Stability Analysis of Rabies Model

with Vaccination Effect and Culling in Dogs

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Abstract

This paper considers a deterministic model for the transmission dynamics of rabies virus in the wild dogs - domestic dogs - human zoonotic cycle. The effect of vaccination and culling in dogs is considered on the model, then the stability was analysed to get basic reproduction number. We use the next generation matrix method and Routh Hurwitz test to analyze the stability of the Disease Free Equilibrium and Endemic Equilibrium of this model.

Keywords: Stability analysis, rabies model, rabies vaccination, culling in dogs.
I Introduction

Rabies is a viral disease that affects the central nervous system. All mammals including domestic and non-domestic animals and humans are susceptible to rabies. The rabies virus is a lyssavirus, a virus in the Rhabdoviridae family responsible for causing encephalities. The name Rhabdo comes from the Greek and identifies the characteristic bullet or rod-shape of the viruses. This virus is transmitted through the saliva of an infected animal. Infections occur primarily via bite wounds, or infected saliva entering an open cut or wound or mucous membrane, such as those in the mouth, nasal cavity or eyes. The virus will generally remain at the entry site for a period of time before traveling along the nerves to the brain. In the brain, the virus multiplies quickly, resulting in clinical signs. The virus then moves from the brain along nerves to the salivary glands. The disease has an incubation period from several days until six months, and symptoms usually take several weeks to appear after infection. Rabies is always fatal in animals one symptoms appear [1].

In all continents except Antarctica the rabies virus is present. In some countries, the disease remains endemic with rabies present mainly in wild animal hosts. Dogs continue to be the main carrier of rabies in Africa and Asia and are responsible for most of the human rabies worldwide [2, 3, 4]. Humans most often become infected with rabies through the bite or scratch of an infected dog or cat [5].

In Indonesia, rabies is a re-emerging disease problem. A recent outbreak of rabies in Bali has drawn international attention because it is one of the popular tourist destinations of South-east Asia. Being an island country, Indonesia is in a better position to prevent and control rabies compared to other countries in the region, but inter-island movement of dogs and existing socio-cultural factors in relation to dogs contribute to the spread of rabies in Indonesia. Different communities in islands of Indonesia, each with their own unique traditions, have different attitudes towards dogs. Fishermen often take their dogs on extended trips which may include visits to several islands. The constant movement of small boats among the islands of the Indonesian archipelago with dogs on board is the most likely way that rabies could affect currently rabies-free islands.

To prevent the spread of rabies disease, vaccinaton is recommended. But, because of the large toll of the economic burden of rabies, culling is considered as one of strategies to control the spread of the disease [6, 7]. Transmission dynamics and control of Rabies in China has been modelled by Zhang et al [8]. This model analyzed the spread of rabies in dogs and humans with the influence of vaccine and culling in dogs.
The goal of this paper is to investigate models for rabies involving wild dogs, domestic dogs and humans. Stability analysis and the basic reproductive number will show the important rates in controlling the spread of this disease.

2 Model Development

Wild dogs, domestic dogs and humans are considered. Wild dogs are defined as dogs that roam the streets with no owner and domestic dog defined as home dogs that gain the attention of the owner. So the populations of wild dogs and domestic dogs are considered separate. One form of attention of dog owners is providing immunization and vaccine against rabies to their dogs. Culling on domestic dogs may be carried out after the dog is proved having rabies. For wild dogs, the lack of public attention, the high price of vaccine and the difficulty of catching stray dogs make culling the only way to reduce the transmission of rabies from wild dogs.

At first rabies can be transmitted by wild dogs to domestic dogs, then domestic dogs transmit rabies to their owner, but humans can also be infected with rabies directly from wild dogs. Prevention and treatment of rabies in humans is conducted by immunization and vaccine delivery. If someone is bitten by a dog, the first step is to wash the wound with soap followed by administering the vaccine and the laboratory examining brain specimens of the dogs. If the result is negative, vaccine is stopped. If positive, vaccine delivery is continued. Based on the time of administration, there are two types of vaccine, i.e preventative vaccine that is given to healthy individuals to prevent transmission of rabies, and the vaccine that can be given to individuals who have been exposed to rabies, known as Post-Exposure Prophylaxis (PEP) vaccine.

Since most people will go to hospital after being bitten by dogs, we assume that infected people will not infect healthy people and dogs. Since domestic dogs that suffer from rabies are usually treated and quarantined by the owner, it is assumed that domestic dogs do not transmit rabies in wild dogs.

We develop a compartmental model of rabies spread. The population of wild dogs is classified into three subclasses, namely susceptible, exposed and infectious with sizes denoted by $S_L$, $E_L$ and $I_L$. Both domestic dogs and humans are classified each of them into four subclasses, susceptible, exposed, infectious, immune due to vaccination with domestic dogs sizes denoted by $S_D$, $E_D$, $I_D$ and $R_D$ and human sizes denoted by $S_p$, $E_p$, $I_p$ and $R_p$ respectively. The model is a system of eleven ordinary differential equations

$$
\begin{align*}
\dot{S}_L &= \partial_L - (\mu_L + \beta_{LL}I_L + c)S_L \\
\dot{E}_L &= \beta_{LL}S_LI_L - (\mu_L + \varepsilon_L + c)E_L \\
\dot{I}_L &= \varepsilon_LE_L - (\mu_L + \alpha_L + c)I_L \\
\dot{S}_D &= \partial_D - (\mu_D + \beta_{DD}I_D + \beta_{DL}I_L + m_D)S_D + \omega_D R_D + p\theta_D E_D \\
\dot{E}_D &= (\beta_{DD}I_D + \beta_{DL}I_L)S_D - (\mu_D + \varepsilon_D + \delta_D)E_D \\
\dot{I}_D &= \varepsilon_D E_D - (\mu_D + \alpha_D + e)I_D
\end{align*}
$$

(1)
\[ \begin{align*}
\dot{R}_D &= m_D S_D - (\mu_D + \omega_D) R_D + (1 - p) \vartheta_p E_D \\
S_P &= \varrho_p - (\mu_p + \beta_D \varrho_D) L_D + \beta_L \varrho_L + m_p S_P + \omega_p R_P + q \varrho_p E_p \\
\dot{E}_P &= (\beta_D \varrho_D + \beta_L \varrho_L) S_P - (\mu_p + \varepsilon_p + \vartheta_p) E_P \\
I_p &= \varepsilon_p E_p - (\mu_p + \alpha_p) I_p \\
\dot{R}_p &= m_P S_P - (\mu_p + \omega_p) R_P + (1 - q) \varrho_p E_p.
\end{align*} \]

In the population of wild dogs, \( \varrho_L \) denotes the birth rate of wild dogs, \( \mu_L \) is the death rate of wild dogs, \( \beta_{LL} \) states the transmission coefficient between wild dogs, \( \varepsilon_L \) is the latency rate in wild dogs, \( \alpha_L \) is the disease induced mortality of wild dog, and \( c \) is the culling rate of wild dogs. In the population of domestic dogs, \( \varrho_D \) denotes the birth rate of domestic dogs, \( \mu_D \) denotes the death rate of domestic dogs, \( \beta_{DD} \) and \( \beta_{DL} \) state the transmission coefficient between domestic dogs and the transmission coefficient between domestic dogs and wild dogs, respectively. Then, \( \varepsilon_D \) is the latency rate in domestic dogs, \( m_D \) is the vaccination rate on domestic dogs, \( \omega_D \) is the waning immunity in domestic dogs, \( \vartheta_p \) denotes the rate of giving PEP to exposed domestic dogs, \( p \) is the proportion of domestic dogs given PEP that go to susceptible class and \( 1 - p \) is the proportion of domestic dogs given PEP that go to recovered class, and the culling rate of domestic dogs is denoted by \( c \). Similarly, in human population, the birth rate of people, denoted by \( \varrho_p \). The death rate of people, denoted by \( \mu_p \). Then \( \alpha_p \) is the disease induced mortality of people, \( \beta_{DP} \) and \( \beta_{LP} \) defined as the transmission coefficient between domestic dogs and people, and the transmission coefficient between wild dogs and people, respectively. Then \( \varepsilon_p \) is the latency rate in people, \( \vartheta_p \) is the rate of giving PEP to exposed people, \( q \) is the proportion of people given PEP that go to susceptible class, \( 1 - q \) is proportion of people given PEP that go to recovered class, \( m_p \) is the vaccination rate on people, and the last, \( \omega_p \) defined as the waning immunity in people.

All parameters in the model are non-negative, and the model will be analyzed in a biologically-feasible region defined as follows:

\[ \mathcal{D} = \{ (S_L, E_L, I_L, S_D, E_D, I_D, R_D, S_P, E_P, I_P, R_P) \in \mathbb{R}^{11}_+ : S_L \geq 0, E_L \geq 0, I_L \geq 0, S_D \geq 0, E_D \geq 0, I_D \geq 0, R_D \geq 0, S_P \geq 0, E_P \geq 0, I_P \geq 0, R_P \geq 0 \}. \]

### 3 Stability Analysis of Disease-Free Equilibrium

**Theorem 1** The DFE of model (1) is given by

\[ (S_L^*, E_L^*, I_L^*, S_D^*, E_D^*, I_D^*, R_D^*, S_P^*, E_P^*, I_P^*, R_P^*) = \left( \frac{\beta_D k_D}{k_5}, 0, 0, \frac{m_p \beta_D}{k_4 k_7 - m_D \omega_D}, 0, 0, \frac{m_p \beta_D}{k_8 k_11 - m_D \omega_D}, 0, 0, \frac{m_p \beta_D}{k_8 k_11 - m_D \omega_D} \right). \]

Then, the basic reproduction number \( R_0 \) is given by

\[ R_0 = \max \left\{ \frac{\beta_D \varrho_D k_D \varepsilon_D}{(k_4 k_7 - m_D \omega_D) k_6 k_5}, \frac{\beta_{LL} \varepsilon_L}{k_1 k_3 k_2} \right\}. \]
where \( k_1 = \mu_L + c, k_2 = \mu_L + \varepsilon_L + c, k_3 = \mu_L + \alpha_L + c, k_4 = \mu_D + m_D, k_7 = \mu_D + \omega_D, k_8 = \mu_P + m_P, k_{11} = \mu_P + \omega_P \). If \( R_0 < 1 \), then DFE is locally asymptotically stable and if \( R_0 > 1 \), then DFE is unstable.

**Proof.** In the next generation matrix technique of Driessche-Watmough [9], we have six ‘infectious’ classes, \( E_L, I_L, E_D, I_D, E_P, I_P \) and

\[
F = \begin{pmatrix}
0 & \frac{\beta_{LL} \partial_L}{k_1} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & \frac{\beta_{DD} \partial_D}{k_7} & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{\beta_{DD} \partial_D}{k_7} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\end{pmatrix}, \quad \text{and } V = \begin{pmatrix}
k_2 & 0 & 0 & 0 & 0 & 0 \\
-\varepsilon_L & k_3 & 0 & 0 & 0 & 0 \\
k_0 & 0 & k_5 & 0 & 0 & 0 \\
0 & -\varepsilon_D & k_6 & 0 & 0 & 0 \\
0 & 0 & 0 & k_9 & 0 & 0 \\
0 & 0 & 0 & 0 & -\varepsilon_P & k_{10} \\
\end{pmatrix}
\]

where \( k_1 = \mu_L + c, k_2 = \mu_L + \varepsilon_L + c, k_3 = \mu_L + \alpha_L + c, k_4 = \mu_D + m_D, k_5 = \mu_D + \varepsilon_D + \partial_D, k_6 = \mu_P + \alpha_D + \varepsilon, k_7 = \mu_D + \omega_D, k_9 = \mu_P + \varepsilon_P + \partial_P, k_{10} = \mu_P + \alpha_P \). Then using \( R_0 = \rho(FV^{-1}) \) with \( \rho \) being the spectral radius, we obtain

\[
R_0 = \max \left\{ \frac{\beta_{DD} \partial_D k_7 \varepsilon_D}{(k_4 k_7 - m_D \omega_D) k_5 k_9}, \frac{\beta_{LL} \partial_L \varepsilon_L}{k_3 k_5 k_2} \right\}
\]

By Theorem 2 of Driessche-Watmough [9], we have the DFE of basic model (1) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

Furthermore, consider the domain \( \mathcal{D}_1 = \{(S_L, E_L, I_L, S_D, E_D, I_D, R_D, S_P, E_P, I_P, R_P) \in \mathcal{D}: S_L' \geq S_L, S_D' \geq S_D, S_P' \geq S_P \} \).

**Theorem 2** The DFE of model (1), is globally asymptotically stable in \( \mathcal{D}_1 \) whenever \( R_0 < 1 \).

**Proof.** The proof is based on using a comparison theorem. The equation of the infected components can be written in terms of

\[
\begin{pmatrix}
\dot{E}_L \\
\dot{I}_L \\
\dot{E}_D \\
\dot{I}_D \\
\dot{E}_P \\
\dot{I}_P \\
\end{pmatrix} = (F - V) \begin{pmatrix}
E_L \\
I_L \\
E_D \\
I_D \\
E_P \\
I_P \\
\end{pmatrix} - M_1 \dot{Q}_1 - M_2 \dot{Q}_2 - M_3 \dot{Q}_3,
\]

where \( M_1 = \frac{\partial_S k_1}{k_1} - S_L, M_2 = \frac{\partial_D k_7}{k_4 k_7 - m_D \omega_D} - S_D, \) and \( M_3 = \frac{\partial_P k_{11}}{k_9 k_{11} - m_P \omega_P} - S_P \).

The matrices \( F \) and \( V \) are given above and \( Q_1, Q_2 \) and \( Q_3 \) are the non-negative matrices given by

\[
Q_1 = \begin{pmatrix}
0 & \beta_{LL} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\end{pmatrix}, \quad Q_2 = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
\end{pmatrix}
\]
Using the fact that the eigenvalues of the matrix $F - V$ all have negative real parts, it follows that the linearized differential inequality system (3) is stable whenever $\mathcal{R}_0 < 1$. Consequently, $(E_L(t), I_L(t), E_D(t), I_D(t), E_P(t), I_P(t)) \to (0, 0, 0, 0, 0, 0)$ as $t \to \infty$. By standard comparison results [10]. Substituting $E_L = I_L = E_D = I_D = E_P = I_P = 0$ in the first, fourth, seventh, eighth and eleventh equation of the basic model (1) gives

\[
S_L(t) \to \frac{\partial L}{\partial L}, \quad S_D(t) \to \frac{\partial D}{\partial D}, \quad R_D = \frac{m_D}{k_{4k_7-m_D\omega_D}}, \quad S_P(t) \to \frac{\partial p_{k_1}}{\partial k_1}, \quad R_P(t) \to \frac{m_p}{k_{8k_{11}-m_p\omega_p}} \text{ as } t \to \infty.
\]

Thus,

\[
(S_L(t), E_L(t), I_L(t), S_D(t), E_D(t), I_D(t), R_D(t), S_P(t), E_P(t), I_P(t), R_P(t)) \to \left(0, 0, 0, 0, \frac{\partial p_{k_7}}{k_{4k_7-m_D\omega_D}}, 0, 0, \frac{\partial p_{k_1}}{k_{8k_{11}-m_p\omega_p}}, 0, 0, \frac{\partial p_{k_1}}{k_{8k_{11}-m_p\omega_p}} \right) \text{ as } t \to \infty
\]

for $\mathcal{R}_0 < 1$. Hence, the DFE is globally asymptotically stable if $\mathcal{R}_0 < 1$. ■

4 Stability Analysis for the Equilibria of the Wild Dogs Population

This section presents a mathematical model to describe the spread of rabies infection in wild dogs.

4.1 Basic Model

Consider the population of wild dogs. The population of wild dogs is divided into three subclasses: susceptible, exposed and infectious with sizes denoted by $S_L$, $E_L$ and $I_L$. The model is a system of three ordinary differential equations

\[
\begin{align*}
\dot{S}_L &= \partial L - \mu_L S_L - \beta_{LL} S_L I_L - c S_L \\
\dot{E}_L &= \beta_{LL} S_L I_L - \mu_L E_L - \varepsilon_L E_L - c E_L \\
\dot{I}_L &= \varepsilon_L E_L - \mu_L I_L - \alpha_L I_L - c I_L.
\end{align*}
\]

(4)
The model (4) will be analyzed in the biologically-feasible region as follows. We consider the region
\[ D^1 = \{ (S_L, E_L, I_L) \in \mathbb{R}_+^3 : S_L \geq 0, E_L \geq 0, I_L \geq 0 \}. \]
A solution of (4) that starts in \( D^1 \) can be shown to remain in \( D^1 \) for all \( t \geq 0 \). Thus \( D^1 \) is positively invariant and it is sufficient to consider a solution in \( D^1 \).

### 4.2 Stability Analysis of Disease Free Equilibrium

**Theorem 3.** The DFE of the model (4), \( (S_L^*, E_L^*, I_L^*) = \left( \frac{\partial L}{L+\mu_L+c}, 0, 0 \right) \), is locally asymptotically stable if \( R_0^1 < 1 \), where \( R_0^1 = \frac{\partial L \beta L \mu_L}{(c+\mu_L)(c+\mu_L+c\mu_L+c\alpha_L)} \).

**Proof.** From the basic model (4) we obtain the Jacobian matrix by evaluation at the DFE point
\[ J_1 = \begin{pmatrix} -\mu_L - c & 0 & -\beta L \mu_L \mu_L + c \\ 0 & -\varepsilon_L - \mu_L - c & \beta L \mu_L + c \\ 0 & \varepsilon_L & -\alpha_L - \mu_L - c \end{pmatrix} \]
with characteristic equation
\[ f(\lambda) = \begin{vmatrix} -\mu_L - c - \lambda & 0 & -\beta L \mu_L \mu_L + c \\ 0 & -\varepsilon_L - \mu_L - c - \lambda & \beta L \mu_L + c \\ 0 & \varepsilon_L & -\alpha_L - \mu_L - c - \lambda \end{vmatrix} = 0. \quad (5) \]
From (5) we infer that the eigenvalues are \( \lambda_1 = -(\mu_L + c) \) and the roots of the quadratic equation \( A\lambda^2 + B\lambda + C = 0 \), where \( A = 1 \), \( B = (\mu_L \varepsilon_L + \alpha_L \mu_L + 4\mu_L c + c e_L + \alpha_L c + 2c^2 + 2\mu_L^2) / \mu_L + c \), and \( C = (c^2 \alpha_L + 3\mu_L c + \mu_L^2 \alpha_L + e_L \mu_L^2 + \mu_L^3 + c^3 + c e_L \alpha_L + e_L c^2 - \partial L \beta L \varepsilon_L + 2\mu_L \varepsilon_L c + 2\mu_L \alpha_L + 3\mu_L c^2 + \mu_L \varepsilon_L \alpha_L) / \mu_L + c \). Furthermore \( A > 0, B > 0 \) and \( C \) can be positive or negative. We distinguish in several cases.

**Case 1.** If \( C < 0 \), then we obtain two real eigenvalues, negative and positive, so DFE is unstable.

**Case 2.** If \( C = 0 \), then we get two real eigenvalues, a negative one and zero, the stability of DFE is undecided.

**Case 3.** If \( C > 0 \) and \( B^2 - 4AC = 0 \), then we obtain two real eigenvalues, equal and negative, so DFE is asymptotically stable.

**Case 4.** If \( C > 0 \) and \( B^2 - 4AC > 0 \), then we obtain two complex conjugate eigenvalues with negative real part, so DFE is asymptotically stable.

**Case 5.** If \( B^2 - 4AC < 0 \), then we obtain two complex conjugate eigenvalues with negative real part, so DFE is asymptotically stable.

From the 5 cases, we can conclude that if \( C < 0 \) then DFE is unstable; if \( C = 0 \), then the stability of the DFE point is undecided; if \( C > 0 \) then the DFE is asymptotically stable, so stability of the DFE point depends only on the value of \( C \), where \( C > 0 \) if
\[ c^2 \alpha_L + 3 \mu_L c + \mu_L \alpha_L + \varepsilon_L \mu_L \alpha_L + \varepsilon_L \mu^2 + \mu^2 + c + c \varepsilon_L \alpha_L + \varepsilon_L c^2 - \partial_L \beta_{LL} \varepsilon_L + 2 \mu_L \varepsilon_L + 2 \mu_L \varepsilon_L \alpha_L + 3 \mu_L c^2 + \mu_L \varepsilon_L \alpha_L > 0, \] or
\[ c^2 \alpha_L + 3 \mu_L c + \mu_L \alpha_L + \varepsilon_L \mu_L \alpha_L + \mu_L^2 + \mu^2 + c + c \varepsilon_L \alpha_L + \varepsilon_L c^2 + 2 \mu_L \varepsilon_L c + 2 \mu_L \varepsilon_L \alpha_L > \partial_L \beta_{LL} \varepsilon_L, \] or
\[ \frac{\partial_L \beta_{LL} \varepsilon_L}{(c + \mu_L)(c + \mu_L + \varepsilon_L)(c + \mu_L + \alpha_L)} < 1. \]

Define basic reproduction number \( R^1_0 = \frac{\partial_L \beta_{LL} \varepsilon_L}{(c + \mu_L)(c + \mu_L + \varepsilon_L)(c + \mu_L + \alpha_L)} \), we get \( C > 0 \) if \( R^1_0 < 1 \). In other word, the DFE is asymptotically stable if \( R^1_0 < 1 \). This proves the Theorem.

We note that \( R^1_0 \) is equal to the second quantity in (2). Consider the domain
\[ D_1^1 = \{(S_L, E_L, I_L) \in \mathbb{R}_+^3: S_L^* \geq S_L \}. \]

**Theorem 4.** The DFE of model (4), is globally asymptotically stable in \( D_1^1 \) whenever \( R^1_0 < 1 \).

**Proof.** The proof is based on using a comparison theorem. The equation of the infected components can be written in terms of
\[ \begin{pmatrix} \dot{E}_L \\ \dot{I}_L \end{pmatrix} = (F - V) \begin{pmatrix} E_L \\ I_L \end{pmatrix} - MQ \begin{pmatrix} E_L \\ I_L \end{pmatrix}, \]
where \( M = \beta_{LL} \frac{\partial_L}{\mu_L + c} - \beta_{LU} S_L \), the matrices \( F, V, \) and \( Q \) are
\[ F = \begin{pmatrix} \beta_{LL} \mu_L + c & 0 \\ 0 & \beta_{LL} \mu_L + c \end{pmatrix}, \quad V = \begin{pmatrix} \mu_L + \varepsilon_L + c & 0 \\ -\varepsilon_L & \mu_L + \alpha_L + c \end{pmatrix}, \quad \text{and} \quad Q = \begin{pmatrix} 0 & 1 \\ 0 & 0 \end{pmatrix}. \]

Thus, since \( M \geq 0 \) for all \( t \geq 0 \) and all parameters are positive, it follows that
\[ \begin{pmatrix} \dot{E}_L \\ \dot{I}_L \end{pmatrix} \leq (F - V) \begin{pmatrix} E_L \\ I_L \end{pmatrix}. \] (6)

Using the fact that the eigenvalues of the matrix \( F - V \) all have negative real parts, it follows that the linearized differential inequality system (6) is stable whenever \( R^1_0 < 1 \). Consequently, \( (E_L(t), I_L(t)) \to (0,0) \) as \( t \to \infty \). By standard comparison results [10]. Substituting \( E_L = I_L = 0 \) in the first equation of the basic model (4) gives \( S_D(t) \to \frac{\partial_L}{\mu_L + c} \) as \( t \to \infty \). Thus, \( (S_L(t), E_L(t), I_L(t)) \to \left( \frac{\partial_L}{\mu_L + c}, 0,0 \right) \) as \( t \to \infty \) for \( R^1_0 < 1 \). Hence, the DFE is globally asymptotically stable if \( R^1_0 < 1 \). ■

### 4.3 Stability Analysis of Endemic Equilibrium (EE)

The endemic equilibrium of (4) is unique as given by
\[(S^*_L, E^*_L, I^*_L) = \left( \frac{\varepsilon_L \alpha_L + \varepsilon_L \mu_L + \varepsilon_L c + \mu_L \alpha_L + \mu_L^2 c + c \alpha_L + c^2}{\beta_{LL} L^3} \right) - \frac{1}{\varepsilon_L (\varepsilon_L + \mu_L + c) \beta_{LL}} (\mu_L^3 + c^3 + \varepsilon_L c^2 + 3 \mu_L^2 c + \varepsilon_L \mu_L^3 + \mu_L^3 \alpha_L - \partial \beta_{LL} \varepsilon_L + \mu_L \varepsilon_L \alpha_L + 2 \mu_L \varepsilon_L c + 2 \mu_L \alpha_L + c \varepsilon_L \alpha_L + c^2 \alpha_L), \right.
\]
\[\left. \frac{- \partial \beta_{LL} \varepsilon_L + \mu_L \varepsilon_L \alpha_L + 2 \mu_L \varepsilon_L c + 2 \mu_L \alpha_L + c \varepsilon_L \alpha_L + c^2 \alpha_L}{\beta_{LL} (\varepsilon_L \alpha_L + \varepsilon_L c + \mu_L \alpha_L + \mu_L^2 c + c \alpha_L + c^2)} \right) \]
\[ \begin{array}{c|cc}
\lambda^3 & A & C \\
\lambda^2 & B & D \\
\lambda^1 & b_1 & D \\
\lambda^0 & D & D
\end{array} \]

We have \( A, B, D > 0 \) and \( BC - AD > 0 \). It can be concluded that \( EE \) is locally asymptotically stable if \( R_0^1 > 1 \). ■

5 Stability Analysis for the Equilibria of the Domestic Dogs Population

This section presents a mathematical model to describe the spread of rabies infection in domestic dogs.

5.1 The basic model where the domestic dogs are in the DFE

Domestic dogs are considered. The population of domestic dogs is divided into four subclasses, susceptible, exposed, infectious, recovered with sizes denoted by \( S_D, E_D, I_D \) and \( R_D \), respectively.

The model is a system of four ordinary differential equations:

\[
\begin{align*}
\dot{S}_D &= \mu_D - (\mu_D + \beta_D D_D + m_D)S_D + \omega_D R_D + p \theta_D E_D \\
\dot{E}_D &= \beta_D D_D I_D - (\mu_D + \varepsilon_D + \theta_D)E_D \\
\dot{I}_D &= \varepsilon_D E_D - (\mu_D + \alpha_D + e)I_D \\
\dot{R}_D &= m_D S_D - (\mu_D + \omega_D)R_D + (1 - p) \theta_D E_D
\end{align*}
\]

One can show that the solutions of the system are non-negative for non-negative initial values, i.e the region

\[ \mathcal{D}^2 = \{ (S_D, E_D, I_D, R_D) \in \mathbb{R}^4 : S_D(0) \geq 0, E_D(0), I_D(0), R_D(0) \geq 0 \} \]

is positively invariant for (8).

5.2 Stability analysis of the Disease Free Equilibrium (DFE)

**Theorem 6.** The DFE of the model (8), \( (S_D^*, E_D^*, I_D^*, R_D^*) = \left( \frac{\partial \rho_D(\omega_D + \mu_D)}{\mu_D(\omega_D + \mu_D + m_D)}, 0, 0, \frac{\partial m_D}{\mu_D(\omega_D + \mu_D + m_D)} \right) \), is locally asymptotically stable if \( R_0^2 < 1 \), where

\[ R_0^2 = \frac{\mu_D \theta_D(\omega_D + \mu_D + m_D)}{\mu_D \theta_D(\omega_D + \mu_D + m_D)} \frac{\mu_D(\omega_D + \mu_D) + \beta_D D_D}{\mu_D(\omega_D + \mu_D + m_D)} \frac{\mu_D(\omega_D + \mu_D + m_D)}{\mu_D(\omega_D + \mu_D + m_D)} \frac{\mu_D(\omega_D + \mu_D + m_D)}{\mu_D(\omega_D + \mu_D + m_D)} \]

**Proof.** From the basic model (8) we obtain the characteristic equation of the Jacobian matrix by evaluation at the DFE point

\[
f(\lambda) = \begin{vmatrix}
-\mu_D - m_D - \lambda & p \theta_D & -\frac{\beta_D \theta_D(\omega_D + \mu_D)}{\mu_D(\omega_D + \mu_D + m_D)} & \omega_D \\
0 & -\varepsilon_D - \mu_D - \theta_D - \lambda & \frac{\beta_D \theta_D(\omega_D + \mu_D)}{\mu_D(\omega_D + \mu_D + m_D)} & 0 \\
0 & \varepsilon_D & -\alpha_D - \mu_D - e - \lambda & 0 \\
m_D & (1 - p) \theta_D & \omega_D - \mu_D - \lambda & 0
\end{vmatrix} = 0.
\]

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From (9) we obtain that two eigenvalues are negative. We just need to find the roots of the quadratic equation \( A\lambda^2 + B\lambda + C = 0 \) for the other eigenvalues, where
\[
A = \omega_d \mu_d + \mu_d^2 + \mu_d m_d,
\]
\[
B = \omega_d \mu_d \alpha_D + \vartheta_D \mu_d \omega_D + \mu_d^2 e + \vartheta_D \mu_d m_d + \mu_d \alpha_D m_d + \omega_d \mu_d e + \mu_d em_D + \omega_D \mu_d e_D + \vartheta_D \mu_d m_D + 2\omega_D \mu_d^2 + 2\mu_d^2 m_d + \mu_d^2 \alpha_D + 2\mu_d^3 + \mu_d^2 \vartheta_D,
\]
\[
C = \mu_d^4 + \omega_d \mu_d^2 \vartheta_D + \mu_d^2 m_d \vartheta_D + \mu_d^2 \vartheta_D + \mu_d^3 m_d + \mu_d^3 \vartheta_D + \mu_d^3 e + \mu_d^3 \alpha_D + \mu_d^3 m_d \alpha_D + \mu_d^3 \vartheta_D \alpha_D + \mu_d^3 \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + 
\]
Furthermore \( A > 0, B > 0 \) and \( C \) can be positive or negative. We distinguish in several cases.

**Case 1.** If \( C < 0, \) then we obtain two real eigenvalues, negative and positive, so DFE is unstable.

**Case 2.** If \( C = 0, \) then we get two real eigenvalues, a negative one and zero, the stability of DFE is undecided.

**Case 3.** If \( C > 0 \) and \( B^2 - 4AC = 0, \) then we obtain two real eigenvalues, equal and negative, so DFE is asymptotically stable.

**Case 4.** If \( C > 0 \) and \( B^2 - 4AC > 0, \) then we obtain two eigenvalues, real and negative, so the DFE is asymptotically stable.

**Case 5.** If \( B^2 - 4AC < 0, \) then we obtain two complex conjugate eigenvalues with negative real part, so DFE is asymptotically stable.

From the 5 cases, we can conclude that if \( C < 0 \) then DFE is unstable; if \( C = 0, \) then the stability of the DFE point is undecided; if \( C > 0 \) then the DFE is asymptotically stable, so the stability of the DFE point depends only on the value of \( C, \) where \( C > 0 \) if
\[
\mu_d^4 + \omega_d \mu_d^2 \vartheta_D + \mu_d^2 m_d \vartheta_D + \mu_d^2 \vartheta_D + \mu_d^3 m_d + \mu_d^3 \vartheta_D + \mu_d^3 e + \mu_d^3 \alpha_D + \mu_d^3 \vartheta_D + \mu_d^3 \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + \mu_d^3 \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D \vartheta_D m_d \alpha_D + 
\]
This proves the theorem.

We consider the domain \( D_1^2 = \{(S_D, E_D, I_D, R_D)\in\mathbb{R}_+^4 : S_D \geq S_D\}.\)

**Theorem 7.** The DFE of model (8), is globally asymptotically stable in \( D_1^2 \) whenever \( R_0^2 < 1.\)

**Proof.** The proof is based on using a comparison theorem. The equation of the infected components can be written in terms of
\[
\begin{pmatrix}
E_D \\
I_D
\end{pmatrix} = (F - V) \begin{pmatrix}
E_D \\
I_D
\end{pmatrix} - MQ \begin{pmatrix}
E_D \\
I_D
\end{pmatrix}.
\]
where \( M = \frac{\partial_p(\omega_p+\mu_p)}{\mu_p(\omega_p+\mu_p+m_p)} - S_D \), the matrices \( F, V \), and \( Q \) are \( F = \\
\begin{pmatrix}
\beta_{DD} - \frac{\partial_p(\omega_p+\mu_p)}{\mu_p(\omega_p+\mu_p+m_p)} & 0 \\
0 & 0
\end{pmatrix}, \ V = \begin{pmatrix}
\mu_D + \varepsilon_D + e & 0 \\
-\varepsilon_D & \mu_D + \alpha_D + e
\end{pmatrix}, \ Q = \begin{pmatrix}
0 & \beta_{DD}
\end{pmatrix}.
\]

Thus, since \( M \geq 0 \) for all \( t \geq 0 \) and all parameters are positive, it follows that

\[
\begin{pmatrix}
\dot{E}_D \\
\dot{I}_D
\end{pmatrix} \leq (F-V) \begin{pmatrix}
E_D \\
I_D
\end{pmatrix}, \tag{10}
\]

Using the fact that the eigenvalues of the matrix \( F-V \) all have negative real parts, it follows that the linearized differential inequality system (10) is stable whenever \( R_0 < 1 \). Consequently, \( (E_D(t), I_D(t)) \to (0,0) \) as \( t \to \infty \). By standard comparison results \([10]\), substituting \( E_D = I_D = 0 \) in the first and fourth equation of the basic model (8) gives \( S_D(t) \to \frac{\partial_p(\omega_p+\mu_p)}{\mu_p(\omega_p+\mu_p+m_p)} \) and \( R_D(t) \to \frac{\partial_pm_D}{\mu_D(\omega_D+\mu_D+m_D)} \) as \( t \to \infty \). Thus,

\[
(S_D(t), E_D(t), I_D(t), R_D(t)) \to \left( \frac{\partial_p(\omega_p+\mu_p)}{\mu_p(\omega_p+\mu_p+m_p)}, 0, \frac{\partial_pm_D}{\mu_D(\omega_D+\mu_D+m_D)} \right) \ 	ext{as} \ t \to \infty \ 	ext{for} \ R_0^2 < 1. \]

Hence, the DFE is globally asymptotically stable in \( D^2 \) if \( R_0^2 < 1 \).■

6 Conclusions

In the model described above, we present a rabies model in wild dogs, domestic dogs and human with the effect of vaccination and culling in dogs. Both vaccine types, pre-exposure prophylaxis and post-exposure prophylaxis applied in the model. For the big model we analyze the stability of the DFE point to get the basic reproduction point \( (R_0) \). By using the next generation matrix method and applying Theorem 2 of Van-Den Driessche and Watmough, we obtain the condition that states if \( R_0 < 1 \), the DFE point is locally asymptotically stable. Furthermore using standard result of the comparison theorem we proved that the DFE point is globally asymptotically stable if \( R_0 < 1 \). Which means that if \( R_0 < 1 \) would exist forever the healthy people, the healthy domestic dogs, the healthy wild dogs, the immune people, and the immune domestic dogs.

Then we split the big model into two smaller parts with wild dogs separately from domestic dogs. For model rabies in wild dogs, using linearization the model around the equilibrium point, we obtain the conditions; the DFE point of the rabies model in wild dogs is locally asymptotically stable if \( R_0 < 1 \) and furthermore we proved that the DFE of this model is globally asymptotically stable whenever \( R_0 < 1 \), which means that over the time passed, the disease will be extinct. We also analyze the stability of the endemic equilibrium (EE) of the model and proved that the EE point of the model is locally asymptotically stable if \( R_0 > 1 \), which means the disease will exist in a certain period of time if \( R_0 > 1 \). For the model in domestic
dogs, we proved the stability of the DFE, both locally asymptotically stability and globally asymptotically stability. The result states that if $R_o < 1$, the infected class will go to 0 as time goes on.

References


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