left(S_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t}$$

Therefore,

$$S(t) \leq \frac{\Lambda}{\mu} + \left(S_0 - \frac{\Lambda}{\mu}\right) e^{-\mu t}$$

As $t \rightarrow \infty, S(t) \rightarrow \frac{\Lambda}{\mu}$ (21)

This simply means that the asymptotic dynamics of the system is independent of the initial conditions which also implies that every solution of the model with initial conditions in Γ approaches $X^* = E_0$ as $t \rightarrow \infty$ and is independent of R_m .

Therefore, the equilibrium X^* is globally asymptotically stable for $\frac{dX}{dt} = F(X, 0)$.

Now, we are also to show the second condition H_2 :

$$H_2: G(X, Y) = AY - \hat{G}(X, Y), \hat{G}(X, Y) \geq 0 \text{ for } (X, Y) \in \Gamma$$

From model (2.1),

$$G(X, Y) = \begin{bmatrix} \left(\frac{\beta_1 A}{1+\alpha_1 A} + \frac{\beta_2 I}{1+\alpha_2 I}\right) S - G_1 E \\ \varepsilon \rho E - G_2 A \\ (1-\varepsilon)\rho E + \sigma A - \left(G_3 + \frac{\alpha \tau}{1+b\tau}\right) I \\ \theta I - G_4 C \end{bmatrix} \quad (22)$$

Where $G_1 = (\mu + \rho), G_2 = (\mu + \sigma + \gamma), G_3 = (\mu + \theta + \gamma), G_4 = (\mu + \delta + d)$ and

$$A = D_Y G(X', 0) = \begin{bmatrix} -G_1 & \beta_1 S^* & \beta_2 S^* & 0 \\ \varepsilon \rho & -G_2 & 0 & 0 \\ (1-\varepsilon)\rho & \sigma & -(G_3 + \alpha \tau) & 0 \\ 0 & 0 & \theta & -G_4 \end{bmatrix} \quad (23)$$

$$AY = \begin{bmatrix} -G_1 & \frac{\beta_1 S^*}{\mu} & \frac{\beta_2 S^*}{\mu} & 0 \\ \varepsilon \rho & -G_2 & 0 & 0 \\ (1-\varepsilon)\rho & \sigma & -(G_3 + \alpha \tau) & 0 \\ 0 & 0 & \theta & -G_4 \end{bmatrix} \begin{bmatrix} E(t) \\ A(t) \\ I(t) \\ C(t) \end{bmatrix} = \begin{bmatrix} -G_1 E(t) + \beta_1 S^* A(t) + \beta_2 S^* I(t) \\ \varepsilon \rho E(t) - G_2 A(t) \\ (1-\varepsilon)\rho E(t) + \sigma A(t) - (G_3 + \alpha \tau) I(t) \\ \theta I - G_4 C(t) \end{bmatrix}$$

Then, $G(X, Y) = AY - \hat{G}(X, Y)$ implies $\hat{G}(X, Y) = AY - G(X, Y)$, so that

$$\hat{G}(X, Y) = \begin{bmatrix} -G_1 E(t) + \beta_1 S^* A(t) + \beta_2 S^* I(t) \\ \varepsilon \rho E(t) - G_2 A(t) \\ (1-\varepsilon)\rho E(t) + \sigma A(t) - (G_3 + \alpha \tau) I(t) \\ \theta I - G_4 C(t) \end{bmatrix} - \begin{bmatrix} \left(\frac{\beta_1 A}{1+\alpha_1 A} + \frac{\beta_2 I}{1+\alpha_2 I}\right) S - G_1 E \\ \varepsilon \rho E - G_2 A \\ (1-\varepsilon)\rho E + \sigma A - \left(G_3 + \frac{\alpha \tau}{1+b\tau}\right) I \\ \theta I - G_4 C \end{bmatrix}$$

$$\hat{G}(X, Y) = \begin{bmatrix} \frac{\beta_1 \Lambda}{\mu} A(t) + \frac{\beta_2 \Lambda}{\mu} I(t) - \left(\frac{\beta_1 A}{1+\alpha_1 A} + \frac{\beta_2 I}{1+\alpha_2 I}\right) S \\ 0 \\ \frac{\alpha \tau^2 b I^2}{(1+b\tau)} \\ 0 \end{bmatrix} = \begin{bmatrix} \hat{G}_1(X, Y) \\ \hat{G}_2(X, Y) \\ \hat{G}_3(X, Y) \\ \hat{G}_4(X, Y) \end{bmatrix} \quad (24)$$

Clearly, it is obvious from equation (24) that $\hat{G}_2(X, Y) = 0, \hat{G}_3(X, Y) \neq 0, \hat{G}_4(X, Y) = 0, \hat{G}_1(X, Y) \neq 0$.

Therefore, since $\hat{G}_1(X, Y) \neq 0$ and $\hat{G}_3(X, Y) \neq 0$, and so the mastitis-free equilibrium, E_0 is not globally asymptotically stable.

F. Bifurcation Analysis

A bifurcation change in a qualitative change is the nature of the solution trajectories due to a parameter change. The point at which this change takes place is called bifurcation point (Otieno *et al.*, 2012). At the bifurcation points, a number of equilibrium points, or their stability properties or both changes. In other words, it is the change that occurs when the system bifurcates from one state to another. Whenever the basic reproduction is less than unity, the disease will die out meaning that the infectious disease will not invade the total population unless otherwise. Hence, the epidemiological consequence of this is that the classical requirement of the reproduction number of the model being less than unity becomes only a necessary, but not sufficient condition, for disease elimination or eradication. The center Manifold theorem as used in (Castillo-Chavez and Song, 2004; Otieno *et al.*, 2012) is employed here to establish the local stability of endemic equilibrium.

Theorem 7: If $R_m \geq 1$, then the Mastitis-endemic equilibrium is locally asymptotically stable, otherwise it is stable

Proof: The center Manifold theory approach, as described by (Castillo-Chavez and Song, 2004) is employed to establish the local asymptotic stability of the mastitis-endemic equilibrium around $R_m = 1$, and to determine if the model exhibit a backward or forward bifurcation at $R_m = 1$,

Now let $S(t) = x_1, E(t) = x_2, A(t) = x_3, I(t) = x_4, C(t) = x_5, R(t) = x_6$ so that $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$ so that the model in terms of these new state variables is re-written in the form:

$$\left. \begin{aligned} \frac{dx_1}{dt} = f_1 &= \Lambda - \left(\frac{\beta_1 x_3}{1 + \alpha_1 x_3} + \frac{\beta_2 x_4}{1 + \alpha_2 x_4} \right) x_1 - \mu x_1 \\ \frac{dx_2}{dt} = f_2 &= \left(\frac{\beta_1 x_3}{1 + \alpha_1 x_3} + \frac{\beta_2 x_3}{1 + \alpha_2 x_3} \right) x_1 - (\mu + \rho) x_2 \\ \frac{dx_3}{dt} = f_3 &= \varepsilon \rho x_2 - (\mu + \sigma + \gamma) x_3 \\ \frac{dx_4}{dt} = f_4 &= (1 - \varepsilon) \rho x_2 + \sigma x_3 - \left(\mu + \theta + \gamma + \frac{\alpha \tau}{1 + b \tau I} \right) x_4 \\ \frac{dx_5}{dt} = f_5 &= \theta x_4 - (\mu + \delta + d) x_5 \\ \frac{dx_6}{dt} = f_6 &= \gamma x_2 + \left(\gamma + \frac{\alpha \tau}{1 + b \tau x_4} \right) x_4 - \mu x_6 \end{aligned} \right\} \quad (25)$$

$$J(E_0) = J_{\beta_1^*} = \begin{bmatrix} -\mu & 0 & -\frac{\beta_1 \Lambda}{\mu} & -\frac{\beta_2 \Lambda}{\mu} & 0 & 0 \\ 0 & -G_1 & -\frac{\beta_1 \Lambda}{\mu} & -\frac{\beta_2 \Lambda}{\mu} & 0 & 0 \\ 0 & \varepsilon \rho & -G_2 & 0 & 0 & 0 \\ 0 & (1 - \varepsilon) \rho & \sigma & -(G_3 + \alpha \tau) & 0 & 0 \\ 0 & 0 & 0 & \theta & -G_4 & 0 \\ 0 & \gamma & \alpha \tau & 0 & 0 & -\mu \end{bmatrix}$$

A right eigenvector (w) of $J_{\beta_1^*}$ associated with the zero eigenvalue is given by applying the Manifold theory, letting $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ be the right eigenvector associated or corresponding to the zero eigen value $\lambda = 0$, where $w_2 = w_2 > 0$ a free eigenvector; and

$$\begin{aligned} w_1 &= - \left\{ \frac{\mu(\beta_1 \Lambda \varepsilon \rho)(G_3 + \alpha \tau) + \beta_2 + \beta_2 \Lambda G_2(1 - \varepsilon) \rho + \sigma \varepsilon \rho}{\mu G_2(G_3 + \alpha \tau)} \right\} w_2, \\ w_2 &= w_2 > 0, \\ w_3 &= \frac{\varepsilon \rho}{G_2} w_2, \\ w_4 &= \frac{[G_2(1 - \varepsilon) + \sigma \varepsilon] \rho}{G_2(G_3 + \alpha \tau)} w_2, \\ w_5 &= \frac{\theta [G_2(1 - \varepsilon) + \sigma \varepsilon] \rho w_2}{G_4 G_2(G_3 + \alpha \tau)}, \\ w_6 &= \frac{(\gamma G_2 + \varepsilon \rho \alpha \tau)}{\mu G_2} w_2 \end{aligned}$$

Similarly, the left eigenvector associated with the zero eigenvalues is given by $v = (v_1, v_2, v_3, v_4, v_5, v_6)^T$, were $v_1 = 0$, $v_2 = v_2 > 0$

$$\begin{aligned} v_3 &= \frac{\beta_2 \Lambda}{(G_3 + \alpha \tau)} v_2, \\ v_4 &= \frac{(G_2 \beta_2 - \beta_1) \Lambda}{\sigma \mu (G_3 + \alpha \tau)} v_2, \end{aligned}$$

Suppose that we choose β_1 as our bifurcation parameter, then from equation (14), and $\beta_1 = \beta_1^*$ where β_1^* is the chosen bifurcation parameter and the case when $R_m = 1$,

Then,

$$\frac{\Lambda \rho (\alpha \varepsilon \tau \beta_1 + \varepsilon \sigma \beta_2 - \varepsilon G_2 \beta_2 + \varepsilon G_3 \beta_1 + G_2 \beta_2)}{\mu G_1 G_2 (\alpha \tau + G_3)} = 1$$

$$(\Lambda \rho \alpha \varepsilon \tau + \Lambda \rho \varepsilon G_3) \beta_1^* + \Lambda \rho \varepsilon \sigma \beta_2 - \Lambda \rho \varepsilon G_2 \beta_2 + \Lambda \rho G_2 \beta_2 = \mu G_1 G_2 (\alpha \tau + G_3)$$

$$\beta_1^* = \frac{\mu G_1 G_2 (\alpha \tau + G_2) - \beta_2 (G_2 (1 - \varepsilon) \rho + \sigma \varepsilon \rho) \Lambda}{\Lambda \rho \varepsilon (\alpha \tau + G_3)} \quad (26)$$

The Jacobian matrix of the model at the mastitis-free equilibrium, E_0 , when $\beta_1 = \beta_1^*$ such that $R_m = 1$, is obtained

$$\begin{aligned} v_5 &= 0, \\ v_6 &= 0 \end{aligned}$$

$v_2 = v_2 > 0$ is free eigenvector

We now reproduce the theorem stated by the approach of Castillo-Chavez and Song, (2004).

Computation of a and h

The coefficients a and h as defined by Castillo-Chavez and Song, (2004) are:

$$a = \sum_{i,j,k=1}^{\infty} v_k w_i w_j \frac{\partial^2 f_k(E_0, \beta^*)}{\partial x_i \partial x_j} \text{ and}$$

$h = \sum_{k,i,j=1}^{\infty} v_k w_i \frac{\partial^2 f_k(E_0, \beta^*)}{\partial x_i \partial \beta^*}$; and are algebraically computed as follows, considering only the nonzero components f_i : $k = 2, 3, 4, 5$. With these definitions, the following were obtained:

$$\begin{aligned} a &= v_2 w_1 w_3 \beta_1^* + v_2 w_1 w_4 \beta_2 + v_2 w_3 w_3 \left(-2 \beta_1^* \alpha_1 \frac{\Lambda}{\mu} \right) \\ &\quad + v_2 w_4 w_4 \left(-2 \beta_2 \alpha_2 \frac{\Lambda}{\mu} \right) \\ &\quad + v_4 w_4 w_4 (2ab\tau^2) \end{aligned} \quad (27)$$

w_1 is non positive, while $v_2, v_4, w_3, w_4 > 0$

$$\begin{aligned} \Rightarrow a &= v_4 w_4 w_4 (2ab\tau^2) \\ &\quad - v_2 \left(w_1 w_3 \beta_1^* + w_1 w_4 \beta_2 \right. \\ &\quad \left. + w_3 w_3 \left(2 \beta_1^* \alpha_1 \frac{\Lambda}{\mu} \right) + w_4 w_4 \left(2 \beta_2 \alpha_2 \frac{\Lambda}{\mu} \right) \right) \end{aligned}$$

On substituting of necessary quantities and simplifying, clearly $a > 0$ if

$$v_4 w_4 w_4 (2ab\tau^2) > v_2 \left(w_1 w_3 \beta_1^* + w_1 w_4 \beta_2 + 2 \frac{\Lambda}{\mu} ((w_3)^2 + (w_4)^2) (\beta_1^* \alpha_1 + \beta_2 \alpha_2) \right) \quad (28)$$

Similarly, the coefficient h is obtained as:

$$h = v_2 w_1 \frac{x_3}{1 + \alpha_1 x_3} + v_2 w_2 \left(\frac{1}{(1 + \alpha_1 x_3)} - \frac{\alpha_1 x_3}{(1 + \alpha_1 x_3)^2} \right) x_1$$

but at $E_0 = \left(\frac{A}{\mu}, 0, 0, 0, 0\right)$

$$h = v_2 w_2 x_1 h$$

$$h = v_2 w_2 \left(\frac{A}{\mu}\right)$$

$$\Rightarrow h = \frac{v_2 w_2 \pi}{\mu} > 0, \tag{29}$$

since v_2, w_2 are non-negative, clearly, from (28) and (29),

$$a > 0, \text{ if } v_4 w_4 w_4 (2ab\tau^2) > v_2 \left(w_1 w_3 \beta_1^* + w_1 w_4 \beta_2 + 2 \frac{A}{\mu} ((w_3)^2 + (w_4)^2) (\beta_1^* \alpha_1 + \beta_2 \alpha_2) \right), \text{ and } b > 0.$$

The presence of saturation in the model plays a central role in shaping the bifurcation dynamics of the system. Saturation effects arising from limited treatment capacity, treatment delays, or nonlinear transmission mechanisms alter the coefficient that determines the local bifurcation behavior. When these saturation effects are substantial, the coefficient becomes positive, leading to a backward bifurcation in which a stable endemic equilibrium can persist even when $R_m < 1$. In contrast, when saturation is absent, particularly in the absence of treatment delay, the coefficient becomes negative and the system exhibits a forward bifurcation consistent with classical epidemiological theory. Under this condition, the infection dies out when $R_m < 1$ and persists only when $R_m > 1$, with no bi-stability. Hence, saturation significantly influences the qualitative behavior of the model and can make disease elimination more challenging. The result is stated in the theorem below.

Theorem 3. For the Mastitis model (1), a backward bifurcation arises when the coefficient $a > 0$. Conversely, a forward bifurcation occurs when $b = 0$ and $a < 0$, provided that the basic reproduction number R_m crosses unity.

This result shows that, in the absence of treatment delay b , a locally asymptotically stable endemic equilibrium E^{**} exists for $R_m > 1$. Consequently, the introduction of even a small number of infectious individuals into a susceptible population can lead to the sustained presence of mastitis whenever $R_m > 1$. This also implies that reducing R_m below unity may not guarantee disease eradication and that additional control measures may be required. In settings where backward bifurcation occurs, elimination depends not only on reducing R_m but also on the initial number of infected individuals. If the initial infection level exceeds a certain threshold, the system will converge to the endemic equilibrium rather than the disease-free state, underscoring the need for early and aggressive intervention strategies.

This however has important implications for disease control and eradication strategies, as it suggests that simply reducing the reproduction number below 1 may not be enough to eradicate the disease, if the initial number of infected individuals is above the threshold.

G. Global stability of endemic equilibrium

Theorem 8: If $R_m > 1$ then the mastitis-endemic equilibrium point of the model equation is globally asymptotically stable in Ω , provided $S \geq S^*, E \geq E^*, A \geq A^*, I \geq I^*, C \geq C^*$, and $R \geq R^*$.

Proof: To establish the global stability of the endemic equilibrium E^* , we analysed by constructing the following quadratic Lyapunov function L , following the approach of (Odebiyi *et al*, 2024), such that

$$L = \frac{1}{2} [(S - S^*) + (E - E^*) + (A - A^*) + (I - I^*) + (C - C^*) + (R - R^*)]^2$$

By direct calculation of the time derivatives $L(t)$ along the solutions of the system (1) is obtained as

$$\begin{aligned} \frac{dL}{dt} = & [(S - S^*) + (E - E^*) + (A - A^*) + (I - I^*) \\ & + (C - C^*) + (R - R^*)] \left(\frac{dS}{dt} + \frac{dE}{dt} + \frac{dA}{dt} \right. \\ & \left. + \frac{dI}{dt} + \frac{dC}{dt} + \frac{dR}{dt} \right) \end{aligned}$$

Substituting the appropriate solutions of the system (1) into the derivative of $L(t)$ gives

$$\frac{dL}{dt} \leq [(S - S^*) + (E - E^*) + (A - A^*) + (I - I^*) + (C - C^*) + (R - R^*)] \frac{dN}{dt} \tag{30}$$

Substituting $\frac{dN}{dt} \leq \Lambda - \mu N$ in equation (30) yields

$$\frac{dL}{dt} \leq [(S - S^*) + (E - E^*) + (A - A^*) + (I - I^*) + (C - C^*) + (R - R^*)] (\Lambda - \mu N) \tag{31}$$

Equation (31) is simplified to give

$$\begin{aligned} \frac{dL}{dt} \leq & [(S + E + A + I + C + R) \\ & - (S^* + E^* + A^* + I^* + C^* + R^*)] (\Lambda - \mu N) \end{aligned}$$

This becomes,

$$\leq \left(N - \frac{\Lambda}{\mu}\right) (\Lambda - \mu N) \tag{32}$$

After some algebraic Simplification we have

$$\begin{aligned} & \leq -\frac{1}{\mu} (\Lambda - \mu N) (\Lambda - \mu N) \\ & \leq -\frac{1}{\mu} (\Lambda - \mu N)^2 \end{aligned} \tag{33}$$

Let $\xi = \Lambda - \mu N$, then from (33), we have $\frac{dL}{dt} \leq -\frac{1}{\mu} \xi^2$.

Hence, $\left(\frac{dL}{dt}\right) (S, E, A, I, C, R) \leq 0$ and $\frac{dL}{dt} = 0$, if and only if $S = S^*, E = E^*, A = A^*, I = I^*, C = C^*, A = A^*$. Therefore, If $X < Y$, then, $\frac{dL}{dt}$ will be negative definite, implying that $\frac{dL}{dt} = 0$, if and only if $S = S^*, E = E^*, A = A^*, I = I^*, C = C^*$, any $R = R^*$. Therefore, the largest positive invariant set in $\{(S^*, E^*, A^*, I^*, C^*, R^*) \in \Omega: \frac{dL}{dt} = 0\}$ is a singleton $\{E_1\}$, where E_1 is globally asymptotically stable in the set Ω in accordance to (LaSalle, 1976), it then implies that E_1 is globally asymptotically stable in Ω .

H. Sensitivity Analysis of the Reproduction Number

Theoretical methods are sufficiently advanced so that it is intellectually dishonest to perform modelling without sensitivity analysis. Following (Chitnis et al, 2008), we used the normalized forward sensitivity index also called elasticity as it is the backbone of nearly all Sensitivity analysis techniques and are computationally efficient. Sensitivity analysis can as well help the reviewer to determine which parameters are the key drivers of a model’s results.

Furthermore, as the basic reproduction number R_m is a measure of the potential for the disease spread in a population, we computed the sensitivity index of the basic reproduction number, R_m with respect to a parameter value, P given by: $S_p^{R_m} = \frac{\partial R_m}{\partial P} * \frac{P}{R_m}$ We therefore evaluate the sensitivity indices at the baseline parameter values given.

In determining how best to reduce human mortality and morbidity due to AIDS, the sensitivity indices of the effective reproduction number R_m to the parameters in the model was calculated following the approach of (Chitnis et al, 2008; Odebiyi and Oladejo 2024). These indices tell us how crucial each parameter is to disease transmission and prevalence and discover parameters that have a high impact on R_m and should be targeted by intervention strategies. More so, sensitivity analysis is commonly used to determine the robustness of model predictions to the parameter values (since there are usually errors in data collection and presumed Description of Sensitivity Analysis parameter values (Chitnis et al, 2008) and more so, to discover parameters that have a high impact in R_m .

Sensitivity analysis allows us to measure the relative change in a state variable when a parameter changes. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives.

Now, it is already shown that the explicit expression of R_m is given by $R_m = \frac{\Lambda\rho(a\varepsilon\tau\beta_1 + \varepsilon\sigma\beta_2 - \varepsilon G_2\beta_2 + \varepsilon G_3\beta_1 + G_2\beta_2)}{\mu G_1 G_2 (\alpha\tau + G_3)}$

The normalized forward sensitivity indices of R_m with respect to its parameters are given in Table 3 below.

It can read from the index Table 3 that the most sensitive parameter is Per capita recruitment rate, Λ . The sensitivity index of Per capita recruitment, $S_\Lambda^{R_m} = 1.0000$ rate implies that increasing or decreasing it by 10% increases or decreases the mastitis reproduction number by 10%. The sensitivity index for the transmission rates for clinical infectious cattle, β_2 is $S_{\beta_2}^{R_m} = 0.9236050116$ meaning that increasing or decreasing it by 10% decreases or increases the mastitis reproduction number R_m by 9.236050%. Also, the sensitivity index for the maximum rate of treatment, τ is $S_\tau^{R_m} = 0.8320370929$ this implies that increasing or decreasing the maximum rate of treatment by 10% increases or decreases R_m by 8.320370929%. The result suggests that

optimizing per capita recruitment rate, Λ reducing transmission rates from clinical, β_2 and sub-clinical infectious cattle, β_1 and maximising treatment rates, τ can be effective intervention to control mastitis.

Table 3: Sensitivity Indices of R_m in terms of the Model’s parameter baseline values

Variables	Baseline Value	Sensitivity Index
	$\frac{1}{5 \times 365}$	
Λ	$\frac{1}{5 \times 365}$	1.00000
β_1	0.002300	0.07639498901
β_2	0.002315	0.9236050116
a	1.005	-0.8320370928
ε	0.333	0.005561511740
ρ	$\frac{1}{6 \times 7}$	0.02583486012
σ	1.005	-0.07059332307
θ	0.010	0.03584159788
μ	$\frac{1}{5 \times 365}$	0.02583486012
γ	0.015	0.02583486012
τ	$\frac{1}{28}$	-0.8320370929

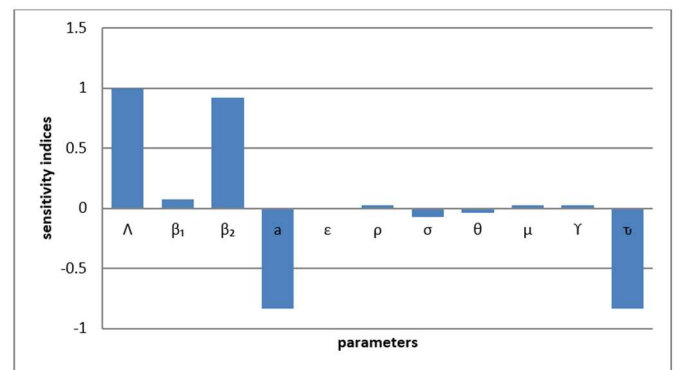


Figure 3: Graphical Representation of the Sensitivity Indices of R_m

Implementing strategies such as improved breeding programs, reduced calf mortality, improved hygiene, segregation, and treatment protocols and farmer education can also contribute to reducing the disease burden

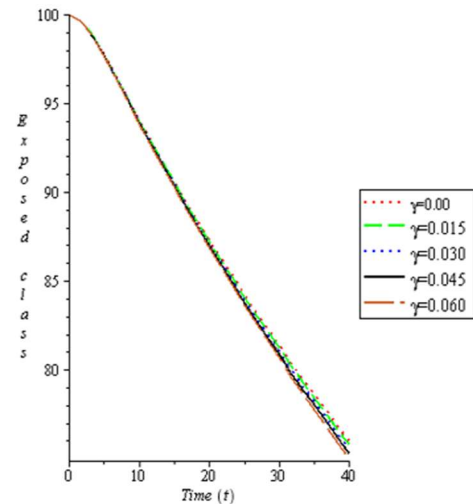
IV. RESULTS AND DISCUSSION

A. Numerical simulations

Simulation of the model (1) was performed for better understanding of dynamical spread of transmission of the mastitis model using Maple 18.0 mathematical software. The results of the model equations are presented below in form of graphs and the results are discussed to illustrate the changes in the compartments. The effects of some of the epidemiological parameters on the susceptible population, exposed infective, subclinical infective, clinical infective, chronic and recovered populations were checked in order to observe their impact on the numerical spread of the disease using a set of reasonable parameters.

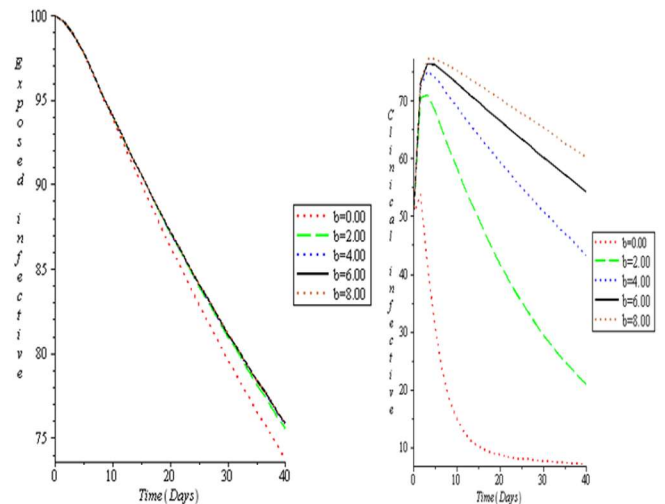
The results in Figure 1 (a)-(c) show that an increase in the rate of spontaneous recovery (γ) has a positive impact on the cattle population. Specifically, the recovered cattle population increases significantly, indicating that more cattle are recovering from the disease. In contrast, the chronic class decreases, suggesting that fewer cattle are progressing to the chronic stage of the disease. Additionally, the exposed cattle population decreases, indicating that fewer cattle are being exposed to the disease. These trends suggest that increasing the rate of spontaneous recovery can help control the spread of the disease and improve the overall health of the cattle population. These findings highlight the importance of promoting spontaneous recovery in controlling the disease.

Figure 2 (a)-(c) shows the effects of the rate of delay in treatment b on cattle populations. It was observed that an increase in the rate of delay in treatment increases the exposed cattle population, clinical infective population, and chronic cattle population. These findings highlight the importance of prompt treatment in controlling disease spread and preventing progression to a chronic state. Delayed



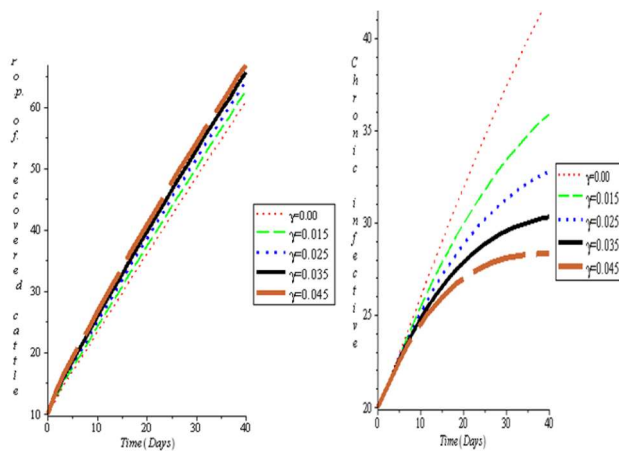
(c)

Figure 1a-c: Plots of the effects of spontaneous recovery rate on the populations.



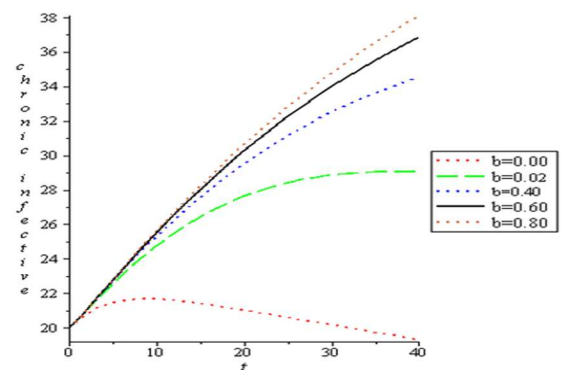
(a)

(b)



(a)

(b)



(c)

Figure 2a-c: Plots of the effects of the rate of delay in treatment (b), on the cattle populations.

treatment allows infected cattle to interact with susceptible cattle, increasing exposed cattle and leading to a longer infectiousness duration, resulting in more clinically infected cattle. Inadequate or delayed treatment can cause disease

progression to a chronic state, increasing the chronic population.

In Figure 3, the effects of sub-clinical infectious cattle transmission rate, β_1 were investigated. An increase in transmission rate through contact with infectious cattle decreased the susceptible cattle population (Figure 3(a)). In contrast, it increased the subclinical infective, clinical infective, and chronic infective cattle populations (Figures 3(b)-3(d)). The implication is that if the cattle transmission rate increases, it will lead to a decline in susceptible cattle population and an increase in infected

cattle populations, potentially resulting in increased disease spread, higher morbidity and mortality rates, and economic losses.

Figure 4(a)-(b) shows the effect of maximum treatment, τ on chronic infective and recovered cattle. As treatment rate increases, chronic infective cattle decrease (Figure 4(a)), and recovered cattle increase (Figure 4(b)). Increased treatment manages the disease, reducing chronic cases and enabling more cattle to recover.

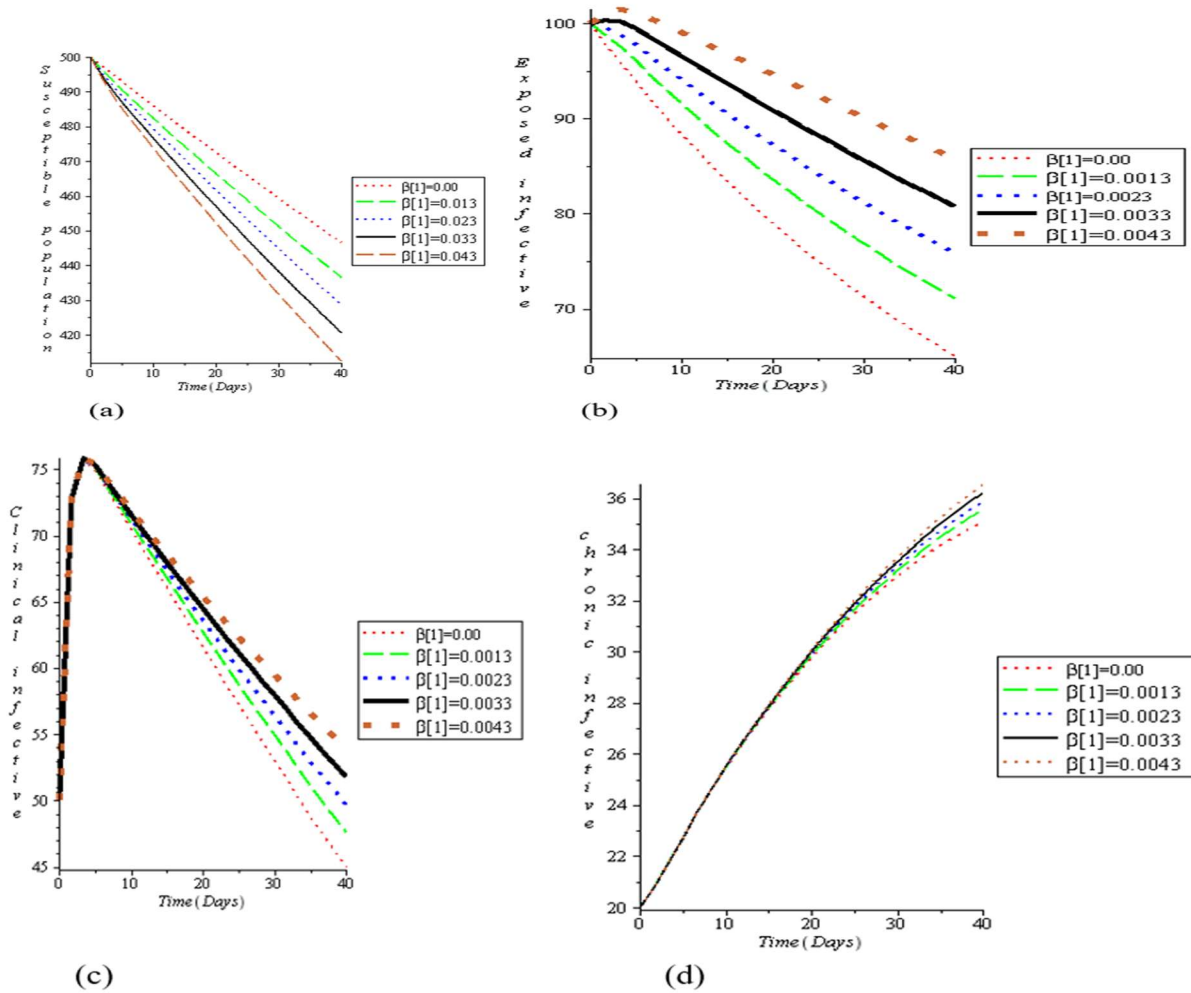


Figure 3a-d: Plots of the effects of cattle transmission rate, β_1 on the population.

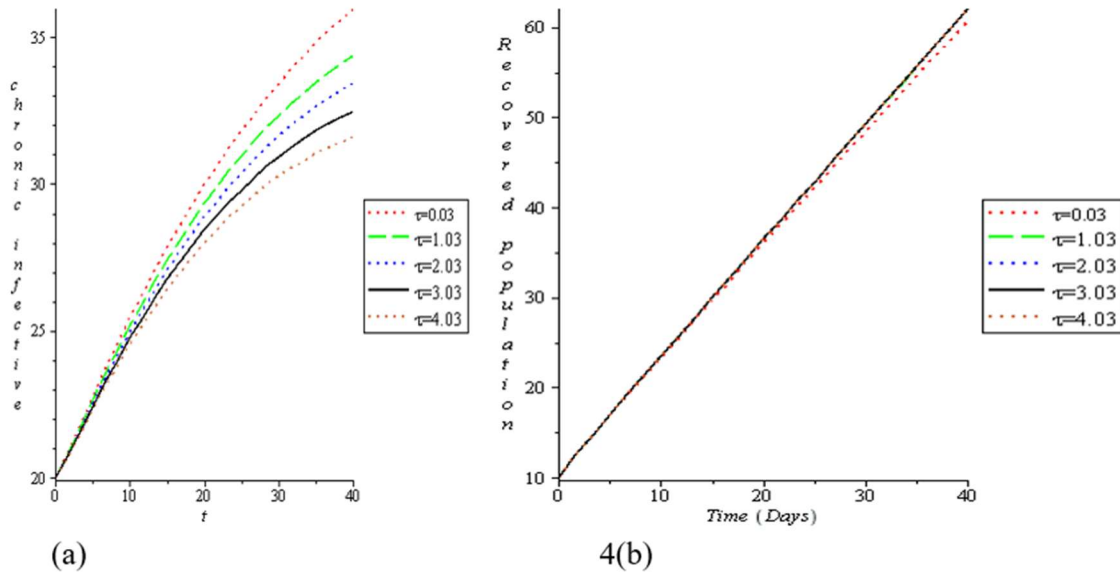


Figure 4a-b: Plots of the effect of maximum treatment ‘ τ ’ on the chronic infective cattle population and Recovered population.

Figure 5 shows the effect of culling rate d on chronic population. Culling improves herd health and prevents disease spread. Increased culling rate reduces chronic infected cattle, removing infectious animals and preventing transmission. This is necessary as chronically infected cattle can harbor mastitis-causing bacteria, risking other cattle.

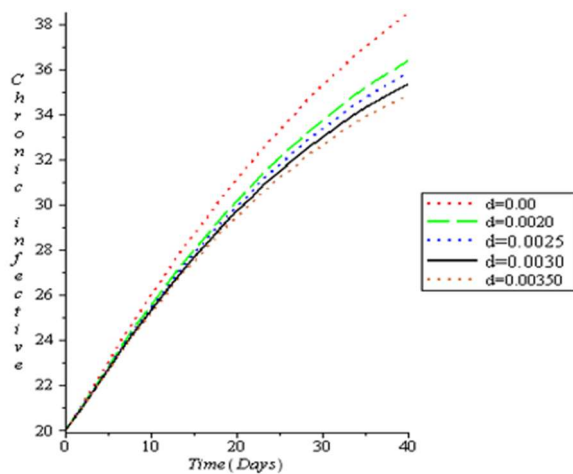


Figure 5: Plot of the effect of rate of culling ‘ d ’ on the dynamics of mastitis disease.

V. CONCLUSION

This study provides comprehensive epidemiological insight into the transmission dynamics of bovine mastitis through the formulation and analysis of a nonlinear six-compartment mathematical model incorporating nonlinear incidence and treatment functions. The model was shown to be epidemiologically well-posed by establishing the positivity of solutions and the existence of an invariant region. The basic reproduction number, R_m , was derived

using the next-generation matrix approach. Stability analyses of the mastitis-free and endemic equilibria were conducted, and all possible root configurations for the associated characteristic equations, $f(I^*)$ were identified and summarized in Table 3. Sensitivity analysis demonstrated that the per capita recruitment rate Λ is the most influential parameter affecting R_m .

Assessment of the global stability of the mastitis-free equilibrium E_0 revealed that it is not globally asymptotically stable. Bifurcation analysis using the center manifold theorem further showed that the model exhibits a backward bifurcation at $R_m = 1$, indicating the existence of bistability in which both a stable mastitis-free equilibrium and a stable endemic equilibrium coexist when $R_m < 1$. This implies that successful eradication of mastitis depends not only on reducing R_m below unity but also on effectively controlling the initial infection burden within the herd. Numerical simulations illustrated the influence of key epidemiological parameters on R_m , highlighting that control strategies should prioritize factors such as the per capita recruitment rate, Λ ; transmission rates for clinical and subclinical infectious cattle, β_2 and β_1 the maximum treatment rate, τ . Parameters with negative sensitivity indices are equally important, as increases in these parameters decrease R_m , thereby promoting a mastitis-free environment.

Bovine mastitis remains one of the most economically and epidemiologically significant diseases in dairy production systems. Its impact extends beyond individual animal health, altering herd structure by increasing morbidity, reducing milk productivity, and influencing culling decisions. The coexistence of clinical and subclinical infections creates hidden reservoirs that sustain ongoing pathogen transmission. Herd demographic processes including recruitment, replacement, and lactation cycles interact with transmission pathways to maintain endemicity. Moreover, milking routines, environmental contamination, and management practices modify within-herd contact

patterns, thereby increasing opportunities for spread. As demonstrated by the model, nonlinear infection processes and treatment responses can lead to complex disease behaviours such as backward bifurcation, reinforcing the necessity of early detection, optimal treatment, and robust management practices. Mastitis therefore holds substantial epidemiological significance because it can persist endemically, reduce productivity, and sustain hidden transmission even under moderate control efforts.

Based on these findings, several recommendations are essential for effective mastitis management. Early detection and routine surveillance should be strengthened, particularly through regular somatic cell count monitoring and bacteriological testing to identify subclinical infections. Interventions should target high-impact epidemiological parameters, especially those strongly influencing R_m . Treatment strategies must be optimized through rapid case identification, appropriate dosage, and adherence to withdrawal periods. Milking hygiene and environmental conditions should be rigorously maintained through proper equipment sanitation, teat-dipping protocols, and effective bedding and moisture management. Because backward bifurcation indicates disease persistence even when $R_m < 1$, efforts to reduce initial infection burden such as segregation, dry-cow therapy, and strategic culling of chronic cases are critical. Recruitment and replacement practices should be optimized to avoid introducing susceptible animals into high-risk settings. Farm management protocols, including training, biosecurity, and systematic record-keeping, should be strengthened to guide informed decision-making. Finally, model outputs such as sensitivity indices and simulation results should be integrated into control planning to ensure that interventions effectively lower R_m and reduce long-term transmission.

While this research provides important insights into mastitis dynamics, it has limitations. Although sensitivity indices were analysed, parameter uncertainty was not incorporated, which may influence the accuracy of the estimated reproduction number and the projected effectiveness of interventions. The deterministic nature of the model does not capture stochastic variations inherent in mastitis transmission within herds. Future work should consider incorporating stochastic effects to more accurately represent random fluctuations in disease spread. Additionally, integrating regression-based or data-driven approaches using real-world herd data could improve parameter estimation and predictive performance. Including control variables in extended models may further clarify how modifying specific management practices affects disease spread and overall herd health.

AUTHOR CONTRIBUTIONS

O. A. Odebiyi: Methodology, Writing – original draft, Software and Validation. **T. O. Oluyo:** Supervision. **M.O Adeyemi:** Conceptualization. **J. K. Oladejo:** Writing – original draft and Writing – review & editing. **O. Olaoye:**

Software. **E. O. Elijah:** Writing – review & editing **W.O. Ayanrinola:** Writing – review & editing.

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