Dynamics of Lumpy Skin Disease Model with Vaccination and Environmental Transmission

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ABSTRACT

Lumpy skin disease (LSD) is a cattle disease that can spread rapidly and is caused by the lumpy skin disease virus (LSDV). LSDV can spread through direct contact, insect vectors, and contaminated environments. In this study, we aim to analyze the dynamics of a lumpy skin disease model that contains seven compartments: susceptible cattle, vaccinated cattle, infected cattle, recovered cattle, susceptible vector, infected vector, and LSDV in the environment. The model's mathematical properties, including non-negativity and boundedness of the solution, are examined. Equilibrium points of the system are determined, along with their local and global stability under specific conditions. The results of research are the identification of two equilibrium points and the most influential parameter of the system. The disease-free equilibrium point is locally and globally asymptotically stable when the basic reproduction number is less than one, and the endemic equilibrium point is locally asymptotically stable under the Lienard-Chipart criteria. Sensitivity analysis reveals that the vaccination rate and the contact rate between susceptible cattle with LSDV are the most influential parameter. The proposed LSD model provides valuable insights into the dynamics of LSD and highlights the importance of considering vaccination and environmental transmission.

Keywords: LSD epidemic model; equilibrium point; stability; sensitivity analysis

INTRODUCTION

Lumpy skin disease (LSD) is a cattle disease that can spread in various transmissions and cause economic losses. LSD is caused by the Capripoxvirus from the Poxviridae family [1],[2],[3]. LSD is not classified as a zoonotic disease, a disease that can naturally be transmitted from animals to humans or conversely [3],[4],[5]. LSD can result in significant economic losses for cattle farmers such as weight loss, reduction in milk production, abortion, infertility and death in cattle [6],[7]. The first case of LSD was found in 1957 at East Africa. Although LSD can be found throughout Middle East and Africa [3], but it can easily spread to Indonesia. The first case of LSD in Indonesia was found in 2022 at Riau and increased every year [8]. According to the report from the Ministry of Agriculture of the Republic of Indonesia in 2023, the number of LSD are recorded 64.000 cases across 16 provinces, the highest number of cases is 23.000 cases in Central Java. The increasing

number of LSD cases become a serious issue that needs to be addressed immediately.

The primary transmission of LSDV is mechanically through arthropod vectors such as mosquitoes (Aedes aegypti), flies (Stomoxys calcitrans), small insects (Culicoides nubeculosus), and ticks (Rhipicephalus appendiculatus). LSDV can spread rapidly during the rainy season because of the abundance of arthropods during this time. Additionally, LSD can spread through contact between cattle and contaminated environments by LSDV [9],[10],[11]. The healthy cattle can develop antibodies if they get vaccination and the recovered cattle from LSD infection have natural antibodies. The control policy for LSD in Indonesia includes emergency vaccination and facility disinfection. Emergency vaccination and facility disinfection is important strategy to reduce the impact of a new infection, protect healthy cattle, and prevent widespread of the LSD outbreaks [12],[13],[14].

In disease epidemiology, mathematical modelling has a great role to study and give the recommendations for disease control. Several researchers have proposed their LSD model with various transmission and intervention strategies. The authors in [15] propose an SEIR model for the LSD and provide vaccination as a control strategy, but they only focus on transmission between cattle population. The other transmission of LSD model is constructed by author in [16], they consider the transmission between cattle and vectors population, they also prove that vaccination for cattle is effectively to reduce the spread of LSD. The most complex transmission of LSD model show in [17], they propose their model by considering transmission through between cattle, vectors, and pathogen (LSDV) population. This model shows reliable transmission, but it does not include an intervention strategy.

In this study, we develop an LSD model which includes disease transmission due to contact between cattle, vector and LSDV population. We take into account the cattle vaccination to prevent the LSD spread and to complete the previous research. As we see in the following section, the proposed model is basically a combination of model in [16], [17].

METHODS

In this study, we develop the LSD model with vaccination for susceptible cattle and consider the environmental transmission. In order to analyze our model, we perform the following steps. First, we construct the proposed LSD model. This step involves formulating a mathematical model to describe the transmission of LSD using various assumption. Second, we prove the non-negativity and boundedness solution. In this stage, the solution of model is analyzed by contrary assumption for non-negativity and comparison lemma for boundedness. Third, we determine the equilibrium point and basic reproduction number. In this part, the equilibrium points of the model are calculated. The basic reproduction number is also determined using the next generation matrix. Fourth, we analyze the local and global stability of the equilibrium point. In this step, we examine the stability of equilibrium points both locally and globally to understand the long-term behavior of the proposed model. Fifth, we analyze the sensitivity of parameters. Sensitivity analysis is conducted to identify the most influential parameters in the basic reproduction number. The last, we conduct numerical simulations to confirm the analytical result. Numerical simulations are performed to verify the analytical result obtained from the proposed model.

RESULTS AND DISCUSSION

Model Formulation

In this section, we formulate an LSD model and assume that the total population of cattle is denoted by (N_c) which consists of susceptible cattle (S_c), vaccinated cattle (V_c), infected cattle (I), and recovered cattle (R_c). Meanwhile, the total population of vector is denoted by (N_v) which consists of susceptible vector (S_v) and infected vector (I_v). The population of pathogen or LSDV in environment is denoted by (P).
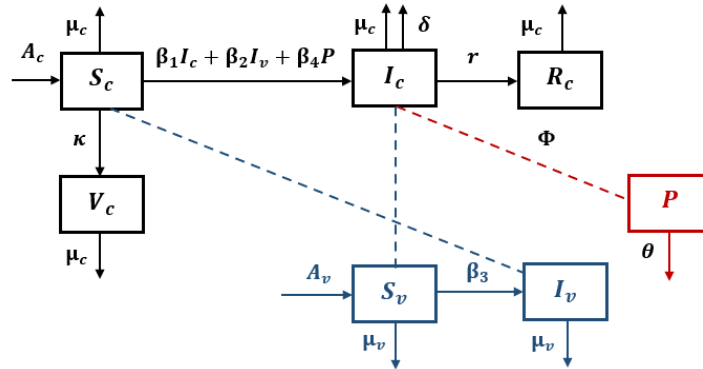

Figure 1. Compartment diagram for lumpy skin disease transmission

The susceptible cattle population grows at a constant birth rate A_c. The susceptible cattle become infected whenever they have contacted with infected cattle at an infection rate β_1, infected vector at an infection rate β_2 and pathogen in the environment at an infection rate β_4. To prevent LSD virus infection, we assume that susceptible cattle are vaccinated at a specific vaccination rate κ. The vaccination is assumed to be perfect such that the vaccinated cattle cannot be infected by the LSD virus [18]. The number of infected cattle may be reduced due to they recover at rate r, or death caused by the LSD at rate δ. The natural death rate of catlle is denoted by μ_c. The birth rate of vector is assumed to be A_v and the death rate of vector is μ_v. After having contact (bite) with the infected cattle, the susceptible vector becomes infected with infection rate β_3. It is assumed that infected cattle can release LSD virus into the environment at a release rate Φ. To reduce pathogens in the environment, we apply disinfectants at a rate of θ. The LSD transmission schematic diagram can be seen in Figure 1, while the constructed mathematical model for LSD transmission is as follows

$$
\begin{aligned}
\frac{dS_c}{dt} &= A_c - \xi S_c - (\kappa + \mu_c)S_c, \\
\frac{dV_c}{dt} &= \kappa S_c - \mu_c V_c, \\
\frac{dI_c}{dt} &= \xi S_c - (r + \delta + \mu_c)I_c, \\
\frac{dR_c}{dt} &= rI_c - \mu_c R_c, \\
\frac{dS_v}{dt} &= A_v - \beta_3 S_v I_c - \mu_v S_v, \\
\frac{dI_v}{dt} &= \beta_3 S_v I_c - \mu_v I_v, \\
\frac{dP}{dt} &= \Phi I_c - \theta P
\end{aligned}
\tag{1}
$$

where

$$\xi = \beta_1 I_c + \beta_2 I_v + \beta_4 P$$

Non-Negativity and Boundedness of Solution

System (1) describes the interaction of cattle, vector, and pathogen population. The solution of this model must be non-negative and bounded. The non-negativity of solution is stated in the following theorem.

Theorem 1. All solutions of system (1) with positive initial values $S_c(0) \geq 0, V_c(0) \geq 0, I_c(0) \geq 0, R_c(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0$ and $P(0) \geq 0$ are always non-negative.

Proof:

We first prove that $S_c(0)$ are non-negative. Assume on the contrary; suppose that $S_c(t)$ is negative for some $t > 0$. If t_1 be the first time such that $S_c(t_1) = 0$. From the first equation of system (1), we get

$$\frac{dS_c}{dt}\bigg|_{t=t_1} = A_c > 0$$

This means that $S_c(t) > 0$ on $t \in (t_1, t_1 + \epsilon)$ for arbitrary small positive constant ϵ. This leads to a contradiction, thus $S_c(t) \geq 0$ for all $t \geq 0$. The non-negativity of $V_c(t), I_c(t), R_c(t), S_v(t), I_v(t), P(t)$ can be shown using the similar way. Therefore, all solutions of system (1) are non-negative.

To show the boundedness solutions of system (1), we apply the comparison lemma [19]. Let $N_c = S_c + V_c + I_c + R_c$ and $N_v = S_v + I_v$. Based on system (1), we have

$$\frac{dN_c}{dt} = A_c - \mu_c(S_c + V_c + I_c + R_c) - \delta I_c \leq A_c - \mu_c N_c,$$

$$N_c \leq \frac{A_c}{\mu_c} + \left(N_c(0) - \frac{A_c}{\mu_c}\right)e^{-\mu_c t} \implies \lim_{t \to \infty} N_c \leq \frac{A_c}{\mu_c},$$

$$\frac{dN_v}{dt} = A_v - \mu_v N_v,$$

$$N_v = \frac{A_v}{\mu_v} + \left(N_v(0) - \frac{A_v}{\mu_v}\right)e^{-\mu_v t} \implies \lim_{t \to \infty} N_v \leq \frac{A_v}{\mu_v}.$$

and

$$\frac{dP}{dt} = \Phi I_c - \theta P < \Phi N_c - \theta P \leq \frac{\Phi A_c}{\mu_c} - \theta P,$$

$$P \leq \frac{A_c \Phi}{\mu_c \theta} + \left(P(0) - \frac{A_c \Phi}{\mu_c \theta}\right)e^{-\theta t} \implies \lim_{t \to \infty} P \leq \frac{A_c \Phi}{\mu_c \theta}.$$

Thus, all the solution of the system (1) are bounded to the region

$$\Omega = \left\{ \Gamma_1 \in \mathbb{R}_+^4 : N_c(t) \leq \frac{A_c}{\mu_c}, \Gamma_2 \in \mathbb{R}_+^2 : N_v(t) = \frac{A_v}{\mu_v}, P(t) \in \mathbb{R} : P(t) \leq \frac{A_c \Phi}{\mu_c \theta} \right\}$$

where $\Gamma_1 = (S_c, V_c, I_c, R_c)$ and $\Gamma_2 = (S_v, I_v)$. A summary of the above analysis can be stated in the following theorem.

Theorem 2. All solutions of system (1) with initial values $S_c(0) \geq 0, V_c(0) \geq 0, I_c(0) \geq 0, R_c(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0, P(0) \geq 0$ are bounded.

Equilibrium Point and Basic Reproduction Number

The equilibrium point is obtained when the right-hand side of the equation in system (1) are zero. In this way, we get two types of equilibrium points: the disease-free equilibrium point (DFE)

$$E^0 = (S_C^0, V_c^0, I_c^0, R_c^0, S_v^0, I_v^0, P^0) = \left(\frac{A_c}{\kappa + \mu_c}, \frac{\kappa A_c}{\mu_c(\kappa + \mu_c)}, 0, 0, \frac{A_v}{\mu_v}, 0, 0 \right),$$

and the endemic equilibrium (EE)

$$E^* = (S_C^*, V_c^*, I_c^*, R_c^*, S_v^*, I_v^*, P^*),$$

where

$$S_C^* = \frac{A_c}{\xi + \kappa + \mu_c}, V_c^* = \frac{\kappa S_c^*}{\mu_c}, R_c^* = \frac{r I_c^*}{\mu_v}, S_v^* = \frac{A_v}{\beta_3 I_c^* + \mu_v}, I_v^* = \frac{\beta_3 S_v^* I_c^*}{\mu_v}, P^* = \frac{\Phi I_c^*}{\theta}.$$

Notice that I_c^* are the real positive roots of the following quadratic equation

$$\Pi_0 I_c^{*2} + \Pi I_c^* + \Pi_2 = 0, \tag{2}$$

where

$$\Pi_0 = (r + \delta + \mu_c)(\mu_v \theta \beta_3 + \beta_3 \beta_4 \mu_v \Phi),$$

$$\Pi_1 = \left((r + \delta + \mu_c)(\beta_1 \mu_v^2 \theta + \beta_2 \beta_3 A_c \theta + \beta_4 \Phi \mu_v^2) + (\mu_v \theta \beta_3)(\kappa + \mu_c) \right)$$
$$- (A_c \beta_1 \mu_v \theta \beta_3 + A_c \beta_3 \beta_4 \mu_v \Phi)$$

$$\Pi_2 = \mu_v^2 \theta (\kappa + \mu_c)(r + \delta + \mu_c) \left(1 - 2 \frac{\beta_1 A_c \theta + \beta_4 A_c \Phi}{2\theta(\kappa + \mu_c)(r + \delta + \mu_c)} - \frac{\beta_3 A_v \beta_2 A_c}{\mu_v^2(\kappa + \mu_c)(r + \delta + \mu_c)} \right)$$

The detail discussion about the existence of EE will be given later. Instead, we first derive the basic reproduction number as follows. By only considering the subpopulation with infection in LSD model (1), we consider the following vector of individual displacement between compartments

$$\mathcal{F} = \begin{pmatrix} (\beta_1 I_c + \beta_2 I_v + \beta_4 P) S_c \\ \beta_3 S_v I_c \\ 0 \end{pmatrix}, \mathcal{V} = \begin{pmatrix} r + \delta + \mu_c \\ \mu_v I_v \\ -\Phi I_c + \theta P \end{pmatrix}.$$

Suppose that F and V are respectively the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the DFE, then we get the next generation matrix

$$FV^{-1} = \begin{pmatrix} \dfrac{\beta_1 A_c}{\beta_1 A_c \theta + \beta_4 A_c \Phi} + \dfrac{\beta_4 A_c \Phi}{(\kappa + \mu_c)(r + \delta + \mu_c)\theta} & \dfrac{\beta_2 A_c}{(\kappa + \mu_c)} & \dfrac{\beta_4 A_c}{(\kappa + \mu_c)\theta} \\ \dfrac{\beta_3 A_v}{(r + \delta + \mu_c)\mu_v} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number is defined as the spectral radius of the matrix FV^{-1}, namely

$$R_0 = \rho(FV^{-1}),$$

$$= \frac{\beta_1 A_c \theta + \beta_4 A_c \Phi}{2\theta(\kappa + \mu_c)(r + \delta + \mu_c)} + \sqrt{\frac{(\beta_1 A_c \theta + \beta_4 A_c \Phi)^2}{4\theta^2(\kappa + \mu_c)^2(r + \delta + \mu_c)^2} + \frac{\beta_3 A_v \beta_2 A_c}{\mu_v^2(\kappa + \mu_c)(r + \delta + \mu_c)}},$$

$$= R_1^* + \sqrt{R_1^{*2} + R_2^*},$$

where

$$R_1^* = \frac{\beta_1 A_c \theta + \beta_4 A_c \Phi}{2\theta(\kappa + \mu_c)(r + \delta + \mu_c)} \text{ and } R_2^* = \frac{\beta_3 A_v \beta_2 A_c}{\mu_v^2(\kappa + \mu_c)(r + \delta + \mu_c)}.$$

By noticing that $R_0^2 = 2R_1^* R_0 + R_2^*$, we can show that condition $(R_0 \leq 1 \text{ or } R_0 > 1)$ is equivalent to condition $(R_0^* \leq 1 \text{ or } R_0^* > 1)$ where $R_0^* = 2R_1^* + R_2^*$. We can imply that R_1^* is the basic reproduction number that caused by the infection rate of infected cattle and virus in environment. Similarly, R_2^* is the basic reproduction number that caused by the infection rate of infected vector.

Next, we can write the coefficient Π_2 of the quadratic equation (2) as
$$\Pi_2 = \mu_v^2 \theta (\kappa + \mu_c)(r + \delta + \mu_c)(1 - R_0^*).$$
By evaluating all possible roots of equation (2), we have the following existence of endemic equilibrium point:

1) If $R_0^* < 1$, $\Pi_1 < 0$ and $\Pi_1^2 - 4\Pi_0 \Pi_2 > 0$ then there exist two endemic equilibrium points $I_{c_1}^*$ and $I_{c_2}^*$;
2) If $R_0^* < 1$, $\Pi_1 < 0$ and $\Pi_1^2 - 4\Pi_0 \Pi_2 = 0$ then there exists a unique endemic equilibrium point $I_{c_3}^*$;
3) If $R_0^* = 1$ and $\Pi_1 < 0$ then there exists a unique endemic equilibrium point $I_{c_4}^*$;
4) If $R_0^* > 1$ then there exists a unique endemic equilibrium point $I_{c_1}^*$;
5) No equilibria otherwise;

where

$$I_{c_1}^* = \frac{-\Pi_1 + \sqrt{\Pi_1^2 - 4\Pi_0 \Pi_2}}{2\Pi_0}, \quad I_{c_2}^* = \frac{-\Pi_1 - \sqrt{\Pi_1^2 - 4\Pi_0 \Pi_2}}{2\Pi_0}, \quad I_{c_3}^* = -\frac{\Pi_1}{2\Pi_0}, \quad I_{c_4}^* = -\frac{\Pi_1}{\Pi_0}.$$

Local Stability

In this section, we investigate the local stability of equilibrium point in model (1). The Jacobian matrix from the sytem (1) at a point $E^k = (S_c^k, V_c^k, I_c^k, R_c^k, S_v^k, I_v^k, P^k)$ is given by

$$J(E^k) = \begin{bmatrix} -s_1^k & 0 & -\beta_1 S_c^k & 0 & 0 & -\beta_2 S_c^k & -\beta_4 S_c^k \\ \kappa & -\mu_c & 0 & 0 & 0 & 0 & 0 \\ s_1^k & 0 & s_2^k & 0 & 0 & \beta_2 S_c^k & \beta_4 S_c^k \\ 0 & 0 & r & -\mu_c & 0 & 0 & 0 \\ 0 & 0 & -\beta_3 S_v^k & 0 & -(\beta_3 I_c^k + \mu_v) & 0 & 0 \\ 0 & 0 & \beta_3 S_v^k & 0 & \beta_3 I_c^k & -\mu_v & 0 \\ 0 & 0 & \Phi & 0 & 0 & 0 & -\theta \end{bmatrix}, \quad (3)$$

where
$$s_1^k = \beta_1 I_c^k + \beta_2 I_v^k + \beta_4 P^k + \kappa + \mu_c,$$
$$s_2^k = \beta_1 S_c^k - \delta - r - \mu_c.$$

We substitute the equilibrium point E^k into the Jacobian matrix to find out the eigenvalues thus we get the stability condition for the DFE and EE.

If we substitute the DFE point E^0 into the Jacobian matrix (3) then we get $J(E^0)$. By using $|J(E^0) - \lambda I| = 0$ to find out the eigenvalues of $J(E^0)$, we get the characteristic equation as follow

$$(-\kappa - \mu_c - \lambda)(-\mu_c - \lambda)(-\mu_c - \lambda)(-\mu_v - \lambda)(\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3) = 0, \quad (4)$$
where
$$a_1 = (r + \delta + \mu_c)\left(1 - \frac{\beta_1 A_c}{(\kappa + \mu_c)(r + \delta + \mu_c)}\right) + \theta + \mu_v,$$

$$a_2 = \theta(r + \delta + \mu_c)(1 - 2R_1^*) + \mu_v(r + \delta + \mu_c)(1 - R_2^*) + \mu_v\left(\theta - \frac{\beta_1 A_c}{\kappa + \mu_c}\right),$$

$$a_3 = \theta(r + \delta + \mu_c)\mu_v^2(1 - R_0^*).$$

It is clear that the real part of the fourth eigenvalues are

$$\lambda_1 = -\kappa - \mu_c < 0, \qquad \lambda_{2,3} = -\mu_c < 0, \qquad \lambda_4 = -\mu_v < 0.$$

Next we find the other eigenvalues in (4) by using the characteristic equation below

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0 \tag{5}$$

Based on the Routh-Hurwitz criteria, the characteristic equation (5) has the negative real part in each of their eigenvalues if $a_1 > 0$ and $a_1 a_2 - a_3 > 0$. We see that if $R_0^* < 1$ then $a_3 > 0$ and other criteria will be proven by numerical simulations. The DFE point is locally asymptotically stable if the LSD model satisfies all of the criteria then the following theorem holds.

Theorem 3. The DFE point E^0 of the LSD model is locally asymptotically stable if $R_0^* < 1$, $a_1 > 0$, and $a_1 a_2 - a_3 > 0$.

To prove the local stability of the EE point E^*, we substitute E^* into the Jacobian matrix (3) then we get $J(E^*)$. By using $|J(E^*) - \lambda I| = 0$ then we get the eigenvalues of $J(E^*)$, from the characteristic equation below

$$(-\mu_c - \lambda)(-\mu_c - \lambda)(\lambda^5 + b_1\lambda^4 + b_2\lambda^3 + b_3\lambda^2 + b_4\lambda + b_5) = 0. \tag{6}$$

It is clear that the real part of the first and second eigenvalues are negative $\lambda_{1,2} = -\mu_c$. We can find out the other eigenvalues by evaluating

$$\lambda^5 + b_1\lambda^4 + b_2\lambda^3 + b_3\lambda^2 + b_4\lambda + b_5 = 0, \tag{7}$$

where

$b_1 = \beta_3 I_c^* + \theta + 2\mu_v + s_1^* - s_2^*,$

$b_2 = -S_c^* S_v^* \beta_2\beta_3 - \Phi S_c^* \beta_4 + \theta I_c^* \beta_3 + I_c^* \beta_3\mu_v + I_c^*\beta_3 s_1^* - I_c^*\beta_3 s_2^* + S_c^*$
$\quad + \beta_1 s_1^* + 2\theta\mu_v\theta s_1^* - \theta s_2^* + \mu_v^2 + 2\mu_v s_1^* - 2\mu_v s_2^* - s_1^* s_2^*,$

$b_3 = -\Phi I_c^* S_c^*\beta_3\beta_4 - \theta S_c^* S_v^*\beta_2\beta_3 - S_c^* S_v^* \beta_2\beta_3\mu_v - 2\Phi S_c^*\beta_4\mu_v + \theta I_c^*\beta_3 (\mu_v + s_1^* - s_2^*)$
$\quad + S_c^*\beta_1 s_1^*(I_c^* + 2\mu_v) + I_c^*\beta_3\mu_v (s_1^* - s_2^*) - I_c^* \beta_3 s_1^* s_2^* + \theta\mu_v^2 + 2\theta \mu_v s_1^* - 2\theta \mu_v s_2^*$
$\quad - \theta s_1^* s_2^* + \mu_v^2 (s_1^* - s_2^*) - 2\mu_v s_1^* s_2^*,$

$b_4 = -\Phi I_c^* S_c^*\beta_3\beta_4\mu_v + \theta I_c^* S_c^*\beta_1\beta_3 s_1^* - \theta S_c^* S_v^*\beta_2\beta_3\mu_v$
$\quad + I_c^* S_c^* \beta_1\beta_3\mu_v s_1^* - \Phi S_c^*\beta_4\mu_v^2\theta I_c^*\beta_3\mu_v s_1^* - \theta I_c^*\beta_3\mu_v s_2^* - \theta I_c^*\beta_3 s_1^* s_2^*$
$\quad + 2\theta S_c^*\beta_1^*\mu_v s_1^* - I_c^*\beta_3\mu_v s_1^* s_2^* + S_c^*\beta_1\mu_v^2 s_1^* + \theta\mu_v^2 s_1^* - \theta\mu_v^2 s_2^*$
$\quad - 2\theta\mu_v s_1^* s_2^* - \mu_v s_1^* s_2^*,$

$b_5 = \theta I_c^* S_c^*\beta_1\beta_3\mu_v s_1^* - \theta I_c^*\beta_3\mu_v s_1^* s_2^* + \theta S_c^*\beta_1\mu_v^2 s_1^* - \theta\mu_v^2 s_1^* s_2^*,$

Based on the Lienard-Chipart criteria[20], the characteristic equation (7) has the negative real part in each of their eigenvalues if and only if $b_1 > 0$, $b_3 > 0$, $b_5 > 0$, $H_2^* > 0$ and $H_4^* > 0$, where

$$H_2^* = b_1 b_2 - b_3$$

And

$$H_4^* = b_1(2b_4 b_5 - b_1 b_4 - b_2^2 b_5 + b_2 b_3 b_4) + b_3(b_2 b_5 - b_3 b_4) - b_5^2$$

Since the forms of b_i, $i = 1, \dots, 5$ are too complicated, the Lienard-Chipart criteria will be calculated numerically. The EE point is locally asymptotically stable if the LSD model satisfies all of the criteria then the following theorem holds.

Theorem 4. Let the EE point E^* of LSD model (1) exists. The EE point E^* is locally asymptotically stable if $b_1 > 0$, $b_3 > 0$, $b_5 0$, $H_2^* > 0$, $H_4^* > 0$.

Global Stability

The method to examine the global stability of DFE is Castillo-Chavez method in [21]. The LSD model (1) can be transformed as follows:

$$\frac{d\vec{Y_1}}{dt} = P(\vec{Y_1}, \vec{Y_2}),$$

$$\frac{d\vec{Y_2}}{dt} = Q(\vec{Y_1}, \vec{Y_2}), Q(\vec{Y_1}, 0) = 0, \tag{8}$$

Where

$$\vec{Y_1} = (S_c, V_c, R_c, S_v)^T, \vec{Y_2} = (I_c, I_v, P)^T,$$

$$\vec{Y_1^0} = (S_c^0, V_c^0, R_c^0, S_v^0)^T = \left(\frac{A_c}{\kappa + \mu_c}, \frac{\kappa A_c}{\mu_c(\kappa + \mu_c)}, 0, \frac{A_v}{\mu_v}\right)^T,$$

$\vec{Y_2^0} = (I_c^0, I_v^0, P^0)^T = (0,0,0)^T = \vec{0}$, and E^0 is the DFE point.

To ensure the global asymptotic stability of the DFE point, the criteria below must be satisfied.

1) For $\frac{d\vec{Y_1}}{dt} = P(\vec{Y_1}, 0), \vec{Y_1^0}$ is globally asymptotically stable.
2) $Q(\vec{Y_1}, \vec{Y_2}) = M\vec{Y_2} - \hat{Q}(\vec{Y_1}, \vec{Y_2}), \hat{Q}(\vec{Y_1}, \vec{Y_2}) \geq 0$ for all $\vec{Y_1}, \vec{Y_2} \in \Omega$ and $M = F - V$

where M is the matrix which the other elements than the main diagonal are non-negative and Ω is the region where the model makes biological sense. Consequently, if the LSD model satisfies the conditions then the following theorem holds.

Theorem 5. The DFE point $E^0 = (\vec{Y_1^0}, 0)$ of the LSD model (1) is globally asymptotically stable in Ω if $R_0^* < 1$.

Proof:

From the system (1), we have $\frac{d\vec{Y_1}}{dt}$ and $\frac{d\vec{Y_2}}{dt}$ below

$$P(\vec{Y_1}, \vec{Y_2}) = \begin{bmatrix} A_c - \xi S_c - (\kappa + \mu_c) \\ \kappa S_c - \mu_c V_c \\ r I_c - \mu_c R_c \\ A_v - \beta_3 S_v I_c - \mu_v S_v \end{bmatrix}, Q(\vec{Y_1}, \vec{Y_2}) = \begin{bmatrix} \xi S_c - (r + \delta + \mu_c) I_c \\ \beta_3 S_v I_c - \mu_v I_v \\ \Phi I_c - \theta P \end{bmatrix},$$

where $\xi = \beta_1 I_c + \beta_2 I_v + \beta_4 P$. If $\vec{Y_2} = \vec{0}$, we get $\frac{d\vec{Y_1}}{dt}$ as follows:

$$\frac{d\vec{Y_1}}{dt} = P(\vec{Y_1}, \vec{0}) = \begin{bmatrix} A_c - (\kappa + \mu_c) \\ \kappa S_c - \mu_c V_c \\ r I_c - \mu_c R_c \\ A_v - \mu_v S_v \end{bmatrix}. \tag{9}$$

Next, we solve the equation (9) to get the solution as follows

$$S_c = \frac{A_c}{\kappa + \mu_c} + Ce^{-(\kappa + \mu_c)t}, V_c = \frac{\kappa A_c}{\mu_c(\kappa + \mu_c)} + Ce^{-\mu_c t}, R_c = e^{-\mu_c t}, S_v = \frac{A_v}{\mu_v} + Ce^{-\mu_v t}.$$

Note that the limiting sytem of $\frac{d\vec{Y_1}}{dt} = P(\vec{Y_1}, \vec{0})$ as follows:

$$\lim_{t \to \infty} S_c = \frac{A_c}{\kappa + \mu_c} + Ce^{-(\kappa + \mu_c)\infty} = \frac{A_c}{\kappa + \mu_c},$$

$$\lim_{t \to \infty} V_c = \frac{\kappa A_c}{\mu_c(\kappa + \mu_c)} + Ce^{-\mu_c \infty} = \frac{\kappa A_c}{\mu_c + (\kappa + \mu_c)},$$

$$\lim_{t \to \infty} R_c = e^{-\mu_c \infty} = 0,$$

$$\lim_{t \to \infty} S_v = \frac{A_v}{\mu_v} + Ce^{-\mu_c \infty} = \frac{A_v}{S_v}.$$

Based on the limiting system, the solution converge to $\vec{Y_1^0} = \left(\frac{A_c}{\kappa + \mu_c}, \frac{\kappa A_c}{\mu_c(\kappa + \mu_c)}, 0, \frac{A_v}{\mu_v}\right)$ thus

$\vec{Y}_1^0$ globally asymptotically stable in $P(\vec{Y}_1^0, \vec{0})$.

Next, we determine the matrix M by calculating $F - V$. We get the matrices of F and V from the section of basic reproduction number then M is stated by

$$M = \begin{bmatrix} \beta_1 S_c^0 - (r + \delta + \mu_c) & \beta_2 S_c^0 & \beta_4 S_c^0 \\ \beta_3 S_v^0 & -\mu_v & 0 \\ \Phi & 0 & -\theta \end{bmatrix}.$$

Next, based on the equation $Q(\vec{Y}_1, \vec{Y}_2) = M\vec{Y}_2 - \hat{Q}(\vec{Y}_1, \vec{Y}_2)$, we get $\hat{Q}(\vec{Y}_1, \vec{Y}_2)$ below

$$\hat{Q}(\vec{Y}_1, \vec{Y}_2) = \begin{bmatrix} (\beta_1 I_c + \beta_2 I_v + \beta_4 P)S_c^0 - (r + \delta + \mu_c)I_c \\ \beta_3 S_v^0 I_c - \mu_v I_v \\ \Phi I_c - \theta P \end{bmatrix} = \begin{bmatrix} (\beta_1 I_c + \beta_2 I_v + \beta_4 P)(S_c^0 - S_c) \\ \beta_3 I_c(S_v^0 - S_v) \\ 0 \end{bmatrix}.$$

The value of $\hat{Q}(\vec{Y}_1, \vec{Y}_2) \geq 0$ if $S_c^0 \geq S_c, S_v^0 \geq S_v$ and M have the other element than the main diagonal are non-negative. Based on the theorem 5, the DFE is globally asymptotically stable when $R_0^* < 1$ for all $(S_c^0 \geq S_c, S_v^0 \geq S_v) \in \Omega$. The first and the second criteria are satisfied then the theorem 5 is proven.

Sensitivity Analysis

In this section, we find out the most influential parameters in the basic reproduction number R_0^*. We must calculate the normalized sensitivity index that defined in [22] as follows

$$\Upsilon_\Psi^{R_0^*} = \frac{\partial R_0^*}{\partial \Psi} \times \frac{\Psi}{R_0^*} \tag{10}$$

where $\Psi = \beta_1, \beta_2, \beta_3, \beta_4, \theta, \Phi, \kappa$. We apply the equation (8) to get the normalized sensitivity index below

$$\begin{aligned}
\Upsilon_{\beta_1}^{R_0^*} &= \frac{\beta_1 \mu_v^2 \theta}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\beta_2}^{R_0^*} &= \frac{\theta \beta_3 A_v \beta_2}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\beta_3}^{R_0^*} &= \frac{\theta \beta_3 A_v \beta_2}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\beta_4}^{R_0^*} &= \frac{\Phi \mu_v^2 \beta_4}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\theta}^{R_0^*} &= -\frac{\Phi \mu_v^2 \beta_4}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\Phi}^{R_0^*} &= \frac{\Phi \mu_v^2 \beta_4}{\beta_1 \mu_v^2 \theta + \mu_v^2 \beta_4 \Phi + \beta_3 A_v \beta_2 \theta} \\
\Upsilon_{\theta}^{R_0^*} &= -\frac{\kappa}{\kappa + \mu_c}
\end{aligned} \tag{11}$$

Based on equation (11), the value of sensitivity index of θ and κ have negative impact on R_0^*. It means, if θ and κ increase then R_0^* will decrease. The parameters $\beta_1, \beta_2, \beta_3, \beta_4$ and Φ have positive impact on R_0^*. It means, if they are increase then R_0^* also increase. Next, we substitute the parameters in Table 1 into (11) to find out the value of sensitivity index in Table 2. By substituting the parameters in Table 1 into basic reproduction number, we get the value of $R_0^* = 0.9919$.

Now, let see in the Table 2 and Table 3, we get the information about the value of sensitivity index and the effect of changes in parameter value on R_0^*. The increasing of the

parameters values β_4 and κ cause the significant changes in R_0^*. This indicates that an effective strategy to reduce LSD outbreaks is to perform environmental disinfection and administer vaccination to susceptible cattle.

Table 1. The parameters of the model and their description

Parameters	Descriptions	Value	Source
A_c	The growth rate of healthy cattle	0.8	[11]
A_v	The growth rate of vectors	0.1	[11]
β_1	The contact rate due to vectors and healthy cattle	0.01120	[11]
β_2	The contact rate due to vectors and healthy cattle	0.030013	[11]
β_3	The contact rate due to infected cattle and vector	0.03	[11]
β_4	The contact rate due to contaminated environment	0.1010	[11]
μ_c	The natural death rate of cattle	0.00045662	[11]
μ_v	The natural death rate of vector	0.07	Assumed
r	The recovery rate of the infected cattle	0.095	Assumed
δ	The death of infected cattle are caused by disease	0.027	[7]
κ	The vaccination rate of healthy cattle	0.851	Assumed
Φ	The rate of LSDV in the environment	0.001	[11]
θ	The virus disinfection rate	0.001013	[11]

Table 2. The sensitivity index value of parameters

Parameters	Sensitivity Index	Parameters	Sensitivity Index
β_1	0.0866	θ	-0.7712
β_2	0.1421	Φ	0.7712
β_3	0.1421	κ	-0.9994
β_4	0.7712		

Table 3. The impact of changes in parameters value on R_0^*

Parameters	Parameters+10%	Parameters−10%
β_1	1.0005	0.9833
β_2	1.0006	0.9778
β_3	1.0006	0.9778
β_4	1.0684	0.9154
θ	0.9223	1.0769
Φ	1.0684	0.9154
κ	0.8527	1.1024

Numerical Simulation

In this section, we present the results of numerical simulations using MATLAB R2021a software to illustrate the dynamics of LSD. In the first numerical simulation, we want to observe the effect of β_4 and κ on R_0^*. We use the parameters from Table 1 and the simulations show in Figure 2. First, if the value of β_4 increase, the value of R_0^* also increase. The blue graph indicates that $R_0^* < 1$ when $\beta_4 < 0.06$. Conversely, the red graph shows that $R_0^* > 1$ when $\beta_4 > 0.06$. Second, if the value of κ increase, the value of R_0^* decrease. The blue graph indicates that $R_0^* < 1$ when $\kappa > 0.84$. Conversely, the red

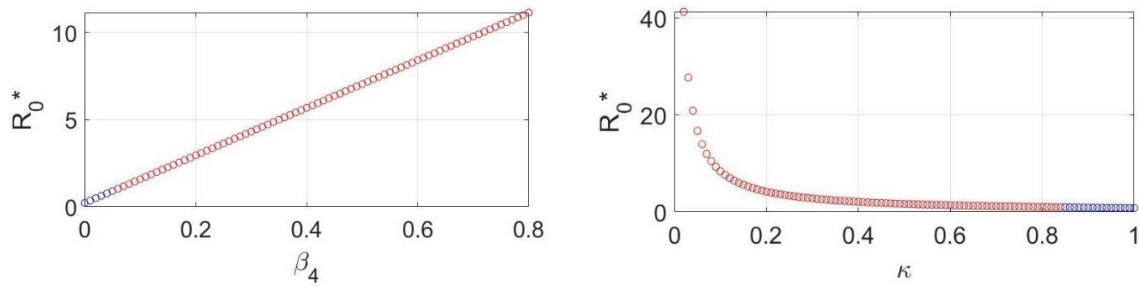graph shows that $R_0^* > 1$ when $\kappa < 0.84$.

Figure 2. The effect of β_4 and κ on R_0^*

In the second numerical simulation, we observe the dynamics of the LSD model (1) using the fourth-order Runge-Kutta method and the parameters in Table 1, except β_4. Here we choose four different values of β_4 to study the effect of the parameter changes in the dynamical population. The values of β_4 are $0.5,\ 0.1, 0.05, 0.01$ and the step size is $h = 0.1$. By selecting these values of β_4, the corresponding values of R_0^* are $4.0140, 0.9843, 0.605, 0.3027$. The initial values are used to be $N(0) = (500,25,15,20,10,10,5)$. This simulation is shown in Figure 3.
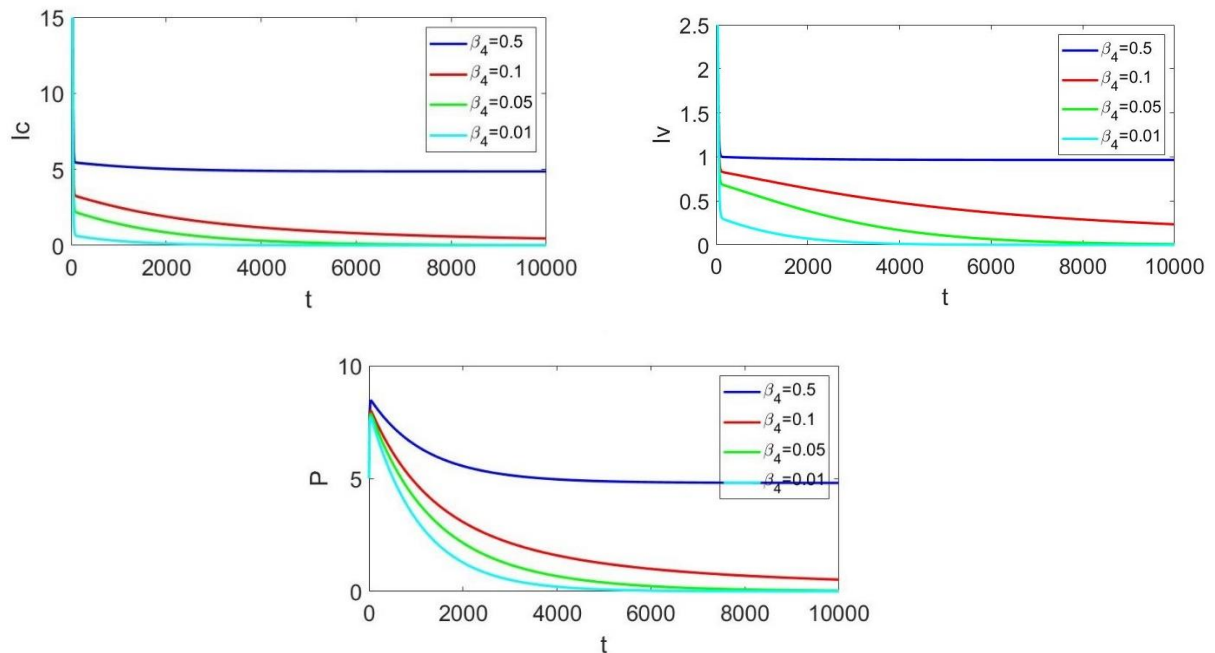

Figure 3. The numerical simulation with several values of β_4 in I_c, I_v, P

From Figure 3 we know that the more interactions between susceptible cattle and the LSD virus in the environment, it can increase the number of infected cattle, infected vectors and virus population. However, the fewer interactions between susceptible cattle and the LSD virus in the environment, it can effectively decrease the number of infected cattle subpopulation, infected vectors subpopulation, and virus population.

Next, we take the values of parameters as in Table 1 but with four different values of κ, namely $\kappa = 1.2, 0.8, 0.5, 0.15$. By selecting these values of κ, the corresponding values of R_0^* are $0.8526, 1.0551, 1.6876,\ 5.6133$. The initial values are used to be $N(0) = (500,25,15,20,10,10,5)$. This simulation is shown in Figure 4.
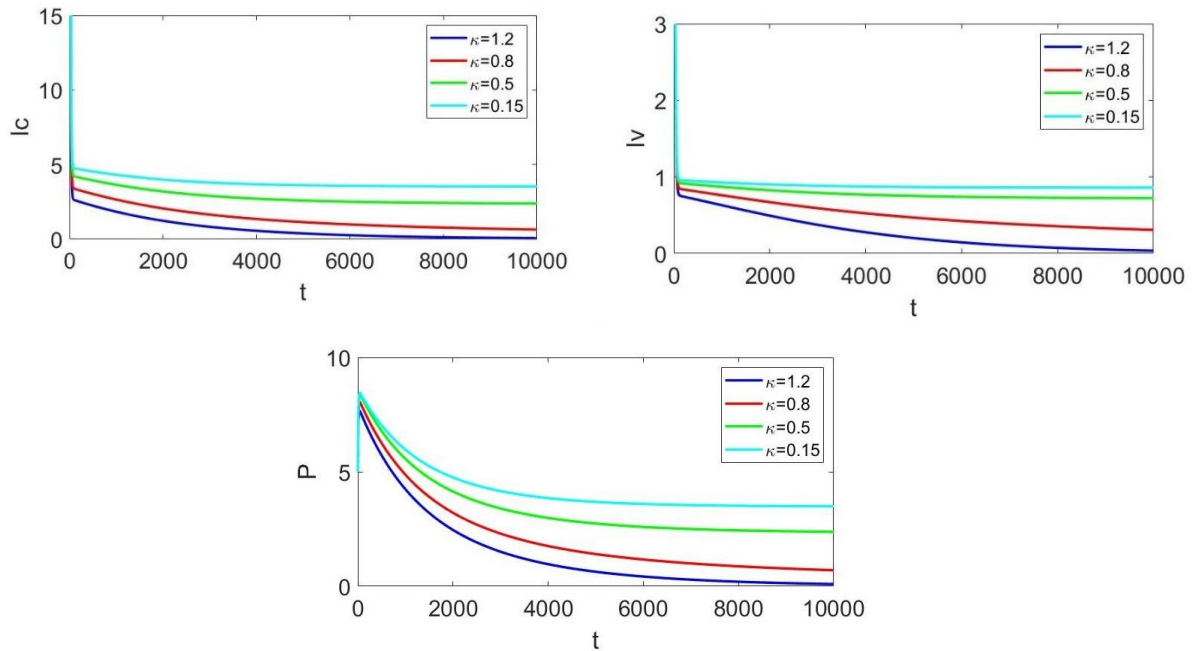
Figure 4. The numerical simulation with several values of κ in I_c, I_v, P

Based on the Figure 4 we get information, by increasing vaccination to susceptible cattle, it is effectively to reduce the number of infected cattle subpopulation, infected vectors subpopulation, and virus population. However, by decreasing vaccinations to susceptible cattle, it can increase the number of infected cattle subpopulation, infected vectors subpopulation, and virus population.

Discussion

In this section, we discuss the comparison of our research and previous research. Based on the results in our study, the proposed LSD model that we introduce similar to the model in [17], the LSD transmission through cattle, vector, and pathogen (virus) population. The key difference of our and their research is that our model consider vaccination for susceptible cattle, we define as vaccinated cattle subpopulation (V_c) and it is not present in their model. Highlighting the novelty of our model that vaccination can control the disease. In addition, the effect of vaccination in our study also give impact for infected cattle and infected vectors, as we see in [16]. By using our proposed model, we know that vaccination not only influences the number of infected individuals but also affects the amount of virus in the environment. The broader perspective about vaccination adds new layer to understand the dynamics of LSD. This comparative analysis illustrates how our study builds on previous research by introducing novel elements.

CONCLUSIONS

In this article, we develop the LSD model that contains seven compartments: susceptible cattle, vaccinated cattle, infected cattle, recovered cattle, susceptible vector, infected vector, and LSDV in the environment. The non-negativity and boundedness of the solutions for the proposed LSD spread model are proven. The LSD model has two equilibrium points, there are the DFE point (E^0) and the EE point (E^*). The DFE point exists and the stability is asymptotically stable for local and global if $R_0^* < 1$. The EE exists if it satisfies the criteria and the stability is locally asymptotically stable if it satisfies the Lienard-Chipart criteria.

From the sensitivity analysis, the parameters β_4 and κ has significant change in the value of R_0^* . It shows the appropriate strategy to address the LSD outbreak is to increase vaccination for healthy cattle and to reduce the LSD virus in the environment by performing environmental disinfection.

REFERENCES

[1] A. Sprygin, Ya. Pestova, D. B. Wallace, E. Tuppurainen, A. V. Kononov, " Transmission of lumpy skin disease virus: A short review," *Virus Research,* pp. 269, 2019. https://doi.org/10.1016/j.virusres.2019.05.015.

[2] F. Namazi, A. K. Tafti, "Lumpy skin disease, an emerging transboundary viral disease: A review," *Vet. Med. Sci,* vol. 7, pp. 888-896, 2021. https://doi.org/10.1002/vms3.434.

[3] M. Das, M. S. R. Chowdhury, S. Akter, A. K. Mondal, M. J. Uddin, M. M. Rahman, "An updated review on lumpy skin disease: Perspective of Southeast Asian countries", *Journal of Advanced Biotechnology and Experimental Therapeutics*, vol.4(3), pp. 322-333, 2021, https://doi.org/10.5455/jabet. 2021.d133.

[4] X. Roche, A. Rozstalnyy, D. TagoPacheco, C. Pittiglio, A. Kamata, D. Beltran, K. Bisht, S. Karki, J. Kayamori, F. Larfaoui, E. Raizman, S. Von Dobschuetz, M. S. Dhingra, K. Sumption, "Introduction and spread of lumpy skin disease in South, East and Southeast Asia,"*FAO*, 2020, https://doi.org/10.4060/cb1892en.

[5] I. Shumilova, A. Nesterov, O. Bykovskaya, P. Prutnikov, D. B. Wallace, M. Mokeeva, V. Pronin, A. Kononov, I. Chvala, A. Sprygin, " A Recombinant Vaccine-like Strain of Lumpy Skin Disease Virus Causes Low-Level Infection of Cattle through Virus-Inoculated Feed," *Pathogens,* vol.11, pp. 920-935, 2022, https://doi.org/10.3390/pathogens11080920.

[6] S. Lee, C. M. Baker, E. Sellens, M. A. Stevenson, S. Roche, R. N. Hall, A. C. Breed, S. M. Firestone, "A systematic review of epidemiological modelling in response to lumpy skin disease outbreaks," *Frontiers in Veterinary Science*, vol. 11, 2024, https://doi.org/10.3389/fvets.2024.1459293.

[7] A. Anwar, K. Na-Lampang, N. Preyavichyapugdee, V. Punyapornwithaya, "Lumpy skin disease outbreaks in Africa, Europe, and Asia (2005-2022): Multiple change point analysis and time series forecast", *Viruses*, vol.14(10), 2022, https://doi.org/10.3390/v14102203.

[8] Direktorat Jenderal Peternakan, Lumpy Skin Disease (LSD), Kementerian Peternakan, 2022.

[9] A. Kononov, P. Prutnikov, I. Shumilova, S. Kononova, A. Nesterov, O. Byadovskaya, Y. Pestova, V. Diev, A. Sprygin, "Determination of lumpy skin disease virus in bovine meat and offal products following experimental infection," *Transboundary and Emerging Disease*, vol. 66, 2019, https://doi.org/10.1111/tbed.13158.

[10] A. A. M. Daud, "A note on lienard-chipart criteria and its application to epidemic models," *Mathematics and Statistics*, vol. 9, pp. 41-45, 2021, http://dx.doi.org/10.13 189/ms.2021.090107.

[11] T. Singhla, K. Boonsri, K. Kreausukon, W. Modethed, K. Pringproa, N. Sthitmatee, V. Punyapornwithaya, P. Vinitchaikul, "Molecular characterization and phylogenetic analysis of lumpy skin disease virus collected from outbreaks in northern Thailand

in 2021," *Veterinary Sciences*, vol.9(4), pp. 194, 2022, https://doi.org/10.3390/vetsci9040194.

[12] X. S. Wang, J. Wu, Y. Yang, "Richards model revisited: Validation by and application to infection dynamics," *Journal of Theoretical Biology*, pp. 313, 2012. https://doi.org/10.1016/j.jtbi.2012.07.024.

[13] H. Y. Abdulqa, H. Rahman, H. Dyary, H. Othman, "Lumpy Skin Disease," *Reproductive Immunology*, vol. 4, 2016, http://dx.doi.org/10.21767/2476-1974.100025.

[14] A. I. Butt, H. Aftab, M. Imran, T. Ismaeel, "Mathematical study of lumpy skin disease with optimal control analysis through vaccination," *Alexandria Engineering Journal,* vol. 72 , 2023, https://doi.org/10.1016/j.aej.2023.03.073.

[15] O. O. Onyejekwe, A. Alemu, B. Ambachew, A. Tigabie. Royle, "Epidemiological Study and Optimal Control for Lumpy Skin Disease (LSD) in Ethiopia,"*Advances in Infectious Diseases,* vol. 9, pp. 8-24, 2019, https://doi.org/10.4236/aid.2019.91002.

[16] O. D. Falowo, J. A. Owolabi, O. Oludoun, R. Akingbade, "Mathematical Modelling of Lumpy Skin Disease in Dairy Cow," *IOP Conf. Series Earth and Env. Sci,* 2023, http://dx.doi.org/10.1088/1755-1315/1219/1/012007.

[17] W. F. Alfwzan, M. H. Dasassi, F. M. Allehiany, M. A. Khan, M. Y. Alshahrani, E. M. Tag-eldin,"A novel mathematical study to understand the Lumpy skin disease (LSD) using modified parameterized approach," *A Result in Physics,* vol. 51, 2023, http://dx.doi.org/10.1016/j.rinp.2023.106626.

[18] H. T. T. Tran, A. D. Truong, A. K. Dang, D. V. Ly, C. T. Nguyen, N. T. Chu, T. V. Hoang, H. T. Nguyen, V. T. Nguyen, H. V. Dang, "Lumpy skin disease outbreaks in Vietnam, 2020," *Transboundary and Emerging Diseases*, vol. 68(3), pp. 977-980, 2020, https://doi.org/10.1111/tbed.14022.

[19] W. N. Li, W. Sheng, "Some gronwall type inequalities on time scales," *Journal of Mathematical Inequalities*, vol. 4, pp.67-76, 2010.

[20] A. A. M. Daud, "A note on lienard-chipart criteria and its application to epidemic models", *Mathematics and Statistics*, vol.9(1), pp.41-45, 2021, DOI: 10.13189/ms.2021.090107

[21] C. Castillo-Chavez, Z. Feng, W. Huanz, P.V.D. Huanz, .E. Kirschner, On the computation of R0 and its role in global stability, Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction, Springer-Verlag, vol. 125, 2002, https://doi.org/10.1007/978.1.4757.3667-013.

[22] N. Chitnis, J. Hyman, dan J. Chusing, "Determining Important Parameters in the Spread of Malaria Through the Sensitivity Analysis of a Mathematical Model", *Bulletin of Mathematical Biology*. vol.70, pp. 1272-1296, 2008, DOI 10.1007/s11538-008-9299-0.