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 analyses, 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 encephalitis. 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, vaccination 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 model is a system of eleven ordinary differential equations. The model consists of: 

1. The domestic dog population. 
2. The wild dog population. 
3. The rabies population. 
4. The susceptible population. 
5. The exposed population. 
6. The infectious population. 
7. The vaccinated population. 
8. The dead population. 

These equations describe the dynamics of the populations over time.

The model is given by the following system of differential equations:

\[
\begin{align*}
\frac{dI}{dt} &= a_I \left( I - I_f \right) - a_1 I - a_2 I - a_3 I \\
\frac{dE}{dt} &= a_E \left( I - I_f \right) - a_1 E - a_2 E - a_3 E \\
\frac{dR}{dt} &= a_R \left( I - I_f \right) - a_1 R - a_2 R - a_3 R \\
\frac{dV}{dt} &= a_V \left( I - I_f \right) - a_1 V - a_2 V - a_3 V \\
\frac{dD}{dt} &= a_D \left( I - I_f \right) - a_1 D - a_2 D - a_3 D \\
\end{align*}
\]

We develop a compartmental model of rabies spread. The population of wild dogs is modeled using a system of ordinary differential equations. The model is given by:

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\frac{dD}{dt} &= a_D \left( I - I_f \right) - a_1 D - a_2 D - a_3 D \\
\end{align*}
\]
\[
\begin{align*}
\dot{R}_D &= m_D S_D - (\mu_D + \omega_D) R_D + (1 - p) \delta_D E_D \\
S_p &= \partial_p - (\mu_p + \beta_{DP} I_D + \beta_{LP} I_L + m_p) S_P + \omega_P R_P + q \delta_P E_P \\
E_p &= (\beta_{DP} I_D + \beta_{LP} I_L) S_P - (\mu_p + \epsilon_p + \partial_p) E_P \\
I_p &= \epsilon_p E_p - (\mu_p + \alpha_p) I_p \\
\dot{R}_p &= m_p S_P - (\mu_p + \omega_p) R_P + (1 - q) \delta_P E_P.
\end{align*}
\]

In the population of wild dogs, \( \partial_L \) 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, \( \epsilon_L \) 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, \( \partial_D \) denotes the birth rate of domestic dogs, \( \mu_D \) denotes the death rate of domestic dogs, \( \beta_{DP} \) and \( \beta_{LP} \) state the transmission coefficient between domestic dogs and the transmission coefficient between domestic dogs and wild dogs, respectively. Then, \( \epsilon_D \) 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, \( \theta_D \) 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 \( \partial_p \). 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 \( \epsilon_p \) is the latency rate in people, \( \partial_p \) 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:

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

### 3 Stability Analysis of Disease-Free Equilibrium

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

\[
\left(\frac{\theta_L}{k_1}, 0, 0, \frac{\partial_p \kappa_7}{k_4 \kappa_7 - m_D \omega_D}, 0, 0, \frac{m_p \theta_D}{k_4 \kappa_7 - m_D \omega_D}, \frac{\partial_p \kappa_11}{k_8 \kappa_11 - m_p \omega_p}, 0, 0, \frac{m_p \theta_p}{k_8 \kappa_11 - m_p \omega_p}\right)
\]

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

\[
R_0 = \max \left\{ \frac{\beta_{DD} \partial_D \kappa_7 \epsilon_D}{(k_4 \kappa_7 - m_D \omega_D) k_6 k_5}, \frac{\beta_{LL} \partial_L \epsilon_L}{k_1 k_3 k_2} \right\}
\]
where \( k_1 = \mu_L + \epsilon_L + c, k_2 = \mu_L + \alpha_L + c, k_3 = k_4 = \mu_D + \epsilon_D + \alpha_D + e, k_5 = k_6 = \mu_D + \omega_D, k_7 = k_8 = \mu_P + \omega_P. \) 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, \( E_L, I_L, I_D, I_P, E_D, E_P \) and

\[
F = \begin{pmatrix}
0 & \frac{\beta_{EL} \theta_L}{\epsilon_L} & 0 & 0 & 0 & 0 \\
0 & 0 & \frac{\beta_{IL} \theta_L}{k_1} & 0 & 0 & 0 \\
0 & \frac{\beta_{ID} \theta_D k_1}{k_1} & 0 & 0 & 0 & 0 \\
0 & \frac{\beta_{IP} \theta_P k_1}{k_2} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \beta_{DL} \theta_D & 0 & 0 \\
0 & 0 & 0 & 0 & \beta_{DP} \theta_P & 0
\end{pmatrix}, \quad \text{and} \quad \mathcal{V} = \begin{pmatrix}
k_2 & 0 & 0 & 0 & 0 & 0 \\
-\epsilon_L & k_3 & 0 & 0 & 0 & 0 \\
0 & 0 & k_5 & 0 & 0 & 0 \\
0 & 0 & -\epsilon_D & k_6 & 0 & 0 \\
0 & 0 & 0 & k_9 & 0 & 0 \\
0 & 0 & 0 & 0 & -\epsilon_P & k_{10}
\end{pmatrix},
\]

where \( k_1 = \mu_L + \epsilon_L + c, k_2 = \mu_L + \alpha_L + c, k_3 = \mu_L + \alpha_L + c, k_4 = \mu_D + \epsilon_D + \alpha_D + e, k_5 = \mu_D + \omega_D, k_6 = \mu_D + \omega_D, k_7 = \mu_D + \omega_D, k_9 = \mu_P + \epsilon_P + \alpha_P. \)

Then using \( R_0 = \rho(F^{-1}V) \) with \( \rho \) being the spectral radius, we obtain

\[
R_0 = \max \left\{ \frac{\beta_{DD} \omega_D k_7}{(k_4 k_7 - m_D \omega_D)k_6 k_8 k_9 k_3 k_2}, \frac{\beta_{LL} \omega_L k_7}{k_1 k_3 k_2} \right\}.
\]

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

**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

\[
\begin{pmatrix}
E_L \\
I_L \\
S_D \\
I_D \\
S_P \\
I_P
\end{pmatrix} = (F - V)
\begin{pmatrix}
E_L \\
I_L \\
E_D \\
I_D \\
E_P \\
I_P
\end{pmatrix} - M_1 Q_1
\begin{pmatrix}
E_L \\
I_L \\
E_D \\
I_D \\
E_P \\
I_P
\end{pmatrix} - M_2 Q_2
\begin{pmatrix}
E_L \\
I_L \\
E_D \\
I_D \\
E_P \\
I_P
\end{pmatrix} - M_3 Q_3
\begin{pmatrix}
E_L \\
I_L \\
E_D \\
I_D \\
E_P \\
I_P
\end{pmatrix},
\]

where \( M_1 = \frac{\delta_L}{k_1} - S_L, M_2 = \frac{\delta_D k_7}{k_4 k_7 - m_D \omega_D} - S_D, \) and \( M_3 = \frac{\delta_P k_{11}}{k_6 k_{11} - m_P \omega_P} - S_P. \)

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

\[
Q_1 = \begin{pmatrix}
0 & \beta_{LL} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix}, \quad Q_2 = \begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & \beta_{DL} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0
\end{pmatrix},
\]
Consider the population of wild dogs. The population of wild dogs is divided into subclasses: susceptible, exposed, infected, and recovered, with sizes denoted by $S$, $E$, $I$, and $R$, respectively. The model is a system of three ordinary differential equations:

\[ \frac{dS}{dt} = \gamma \cdot (I - S - I - E) \]
\[ \frac{dE}{dt} = \gamma S - \sigma E \]
\[ \frac{dI}{dt} = \sigma E - \beta (R + I) I \]
\[ \frac{dR}{dt} = \beta (R + I) I - \gamma R \]

These subequations are described by the system of differential equations:

\[ \begin{align*}
    \frac{dS}{dt} &= \gamma(1 - S) - \frac{\sigma E}{1 + \beta R} \\
    \frac{dE}{dt} &= \frac{\sigma E}{1 + \beta R} - \frac{\gamma E}{\gamma + \sigma} \\
    \frac{dI}{dt} &= \frac{\gamma E}{\gamma + \sigma} - \frac{\beta (R + I) I}{\beta + \gamma} \\
    \frac{dR}{dt} &= \frac{\beta (R + I) I}{\beta + \gamma} - \frac{\gamma R}{\gamma + \gamma} 
\end{align*} \]

This section presents a mathematical model to describe the spread of rabies.
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), \((S_L^*, E_L^*, I_L^*) = \left( \frac{\partial L}{\mu_L + c}, 0, 0 \right)\), is locally asymptotically stable if \( R_0^1 < 1 \), where \( R_0^1 = \frac{\beta - \frac{\partial L}{\mu_L + c}}{(c + \mu_L)(c + \mu_L + \epsilon_L)(c + \mu_L + \alpha_L)} \).

**Proof.** From the basic model (4) we obtain the Jacobian matrix by evaluation at the DFE point \( J_1 = \left( \begin{array}{ccc} -\mu_L - c & 0 & \frac{-\beta \partial L}{\mu_L + c} \\ 0 & -\epsilon_L - \mu_L - c & \frac{\beta \partial L}{\mu_L + c} \\ 0 & \epsilon_L & -\alpha_L - \mu_L - c \end{array} \right) \)

with characteristic equation

\[ f(\lambda) = \begin{vmatrix} -\mu_L - c - \lambda & 0 & \frac{-\beta \partial L}{\mu_L + c} \\ 0 & -\epsilon_L - \mu_L - c - \lambda & \frac{\beta \partial L}{\mu_L + c} \\ \epsilon_L & -\alpha_L - \mu_L - c - \lambda \end{vmatrix} = 0. \tag{5} \]

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 \( A = 1, B = (\mu_L \epsilon_L + \alpha_L \mu_L + 4\mu_L c + c \epsilon_L + \alpha_L c + 2c^2 + 2\mu_L^2)/\mu_L + c, \) and \( C = (c^2 \alpha_L + 3\mu_L^2 c + \mu_L^2 \alpha_L + c \epsilon_L \alpha_L + \epsilon_L c^2 - \partial L \beta \partial L \epsilon_L + 2 \mu_L \epsilon_L c + 2 \mu_L c \alpha_L + 3 \mu_L c^2 + \mu_L \epsilon_L \alpha_L)/\mu_L + c \). 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 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 stability of the DFE point depends only on the value of \( C \), where \( C > 0 \) if
4.3 Stability Analysis of Fundamental Equilibrium (E)

The endemic equilibrium of (4) is uniquely given by $E = (0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0...
Theorem 5. The EP point of the model (4) is locally asymptotically stable if

\[ \lambda > 0, \quad \text{and} \quad \Delta > 0. \]

which is equivalent to

\[ \Delta = \lambda - \left( \sum_{i=1}^{n} \frac{\alpha_i + \beta_i \gamma_i}{\gamma_i} \right) > 0. \]

It is physically meaningful if all components are non-negative, i.e.,

\[ \sum_{i=1}^{n} \frac{\alpha_i + \beta_i \gamma_i}{\gamma_i} > \lambda. \]
Stability analysis of the Disease-Free Equilibrium (DFE)

The basic model is a system of ordinary differential equations:

\[ \begin{align*}
&\dot{a}_f = a_f (a_r + a_d) - a_f a_w = a_f \\
&\dot{a}_f = a_f (a_r + a_d + a_d g) - a_f a_d = a_f \\
&\dot{a}_d = a_d (a_r + a_d + a_d g) - a_d a_f = a_d \\
&\dot{a}_w = a_w (a_r + a_d + a_d g) - a_w a_d = a_w \\
&\dot{a}_g = a_g (a_r + a_d + a_d g) - a_g a_d = a_g \\
&\dot{a}_h = a_h (a_r + a_d + a_d g) - a_h a_d = a_h \\
\end{align*} \]

The model is a system of four ordinary differential equations.

Domestic dogs are considered. The population of domestic dogs is divided into four compartments: susceptible, exposed, infected, and recovered, expressed by the subscripts s, e, i, and r, respectively.

The basic model is the following system of nonlinear difference equations:

\[ \begin{align*}
&\dot{a}_f = a_f (a_r + a_d) - a_f a_w = a_f \\
&\dot{a}_f = a_f (a_r + a_d + a_d g) - a_f a_d = a_f \\
&\dot{a}_d = a_d (a_r + a_d + a_d g) - a_d a_f = a_d \\
&\dot{a}_w = a_w (a_r + a_d + a_d g) - a_w a_d = a_w \\
&\dot{a}_g = a_g (a_r + a_d + a_d g) - a_g a_d = a_g \\
&\dot{a}_h = a_h (a_r + a_d + a_d g) - a_h a_d = a_h \\
\end{align*} \]

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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

\[
A = \omega D\mu D + \mu D^2 + \mu D m_D,
\]
\[
B = \omega D\mu D \alpha D + \theta D\mu D \omega D + \mu D^2 e + \theta D\mu D m_D + \mu D \alpha D m_D + \omega D \mu D e + \mu D e m_D + \omega D \mu D e \mu D + \epsilon D \mu D m_D + \mu D^2 m_D + \mu D^2 \alpha D + 2\mu D^3 + \mu D^2 e,
\]
\[
C = \mu D^4 + \omega D\mu D^2 e \epsilon D + \mu D^3 m_D \alpha D + \mu D^2 m_D e + \mu D^3 m_D + \mu D^3 \alpha D + \mu D^3 e + \mu D^2 a D + \omega D\mu D^2 \epsilon D + \mu D^2 m_D \alpha D + \omega D\mu D e + \omega D\mu D^2 e + \mu D^2 \epsilon D \alpha D + \omega D\mu D e \delta D + \mu D m_D \epsilon D + \mu D m_D e + \mu D m_D \alpha D + \mu D m_D \delta D - \omega D\delta D e \beta D + \omega D \delta D e + \omega D \mu D \delta D + \omega D \mu D \delta D e.
\]

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

\[
\mu D^4 + \omega D\mu D^2 e \epsilon D + \mu D^3 m_D \alpha D + \mu D^2 m_D e + \mu D^3 m_D + \mu D^3 e + \mu D^2 \alpha D + \mu D^2 \epsilon D + \omega D\mu D^3 e + \omega D\mu D^2 e + \omega D\mu D \alpha D + \omega D\mu D^2 e + \mu D^2 \epsilon D \alpha D + \mu D^2 m_D \alpha D + \mu D^2 m_D e + \mu D^2 \alpha D + \mu D m_D \epsilon D + \mu D m_D e + \mu D m_D \alpha D + \mu D m_D \delta D + \omega D \mu D e + \omega D \mu D \epsilon D + \mu D \mu D \epsilon D + \mu D m_D \epsilon D + \mu D m_D \alpha D + \mu D m_D \delta D + \omega D \mu D e + \omega D \mu D e + \omega D \mu D \epsilon D e + \omega D \mu D \epsilon D + \mu D \mu D \delta D = \omega D \mu D e \beta D + \mu D \mu D \epsilon D \beta D, \quad \text{or} \quad \mathcal{R}^2 = \frac{\mu D (\mu D + e + \mu D)(\mu D + \omega D + \mu D)(\mu D + \mu D + e)}{\mu D (\mu D + \omega D + \mu D)(\mu D + \omega D + \mu D + \mu D + e)} < 1.
\]

This proves the theorem.■

We consider the domain $\mathcal{D}^2 = \{(S_D, E_D, D_D, R_D) \in \mathbb{R}^4 \mid S_D \geq S_D \}$.

**Theorem 7.** The DFE of model (8), is globally asymptotically stable in $\mathcal{D}^2$ whenever $\mathcal{R}^2 < 1$.

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

\[
\begin{pmatrix}
E_D \\
D_D
\end{pmatrix} = (F - V) \begin{pmatrix}
E_D \\
D_D
\end{pmatrix} - M \begin{pmatrix}
E_D \\
D_D
\end{pmatrix}.
\]
Disease will exist in a certain period of time if \( R_0 < 1 \). For the model in domestic

the stability of the disease-free equilibrium (DFE) of the model is globally asymptotically stable if \( R_0 > 1 \), which means the disease will be extinct. We also analyze

the equilibrium point, we obtain the conditions for the DFE point. The DFE point of the model is globally asymptotically stable whenever \( R_0 > 1 \) and is given by

\[
\frac{\sigma}{\sigma (\alpha_1 + \alpha_2 + \sigma)} \begin{pmatrix} a_1 \\ a_2 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}
\]

Then we split the model into two smaller parts with each separately from

\[
\begin{align*}
\text{people, and the immune domestic drafts,}
\end{align*}
\]

\[
\begin{align*}
\text{the healthy people, the healthy domestic drafts, and the immune dominate drafts.}
\end{align*}
\]

\[
\begin{align*}
\text{asymptotically stable.}
\end{align*}
\]

\[
\begin{align*}
\text{NFEP, there exists}
\end{align*}
\]

\[
\begin{align*}
\text{standard result of the comparison theorem. We proved that the DFE point in globally}
\end{align*}
\]

\[
\begin{align*}
\text{equation, we prove that the DFE point is locally}
\end{align*}
\]

\[
\begin{align*}
\text{stability result (1) implies that the next generation matrix}
\end{align*}
\]

\[
\begin{align*}
\text{Bry using the next generation matrix method and applying the}
\end{align*}
\]

\[
\begin{align*}
\text{lypse, and we compare the basic reproductive number and the basic reproductive}
\end{align*}
\]

\[
\begin{align*}
\text{disease dominates model. For the basic model we apply the stability of the}
\end{align*}
\]

\[
\begin{align*}
\text{the DFE point to get the basic}
\end{align*}
\]

\[
\begin{align*}
\text{in the disease dominated model.}
\end{align*}
\]

\[
\begin{align*}
\text{we present a reduced model in wild, and disease, domestic}
\end{align*}
\]

\[
\begin{align*}
\text{In the model described above, we present a reduced model in wild, and disease, domestic}
\end{align*}
\]

\[
\begin{align*}
\text{Conclusions}
\end{align*}
\]

\[
\begin{align*}
\text{1.} \quad R_0 > 1. \quad \text{Hence, the DFE is globally asymptotically stable in}
\end{align*}
\]

\[
\begin{align*}
\text{as}
\end{align*}
\]

\[
\begin{align*}
\text{Thus, using the}\end{align*}
\]

\[
\begin{align*}
\text{the basic model (1) gives S}\begin{pmatrix} a_1 \\ a_2 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}
\end{align*}
\]

\[
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\begin{align*}
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\end{align*}
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dogs, we proved the stability of the DFE, both locally asymptotically stability and globally asymptotically stability. The result states that if $R_0 < 1$, the infected class will go to 0 as time goes on.

References


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