

STABILITY ANALYSIS OF A MATHEMATICAL MODEL OF RABIES SPREAD WITH VACCINATION IN HUMAN AND DOG POPULATIONS, INCLUDING AWARE AND UNAWARE EXPOSED SUBPOPULATIONS

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ABSTRACT

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Rabies is a zoonotic disease that causes progressive and fatal inflammation of the brain and spinal cord, which can be prevented by vaccination. This study aims to analyze the stability of a mathematical model of rabies disease spread with vaccination in human and dog populations in Maluku Province. The model uses a system of ordinary differential equations that separates the human population into six subpopulations (6 variables) and the dog population into three subpopulations (3 variables). The new variables are unaware subpopulations that we divide from aware subpopulations. The results showed that disease-free and endemic equilibrium points could be achieved, and the stability of these equilibrium points was analyzed using basic reproduction numbers (R_0). Both disease-free and endemic equilibrium points are locally asymptotically stable. The Numerical simulations were also conducted to determine the characteristics of each subpopulation. This study was to provide better insight into controlling the spread of rabies in Maluku Province and it can be used as a reference in developing mathematical models for other infectious diseases.



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1. INTRODUCTION

Rabies is an acute infectious disease that affects the central nervous system and is caused by the Lyssavirus. Once clinical symptoms appear, rabies is almost always fatal up to 100% [1]. The rabies virus can infect both domestic and wild animals and is spread through saliva by direct contact, such as bites, scratches, or contact with mucous membranes (eyes, mouth, or open wounds) [2]. Children between the ages of 5 and 14 are often victims due to their frequent interaction with animals [3][4]. Rabies virus belongs to the order Mononegavirales, a group of viruses with unsegmented negative-strand RNA genomes [5][6][7].

The initial symptoms of rabies generally appear 30-90 days after exposure to the virus from an infected animal. However, in some cases, symptoms may appear within weeks or even years [8]. Early symptoms of rabies include fever, weakness, loss of appetite, headache, chills, sore throat, diarrhea, anxiety, depression, insomnia, and muscle pain [9]. Rabies can also cause characteristic symptoms such as tingling, pain, and severe itching at the bite site [5].

Rabies occurs in more than 150 countries and territories worldwide and is exceptionally high in developing countries such as Africa and Asia [10]. Globally, 55,000 people die from rabies each year, although this figure is thought to be higher due to under-reporting in developing countries [11]. More than 40% of people bitten by rabies-infected animals are children, who have a higher propensity to play with animals [12]. More than 99% of human rabies deaths occur in developing countries, and despite effective and economical control efforts, the disease remains uncontrolled in most affected countries [1]. In the last 12 years, there have been 178 rabies deaths in Indonesia [13].

As of April 2023, there were 31,113 bite cases from rabies-infected animals in Indonesia, with 23,211 cases receiving rabies vaccination and 11 deaths [14]. In Indonesia, 26 provinces are rabies-endemic areas. However, only 11 provinces are rabies-free, including Riau Islands, Bangka Belitung, DKI Jakarta, Central Java, DI Yogyakarta, East Java, West Papua, Papua, South Papua, Central Papua, and Papua Mountains [14].

Maluku, one of the provinces in Eastern Indonesia, was historically rabies-free until 2003 when the first outbreak occurred in the provincial capital, Ambon Island [15]. Treatment of animal bite wounds in humans includes immediate wound washing with running water and soap for 15 minutes, followed by antiseptic treatment and immediate medical treatment to receive the Anti-Rabies Vaccine (VAR) and Anti-Rabies Serum (SAR) [2]. Prompt wound care effectively prevents the onset of rabies [9].

Rabies control efforts include mass vaccination of domestic animals, especially dogs, which are the primary source of human rabies cases [16]. Public awareness campaigns and community involvement are essential in rabies prevention and control strategies [17]. In addition, international collaboration and support are essential to eliminate rabies, especially in regions where the disease remains endemic [12]. In addition, this study develops the model from Eze et al., 2020 [20] and Ruan, 2017 [21]. New models in this study refer to the model from both articles, by developing exposed sub-populations into two variables or two sub-populations, such as the sub-population aware (E_{h1}) and the sub-population unaware (E_{h2}). Sub-population unaware is a new variable that we added to this system. The reason to add this sub-population of unaware, is because when a dog bites someone, sometimes they are not aware of the effect of being bitten.

The model developed in this study is novel in that it adds sub-populations (variables) of exposed individuals who are not aware and exposed individuals who are aware. The exposed individuals distinguish it from previous models. This model includes more complex transitions between compartments, such as from unaware exposed to aware exposed, which better reflects the reality of disease dynamics.

2. RESEARCH METHODS

This research uses a quantitative research approach because it involves numerical calculations and research data in numerical form. The data source of this research is secondary data from the Central Bureau of Statistics and the Maluku Provincial Health Office in 2021-2024. The research steps are as follows:

1. Identify the research problem.
2. Search for materials and literature related to the Rabies Mathematical Model.
3. Apply Mathematical Models to the spread of rabies.

4. Determine the equilibrium points of the Mathematical Model for rabies.
5. Analyze the stability at equilibrium points of the Mathematical Model for rabies.
6. Determine the primary reproduction number.
7. Simulate the model.

3. RESULTS AND DISCUSSION

3.1 Mathematical Model of Rabies Disease Spread

Research on rabies disease spread models in human and dog populations has been conducted by many researchers, including Kunwar et al., entitled *Mathematical Analysis of Rabies Transmission Dynamics in Nepal*. This study used a mathematical model for the dog population with four subpopulations (4 variables). It is assumed that susceptible dogs that receive treatment in the form of vaccination can be exposed if they encounter other infected dogs [18]. Furthermore, research by Chapwanya et al. with the title *Analysis and dynamically consistent nonstandard discretization for a rabies model in humans and dogs*. The study used the SEIV-SEI mathematical model by assuming that vaccinated individuals are vulnerable and exposed individuals, and vaccination aims to reduce the risk of rabies infection and prevent the virus from developing into an active infection [19].

Furthermore, a study by Eze et al. titled *Mathematical Modeling of Transmission Dynamics of Rabies Virus*. This study used the SEIR model for human rabies and the SEIV model for animal rabies [20]. Furthermore, in *Modeling the transmission dynamics and control of rabies in China*, Shigui Ruan used the SEIR-SEIR mathematical model [21]. Therefore, referring to the model from both articles, we developed and divided exposed sub-populations into two variables or two sub-populations, such as the sub-population aware (E_{h1}) and the sub-population unaware (E_{h2}). Sub-population unaware is a new variable that we add to this system. The reason to add this sub-population of unawareness is because when someone is bitten by a dog, sometimes they are not aware of the effect of being bitten.

The mathematical model of Rabies disease spread in human and dog populations consists of 9 subpopulation variables with the following assumptions:

1. Closed Population
2. Equal Birth and Death Rates: Human and dog birth rates are assumed to be equal to their respective natural death rates, ensuring the population size remains constant in the absence of disease.
3. Susceptible Subpopulation: Individuals (both humans and dogs) are born susceptible to rabies.
4. Exposed Individuals Subpopulation: Exposed humans are divided into two subpopulations: Unaware Exposed, i.e., individuals who do not realize they are exposed to rabies, and Aware Exposed, i.e., individuals who realize their exposure after receiving education on rabies.
5. Only individuals who are exposed and aware of the exposure receive vaccination.
6. Vulnerable Dogs receive vaccinations to prevent the spread of rabies.
7. The subpopulation of recovered individuals comprises individuals who received an entire course of anti-rabies vaccine (VAR) and anti-rabies serum (SAR) treatment.
8. The Infected Dog subpopulation does not recover or become susceptible again.
9. The model assumes homogeneous mixing in the population, meaning that everyone has an equal chance of interacting with other individuals.

Based on these assumptions, the spread of Rabies disease in human and dog populations is presented schematically in a compartment diagram, as shown in **Figure 1**.

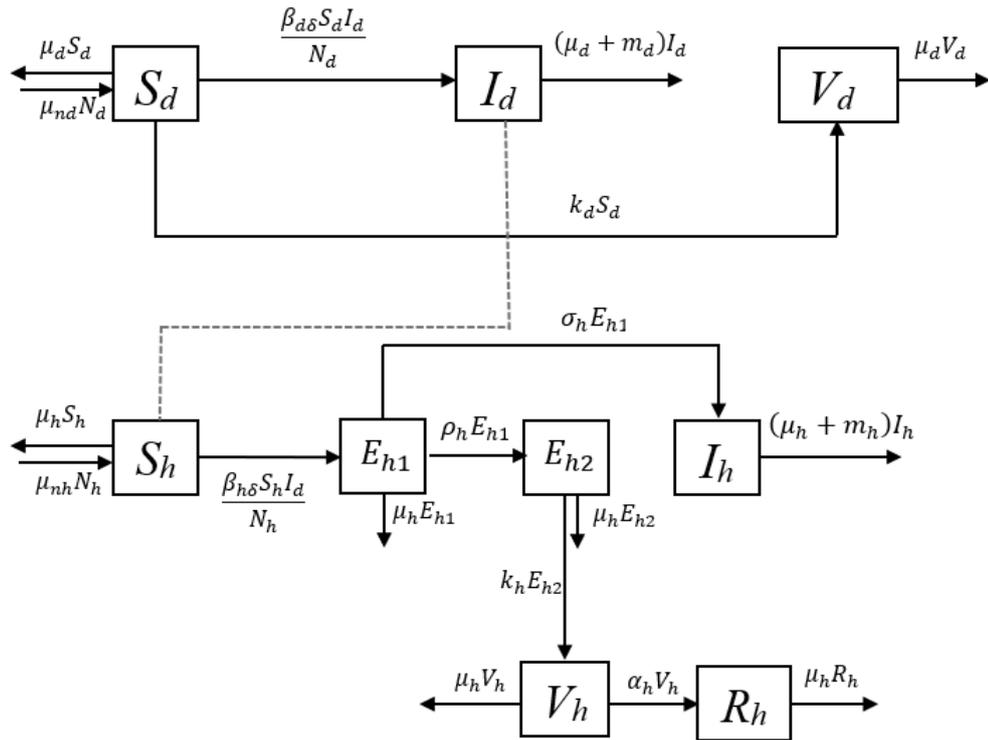


Figure 1. Compartmental Diagram of the Rabies Disease Spread Model

Based on the compartment diagram above, the mathematical model for the spread of Rabies disease in human and dog populations can be obtained by **Equation (1)** to **Equation (9)**.

$$\frac{dS_h}{dt} = \mu_{nh}N_h - \left(\frac{\beta_h\delta I_d}{N_h} + \mu_h\right)S_h \tag{1}$$

$$\frac{dE_{h1}}{dt} = \frac{\beta_h\delta S_h I_d}{N_h} - (\sigma_h + \rho_h + \mu_h)E_{h1} \tag{2}$$

$$\frac{dE_{h2}}{dt} = \rho_h E_{h1} - (k_h + \mu_h)E_{h2} \tag{3}$$

$$\frac{dI_h}{dt} = \sigma_h E_{h1} - (\mu_h + m_h)I_h \tag{4}$$

$$\frac{dV_h}{dt} = k_h E_{h2} - (\alpha_h + \mu_h)V_h \tag{5}$$

$$\frac{dR_h}{dt} = \alpha_h V_h - \mu_h R_h \tag{6}$$

$$\frac{dS_d}{dt} = \mu_{nd}N_d - \frac{\beta_{d\delta}S_d I_d}{N_d} - (k_d + \mu_d)S_d \tag{7}$$

$$\frac{dI_d}{dt} = \frac{\beta_{d\delta}S_d I_d}{N_d} - (\mu_d + m_d)I_d \tag{8}$$

$$\frac{dV_d}{dt} = k_d S_d - \mu_d V_d \tag{9}$$

A description of each variable and parameter is as follows:

- S_h : Subpopulation of infected susceptible individuals
- E_{h1} : Subpopulation of unaware exposed individuals
- E_{h2} : Subpopulation of exposed individuals who are aware due to education
- I_h : Subpopulation of infected individuals
- V_h : Subpopulation of vaccinated individuals

- R_h : Subpopulation of recovered individuals
 S_d : Subpopulations of infected susceptible dogs
 I_d : Subpopulation of infected dogs
 V_d : Subpopulation of vaccinated dogs
 μ_{nh} : Natural birth rate of individuals
 μ_h : Natural mortality rate of individuals
 β_h : Chance of disease spread from an infected dog to a susceptible individual
 $\beta_{h\delta}$: Contact rate of infected dogs with susceptible individuals
 σ_h : The transfer rate from exposure to infected individuals is characterized by the onset of symptoms.
 ρ_h : Educational Effectiveness
 k_h : Vaccination rate in individuals
 m_h : Individual death rate due to rabies infection
 α_h : Individual recovery rate after vaccination
 μ_{nd} : Natural birth rate of dogs
 μ_d : Natural mortality rate of dogs
 β_d : Chance of disease spread from infected dog to susceptible dog
 $\beta_{d\delta}$: Contact rate of infected dogs with susceptible dogs
 k_d : Vaccination rate in dogs
 m_d : Death rate of dogs infected with rabies

3.2 Equilibrium Point Analysis

The equilibrium point can be obtained by stating the initial value $\frac{dS_h}{dt} = 0, \frac{dE_{h1}}{dt} = 0, \frac{dE_{h2}}{dt} = 0, \frac{dI_h}{dt} = 0, \frac{dV_h}{dt} = 0, \frac{dR_h}{dt} = 0, \frac{dS_d}{dt} = 0, \frac{dI_d}{dt} = 0, \frac{dV_d}{dt} = 0$. There are two equilibrium points, namely, the disease-free equilibrium point and the endemic equilibrium point.

3.2.1 Disease-Free Equilibrium Point

A disease-free equilibrium point is one where the population has no more disease. If $I_d = 0$, then no infected dog can transmit the disease. If $I_h = 0$, then all individuals enter the population of susceptible individuals, and there are no infected individuals.

Thus, the rabies disease spread model's disease-free equilibrium points are obtained:

$$E_1(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) = \left(\frac{\mu_{nh}N_h}{\mu_h}, 0, 0, 0, 0, 0, \frac{\mu_{nd}N_d}{(k_d + \mu_d)}, 0, \frac{k_d\mu_{nd}N_d}{\mu_d(k_d + \mu_d)} \right)$$

3.2.2 Endemic Equilibrium Point

The disease equilibrium point is affected by the population ($I \neq 0$). If $I_d \neq 0$, there are infected dogs that can transmit the disease and cause endemism. If $I_h \neq 0$, there are infected individuals. The endemic equilibrium point in the mathematical model system for the spread of rabies is obtained: $E_2(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) = (S_h^*, E_{h1}^*, E_{h2}^*, I_h^*, V_h^*, R_h^*, S_d^*, I_d^*, V_d^*)$ for

$$S_h^* = \frac{\beta_{d\delta}\mu_{nh}N_h^2(\mu_d + m_d)}{\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_d\delta\mu_hN_h)(\mu_d + m_d)}$$

$$\begin{aligned}
&= \frac{\beta_{d\delta}\mu_{nh}N_h^2(\mu_d + m_d)}{c} \\
E_{h1}^* &= \frac{\beta_{h\delta}\mu_{nh}N_hN_d(\beta_{d\delta}\mu_1 - (k_d + \mu_d)(\mu_d + m_d))}{(\sigma_h + \rho_h + \mu_h)(\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d))} \\
&= \frac{fe}{(\sigma_h + \rho_h + \mu_h)c} \\
E_{h2}^* &= \frac{\rho_h\beta_{h\delta}\mu_{nh}N_hN_d(\beta_{d\delta}\mu_1 - (k_d + \mu_d)(\mu_d + m_d))}{(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)(\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d))} \\
&= \frac{\rho_hfe}{(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)c} \\
I_h^* &= \frac{\sigma_h\beta_{h\delta}\mu_{nh}N_hN_d(\beta_{d\delta}\mu_1 - (k_d + \mu_d)(\mu_d + m_d))}{(\mu_h + m_h)(\sigma_h + \rho_h + \mu_h)(\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d))} \\
&= \frac{\sigma_hfe}{(\mu_h + m_h)(\sigma_h + \rho_h + \mu_h)c} \\
V_h^* &= \frac{k_h\rho_h\beta_{h\delta}\mu_{nh}N_hN_d(\beta_{d\delta}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d))}{(\alpha_h + \mu_h)(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)(\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d))} \\
&= \frac{k_h\rho_hfe}{(\alpha_h + \mu_h)(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)c} \\
R_h^* &= \frac{\alpha_hk_h\rho_h\beta_{h\delta}\mu_{nh}N_hN_d(\beta_{d\delta}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d))}{\mu_h(\alpha_h + \mu_h)(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)(\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d))} \\
&= \frac{\alpha_hk_h\rho_hfe}{\mu_h(\alpha_h + \mu_h)(k_h + \mu_h)(\sigma_h + \rho_h + \mu_h)c} \\
S_d^* &= \frac{(\mu_d + m_d)N_d}{\beta_{d\delta}} \\
I_d^* &= \frac{N_d(\beta_{d\delta}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d))}{\beta_{d\delta}(\mu_d + m_d)} = \frac{N_d e}{\beta_{d\delta}(\mu_d + m_d)} \\
V_d^* &= \frac{k_dN_d(\mu_d + m_d)}{\beta_{d\delta}\mu_d}
\end{aligned}$$

by stating

$$\begin{aligned}
c &= (\beta_{h\delta}\beta_{d\delta}\mu_{nd}N_d - (\beta_{h\delta}N_d(k_d + \mu_d) - \beta_{d\delta}\mu_hN_h)(\mu_d + m_d)) \\
e &= \beta_{d\delta}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d) \\
f &= \beta_{h\delta}\mu_{nh}N_hN_d
\end{aligned}$$

3.2.3 Basic Reproduction Number

If $R_0 < 1$, the disease will disappear from the population. If $R_0 = 1$, the disease will persist with a stable number of cases without causing an outbreak. If $R_0 > 1$, the disease will increase to an outbreak, to determine the basic reproduction number (R_0), disease-free equilibrium point is used. The compartments used in this case are for humans, compartment E_{h1} , E_{h2} , I_h while in dogs the compartment is used I_d . By using the following generation matrix, the following basic reproductive numbers are obtained:

$$R_0 = \frac{\beta_{d\delta}\mu_{nd}}{(k_d + \mu_d)(\mu_d + m_d)} \quad (10)$$

Thus, the rate of contact of susceptible dogs with infected dogs, natural births of dogs, anti-rabies vaccination of dogs, and deaths of dogs due to rabies or natural death significantly affect the rate of rabies transmission. The greater the vaccination and mortality rates of infected and naturally born dogs, the lower the chance of rabies spreading in a population. Meanwhile, the higher the rate of contact between infected and susceptible dogs and the rate of dog births, the greater the chance of rabies spread.

3.3 Stability Point Analysis

A stability analysis was conducted to determine the rate of change in the rabies disease spread model. It can be written from **Equation (1)** to **Equation (9)**.

$$\begin{aligned}
 f_1(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \mu_{nh}N_h - \left(\frac{\beta_{h\delta}I_d}{N_h} + \mu_h\right)S_h \\
 f_2(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \frac{\beta_{h\delta}S_hI_d}{N_h} - (\sigma_h + \rho_h + \mu_h)E_{h1} \\
 f_3(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \rho_hE_{h1} - (k_h + \mu_h)E_{h2} \\
 f_4(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \sigma_hE_{h1} - (\mu_h + m_h)I_h \\
 f_5(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= k_hE_{h2} - (\alpha_h + \mu_h)V_h \\
 f_6(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \alpha_hV_h - \mu_hR_h \\
 f_7(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \mu_{nd}N_d - \frac{\beta_{d\delta}S_dI_d}{N_d} - (k_d + \mu_d)S_d \\
 f_8(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= \frac{\beta_{d\delta}S_dI_d}{N_d} - (\mu_d + m_d)I_d \\
 f_9(S_h, E_{h1}, E_{h2}, I_h, V_h, R_h, S_d, I_d, V_d) &= k_dS_d - \mu_dV_d
 \end{aligned}$$

Furthermore, the following Jacobian matrix with order 9 rows and 9 columns, based on the number of variables is formed:

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S_h} & \frac{\partial f_1}{\partial E_{h1}} & \frac{\partial f_1}{\partial E_{h2}} & \frac{\partial f_1}{\partial I_h} & \frac{\partial f_1}{\partial V_h} & \frac{\partial f_1}{\partial R_h} & \frac{\partial f_1}{\partial S_d} & \frac{\partial f_1}{\partial I_d} & \frac{\partial f_1}{\partial V_d} \\ \frac{\partial f_2}{\partial S_h} & \frac{\partial f_2}{\partial E_{h1}} & \frac{\partial f_2}{\partial E_{h2}} & \frac{\partial f_2}{\partial I_h} & \frac{\partial f_2}{\partial V_h} & \frac{\partial f_2}{\partial R_h} & \frac{\partial f_2}{\partial S_d} & \frac{\partial f_2}{\partial I_d} & \frac{\partial f_2}{\partial V_d} \\ \frac{\partial f_3}{\partial S_h} & \frac{\partial f_3}{\partial E_{h1}} & \frac{\partial f_3}{\partial E_{h2}} & \frac{\partial f_3}{\partial I_h} & \frac{\partial f_3}{\partial V_h} & \frac{\partial f_3}{\partial R_h} & \frac{\partial f_3}{\partial S_d} & \frac{\partial f_3}{\partial I_d} & \frac{\partial f_3}{\partial V_d} \\ \frac{\partial f_4}{\partial S_h} & \frac{\partial f_4}{\partial E_{h1}} & \frac{\partial f_4}{\partial E_{h2}} & \frac{\partial f_4}{\partial I_h} & \frac{\partial f_4}{\partial V_h} & \frac{\partial f_4}{\partial R_h} & \frac{\partial f_4}{\partial S_d} & \frac{\partial f_4}{\partial I_d} & \frac{\partial f_4}{\partial V_d} \\ \frac{\partial f_5}{\partial S_h} & \frac{\partial f_5}{\partial E_{h1}} & \frac{\partial f_5}{\partial E_{h2}} & \frac{\partial f_5}{\partial I_h} & \frac{\partial f_5}{\partial V_h} & \frac{\partial f_5}{\partial R_h} & \frac{\partial f_5}{\partial S_d} & \frac{\partial f_5}{\partial I_d} & \frac{\partial f_5}{\partial V_d} \\ \frac{\partial f_6}{\partial S_h} & \frac{\partial f_6}{\partial E_{h1}} & \frac{\partial f_6}{\partial E_{h2}} & \frac{\partial f_6}{\partial I_h} & \frac{\partial f_6}{\partial V_h} & \frac{\partial f_6}{\partial R_h} & \frac{\partial f_6}{\partial S_d} & \frac{\partial f_6}{\partial I_d} & \frac{\partial f_6}{\partial V_d} \\ \frac{\partial f_7}{\partial S_h} & \frac{\partial f_7}{\partial E_{h1}} & \frac{\partial f_7}{\partial E_{h2}} & \frac{\partial f_7}{\partial I_h} & \frac{\partial f_7}{\partial V_h} & \frac{\partial f_7}{\partial R_h} & \frac{\partial f_7}{\partial S_d} & \frac{\partial f_7}{\partial I_d} & \frac{\partial f_7}{\partial V_d} \\ \frac{\partial f_8}{\partial S_h} & \frac{\partial f_8}{\partial E_{h1}} & \frac{\partial f_8}{\partial E_{h2}} & \frac{\partial f_8}{\partial I_h} & \frac{\partial f_8}{\partial V_h} & \frac{\partial f_8}{\partial R_h} & \frac{\partial f_8}{\partial S_d} & \frac{\partial f_8}{\partial I_d} & \frac{\partial f_8}{\partial V_d} \\ \frac{\partial f_9}{\partial S_h} & \frac{\partial f_9}{\partial E_{h1}} & \frac{\partial f_9}{\partial E_{h2}} & \frac{\partial f_9}{\partial I_h} & \frac{\partial f_9}{\partial V_h} & \frac{\partial f_9}{\partial R_h} & \frac{\partial f_9}{\partial S_d} & \frac{\partial f_9}{\partial I_d} & \frac{\partial f_9}{\partial V_d} \end{pmatrix}$$

$$J = \begin{pmatrix} -\left(\frac{\beta_{h\delta}I_d}{N_h} + \mu_h\right) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_{h\delta}S_h}{N_h} & 0 \\ \frac{\beta_{h\delta}I_d}{N_h} & -(\sigma_h + \rho_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{h\delta}S_h}{N_h} & 0 \\ 0 & \rho_h & -(k_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & -(\mu_h + m_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & -(\alpha_h + \mu_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\left(\frac{\beta_{d\delta}I_d}{N_d} + k_d + \mu_d\right) & -\frac{\beta_{d\delta}S_d}{N_d} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{d\delta}I_d}{N_d} & \frac{\beta_{d\delta}S_d}{N_d} - (\mu_d + m_d) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & 0 & -\mu_d \end{pmatrix}$$

3.3.1 Disease-Free Stability Point Analysis

To analyze the disease-free stability point, then substitute the disease-free equilibrium point (E_1) to the Jacobian matrix, it is obtained:

$$J(E_1) = \begin{bmatrix} -\left(\frac{\beta_{hs}0}{N_h} + \mu_h\right) & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_h\delta}{N_h}\left(\frac{\mu_{nh}N_h}{\mu_h}\right) & 0 \\ \frac{\beta_{hs}0}{N_h} & -(\sigma_h + \rho_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{hs}}{N_h}\left(\frac{\mu_{nh}N_h}{\mu_h}\right) & 0 \\ 0 & \rho_h & -(k_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & -(\mu_h + m_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & -(\alpha_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\left(\frac{\beta_{as}0}{N_d} + k_d + \mu_d\right) & -\frac{\beta_{as}}{N_d}\left(\frac{\mu_{nd}N_d}{k_d + \mu_d}\right) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{as}0}{N_d} & \frac{\beta_{as}}{N_d}\left(\frac{\mu_{nd}N_d}{k_d + \mu_d}\right) - (\mu_d + m_d) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & -\mu_d \end{bmatrix}$$

$$(E_1) = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_{hs}\mu_{nh}}{\mu_h} & 0 \\ 0 & -(\sigma_h + \rho_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{hs}\mu_{nh}}{\mu_h} & 0 \\ 0 & \rho_h & -(k_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & -(\mu_h + m_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & -(\alpha_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(k_d + \mu_d) & -\frac{\beta_{as}\mu_{nd}}{k_d + \mu_d} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{as}\mu_{nd}}{k_d + \mu_d} - (\mu_d + m_d) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & -\mu_d \end{bmatrix}$$

Assume

$$J(E_1) = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & a_1 & 0 \\ 0 & a_2 & 0 & 0 & 0 & 0 & 0 & a_3 & 0 \\ 0 & \rho_h & a_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & a_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & a_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_7 & a_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & -\mu_d \end{bmatrix}$$

With

$$\begin{aligned}
 a_1 &= -\frac{\beta_{hs}\mu_{nh}}{\mu_h} \\
 a_2 &= -(\sigma_h + \rho_h + \mu_h) \\
 a_3 &= \frac{\beta_{hs}\mu_{nh}}{\mu_h} \\
 a_4 &= -(k_h + \mu_h) \\
 a_5 &= -(\mu_h + m_h) \\
 a_6 &= -(\alpha_h + \mu_h) \\
 a_7 &= -(k_d + \mu_d) \\
 a_8 &= -\frac{\beta_{as}\mu_{nd}}{k_d + \mu_d} \\
 a_9 &= \frac{\beta_{as}\mu_{nd}}{k_d + \mu_d} - (\mu_d + m_d)
 \end{aligned}$$

Furthermore, the eigenvalue can be determined using $\det(\lambda I - J(E_1)) = 0$ to get the characteristic equation. It is obtained:

$$\det(\lambda I - J(E_1)) = 0$$

$$\det \begin{pmatrix} \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \lambda \end{pmatrix} - \begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & a_1 & 0 \\ 0 & a_2 & 0 & 0 & 0 & 0 & 0 & a_3 & 0 \\ 0 & \rho_h & a_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & a_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & a_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_7 & a_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & k_d & -\mu_d \end{pmatrix} = 0$$

$$\det \begin{pmatrix} \lambda + \mu_h & 0 & 0 & 0 & 0 & 0 & 0 & a_1 & 0 \\ 0 & \lambda - a_2 & 0 & 0 & 0 & 0 & 0 & a_3 & 0 \\ 0 & \rho_h & \lambda - a_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & \lambda - a_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & \lambda - a_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & \lambda + \mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda - a_7 & a_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \lambda - a_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & \lambda + \mu_d \end{pmatrix} = 0$$

by using the cofactor rule, it is obtained

$$(\lambda + \mu_h)(\lambda - a_5)(\lambda + \mu_h)(\lambda - a_6)(\lambda - a_4)(\lambda - a_2)(\lambda + \mu_d)(\lambda - a_7)(\lambda - a_9) = 0$$

The value obtained by $\lambda_1 - \lambda_8$ is negative, but based on the stability conditions, the disease-free equilibrium point will be stable if all eigenvalues obtained are negative. For λ_9 is negative, then $\lambda_9 < 0$

$$\frac{\beta_a \delta \mu_{nd}}{k_d + \mu_d} - (\mu_d + m_d) < 0$$

Because λ_9 obtained is positive, then λ_9 depends on R_0 . The disease-free equilibrium point is stable if $R_0 < 1$, thus:

$$\frac{\beta_a \delta \mu_{nd}}{k_d + \mu_d} - (\mu_d + m_d) < 0$$

$$\frac{\beta_a \delta \mu_{nd}}{k_d + \mu_d} < (\mu_d + m_d)$$

$$\frac{\beta_a \delta \mu_{nd}}{(k_d + \mu_d)(\mu_d + m_d)} < 1$$

$$R_0 < 1$$

Then, the disease-free equilibrium point is locally asymptotically stable.

3.3.2 Endemic Stability Point Analysis

Substitute the endemic equilibrium point into the Jacobian matrix to obtain:

$$J(E_2) = \begin{pmatrix} -\left(\frac{\beta_{hs}}{N_h} \left(\frac{N_d e}{\beta_{ds}(\mu_d + m_d)}\right) + \mu_h\right) & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_{hs}}{N_h} \left(\frac{\beta_{ds} \mu_{nh} N_h^2 (\mu_d + m_d)}{c}\right) & 0 \\ \frac{\beta_{hs}}{N_h} \left(\frac{N_d e}{\beta_{ds}(\mu_d + m_d)}\right) & -(\sigma_h + \rho_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{hs}}{N_h} \left(\frac{\beta_{ds} \mu_{nh} N_h^2 (\mu_d + m_d)}{c}\right) & 0 \\ 0 & \rho_h & -(k_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & -(\mu_h + m_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & -(\alpha_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\left(\frac{\beta_{ds}}{N_d} \left(\frac{N_d e}{\beta_{ds}(\mu_d + m_d)}\right) + k_d + \mu_d\right) & -\frac{\beta_{ds}}{N_d} \left(\frac{(\mu_d + m_d) N_d}{\beta_{ds}}\right) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_{ds}}{N_d} \left(\frac{N_d e}{\beta_{ds}(\mu_d + m_d)}\right) & \frac{\beta_{ds}}{N_d} \left(\frac{(\mu_d + m_d) N_d}{\beta_{ds}}\right) - (\mu_d + m_d) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & -\mu_d \end{pmatrix}$$

$$J(E_2) = \begin{bmatrix} -\left(\frac{\beta_h \delta N_d e}{N_h \beta_{d\delta}(\mu_d + m_d)} + \mu_h\right) & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_h \delta (\beta_{d\delta} \mu_{nh} N_h^2 (\mu_d + m_d))}{N_h c} & 0 \\ \frac{\beta_h \delta N_d e}{N_h \beta_{d\delta}(\mu_d + m_d)} & -(\sigma_h + \rho_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & \frac{\beta_h \delta (\beta_{d\delta} \mu_{nh} N_h^2 (\mu_d + m_d))}{N_h c} & 0 \\ 0 & \rho_h & -(k_h + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_h & 0 & -(\mu_h + m_h) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_h & 0 & -(\alpha_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\left(\frac{e}{(\mu_d + m_d)} + k_d + \mu_d\right) & -(\mu_d + m_d) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{e}{(\mu_d + m_d)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_d & 0 & -\mu_d \end{bmatrix}$$

Assume

$$J(E_2) = \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 & 0 & 0 & a_2 & 0 \\ a_3 & a_4 & 0 & 0 & 0 & 0 & 0 & a_5 & 0 \\ 0 & a_6 & a_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_8 & 0 & a_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & 0 & a_{11} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & a_{12} & a_{13} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{14} & a_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{16} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{17} & 0 & a_{18} \end{bmatrix}$$

with

$$\begin{aligned} a_1 &= -\left(\frac{\beta_h \delta N_d e}{N_h \beta_{d\delta}(\mu_d + m_d)} + \mu_h\right) \\ a_2 &= -\frac{\beta_h \delta (\beta_{d\delta} \mu_{nh} N_h^2 (\mu_d + m_d))}{N_h c} \\ a_3 &= \frac{\beta_h \delta N_d e}{N_h \beta_{d\delta}(\mu_d + m_d)} \\ a_4 &= -(\sigma_h + \rho_h + \mu_h) \\ a_5 &= \frac{\beta_h \delta (\beta_{d\delta} \mu_{nh} N_h^2 (\mu_d + m_d))}{N_h c} \\ a_6 &= \rho_h \\ a_7 &= -(k_h + \mu_h) \\ a_8 &= \sigma_h \\ a_9 &= -(\mu_h + m_h) \\ a_{10} &= k_h \\ a_{11} &= -(\alpha_h + \mu_h) \\ a_{12} &= \alpha_h \\ a_{13} &= -\mu_h \\ a_{14} &= -\left(\frac{e}{(\mu_d + m_d)} + k_d + \mu_d\right) \\ a_{15} &= -(\mu_d + m_d) \\ a_{16} &= \frac{e}{(\mu_d + m_d)} \\ a_{17} &= k_d \\ a_{18} &= -\mu_d \end{aligned}$$

Furthermore, the eigenvalue can be determined by using $\det(\lambda I - J(E_2)) = 0$ to get the characteristic equation. It is obtained:

$$\det \begin{pmatrix} \begin{bmatrix} \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \lambda \end{bmatrix} - \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 & 0 & 0 & a_2 & 0 \\ a_3 & a_4 & 0 & 0 & 0 & 0 & 0 & a_5 & 0 \\ 0 & a_6 & a_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_8 & 0 & a_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & 0 & a_{11} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & a_{12} & a_{13} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{14} & a_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{16} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{17} & 0 & a_{18} \end{bmatrix} \end{pmatrix} = 0$$

$$\det \begin{bmatrix} \lambda - a_1 & 0 & 0 & 0 & 0 & 0 & 0 & a_2 & 0 \\ a_3 & \lambda - a_4 & 0 & 0 & 0 & 0 & 0 & a_5 & 0 \\ 0 & a_6 & \lambda - a_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_8 & 0 & \lambda - a_9 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_{10} & 0 & \lambda - a_{11} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & a_{12} & \lambda - a_{13} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda - a_{14} & a_{15} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{16} & \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{17} & 0 & \lambda - a_{18} \end{bmatrix} = 0$$

By using the cofactor rule, the characteristic equation is obtained:

$$(\lambda - a_{18})(\lambda - a_9)(\lambda - a_{13})(\lambda - a_{11})(\lambda - a_7)(\lambda - a_4)(\lambda - a_1)((\lambda - a_{14})(\lambda) - a_{16}a_{15}) = 0$$

$$(\lambda - a_{18})(\lambda - a_9)(\lambda - a_{13})(\lambda - a_{11})(\lambda - a_7)(\lambda - a_4)(\lambda - a_1)(\lambda^2 - a_{14}\lambda - a_{15}a_{16}) = 0$$

To determine the stability analysis of the endemic equilibrium point, then $R_0 > 1$.

The value obtained by $\lambda_1 - \lambda_7$ is negative. For $\lambda_{8,9}$ a quadratic equation is obtained:

$$\lambda^2 - a_{14}\lambda - a_{15}a_{16} = 0$$

$$\lambda^2 - \left(-\left(\frac{e}{(\mu_d+m_d)} + k_d + \mu_d \right) \right) - (-\mu_d + m_d) \left(\frac{e}{(\mu_d+m_d)} \right) = 0$$

$$\lambda^2 + \left(\frac{\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d)}{(\mu_d+m_d)} + k_d + \mu_d \right) \lambda + (\mu_d + m_d) \left(\frac{\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d)}{(\mu_d+m_d)} \right) = 0$$

$$\lambda^2 + \left(\frac{\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d) + (k_d + \mu_d)(\mu_d + m_d)}{(\mu_d+m_d)} \right) \lambda + (\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d)) = 0$$

$$\lambda^2 + \left(\frac{\beta_{ad}\mu_{nd}}{(\mu_d+m_d)} \right) \lambda + (\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d)) = 0$$

Because the equation is a quadratic equation where $\frac{\beta_{ad}\mu_{nd}}{(\mu_d+m_d)}$ is positive, then to ensure that the eigenvalue is negative, then $(\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d)) > 0$, therefore:

$$\beta_{ad}\mu_{nd} - (k_d + \mu_d)(\mu_d + m_d) > 0$$

$$\beta_{ad}\mu_{nd} > (k_d + \mu_d)(\mu_d + m_d)$$

$$\frac{\beta_{ad}\mu_{nd}}{(k_d + \mu_d)(\mu_d + m_d)} > 1$$

$$R_0 > 1$$

Then, the endemic equilibrium point is locally asymptotically stable.

3.4 Model Simulation

Next, a simulation model of the spread of rabies in humans and dogs in Maluku Province will be conducted. The initial value of each sub-population and parameter is determined based on data from several agencies related to population conditions in Maluku Province. This simulation was conducted using the *Matlab v920538062* program.

The summary value of each variable is presented in the following **Table 1**.

Table 1. Variable Values

Variables	Value	Source
S_h	1,875,088	
E_{h1}	87	Maluku Provincial Health Office
E_{h2}	2,548	Maluku Provincial Health Office
I_h	10	Maluku Provincial Health Office
V_h	2,086	Maluku Provincial Health Office
R_h	1,908	Maluku Provincial Health Office
S_d	97,813	
I_d	2,545	Maluku Provincial Health Office
V_d	25,090	Assumption

Source: Central Bureau of Statistics and Maluku Provincial Health Office [22][23]

According to data from the Central Bureau of Statistics of Maluku Province, the human population in 2022 reached 1,881,727 souls [24]. Since it is assumed that every individual born is vulnerable, it is obtained $N_h = 1,881,727$. The assumption value of V_d is 25,090, was determined because there is no real data of the vaccine dog population in Maluku Province in a specific year. Therefore, the value of V_d was estimated based on some other data from data in previous year.

Furthermore, the natural mortality rate of individuals can be obtained based on the life expectancy of people in Maluku Province. According to data from the Central Bureau of Statistics of Maluku Province, the life expectancy of people in Maluku Province in 2022 is 66.45 [25]. Then, the human natural mortality rate is obtained.

$$\mu_h = \frac{1}{\text{Life expectancy}} = \frac{1}{66.45} = 0.01504 \text{ per year}$$

Thus, since it is assumed that the birth rate is equal to the death rate (μ_h), then the birth rate μ_{nh} is 0,01504 per year.

$$\begin{aligned} \sigma_h &= \frac{1}{\text{Average incubation period}} = \frac{1}{90 \text{ days}} = \frac{1}{0.25 \text{ year}} = 4 \\ \rho_h &= \frac{\text{Exposed individuals who are undergoing treatment}}{\text{number of bite cases}} = \frac{2548}{2635} = 0.96698 \\ k_h &= \frac{\text{Number of vaccinated individuals}}{\text{number of bite cases}} = \frac{2086}{2635} = 0.79165 \text{ per year} \\ \alpha_h &= \frac{\text{Number of individuals who recovered after vaccination}}{\text{Total number of exposed and vaccinated individuals}} = \frac{1908}{2086} = 0.91467 \\ m_h &= \frac{\text{Number of individuals who died from rabies}}{\text{Total number of individuals infected with rabies}} = \frac{10}{10} = 1 \text{ per year} \end{aligned}$$

Furthermore, data on the total dog population in Maluku Province is not available, so it is assumed by using the ratio of 1: 15 with the total population, the dog population is obtained:

$$N_d = \frac{1,881,727}{15} = 125,448 \text{ head of dogs}$$

Since rabies-infected dogs are certain to die, the parameter values for the death rate of rabies-infected dogs are as follows:

$$m_d = 1$$

Table 2. Estimated Parameter Values

Parameters	Definition	Value	Source
μ_{nh}	Natural birth rate of individuals	0.0150	Central Bureau of Statistics
μ_h	Natural mortality rate of individuals	0.0150	Assumption
$\beta_h \delta$	Contact rate of infected dogs with susceptible individuals	0.03	[19]
σ_h	Incubation period rate	4	Maluku Health Office
ρ_h	Effectiveness of education	0.9670	Maluku Health Office
k_h	Vaccination rate in individuals	0.7916	Maluku Health Office

Parameters	Definition	Value	Source
m_h	Individual death rate due to disease	1	Maluku Health Office
α_h	Post-vaccination cure rate	0.9147	Maluku Health Office
μ_{nd}	Natural birth rate of dogs	0.1	Assumption
μ_d	Natural mortality rate of dogs	0.1	Assumption
$\beta_d \delta$	Chance of disease spread from infected to susceptible dogs	0.5	[19]
k_d	Vaccination rate in dogs	0.02	[26]
m_d	Dog death rate due to disease	1	Maluku Health Office

Source: Central Bureau of Statistics and Maluku Provincial Health Office [19][26]

From data of estimate parameter value in Table 2, can obtain the value of R_0 of the disease-free stability analysis and endemic stability analysis when:

$$R_0 = \frac{\beta_d \delta \mu_{nd}}{(k_d + \mu_d)(\mu_d + m_d)} = \frac{\beta_d \delta \mu_d}{(k_d + \mu_d)(\mu_d + m_d)} = 0,37878 < 1$$

and analysis of the stability of the locally asymptotically stable endemic, when:

$$R_0 \frac{\beta_d \delta \mu_{nd}}{(k_d + \mu_d)(\mu_d + m_d)} = \frac{\beta_d \delta \mu_d}{(k_d + \mu_d)(\mu_d + m_d)} = 1,0682 > 1$$

In simulations with initial $t_0 = 0$ and end time $t_f = 20$ years for disease-free stability analysis and $t_f = 50$ years for endemic stability analysis.

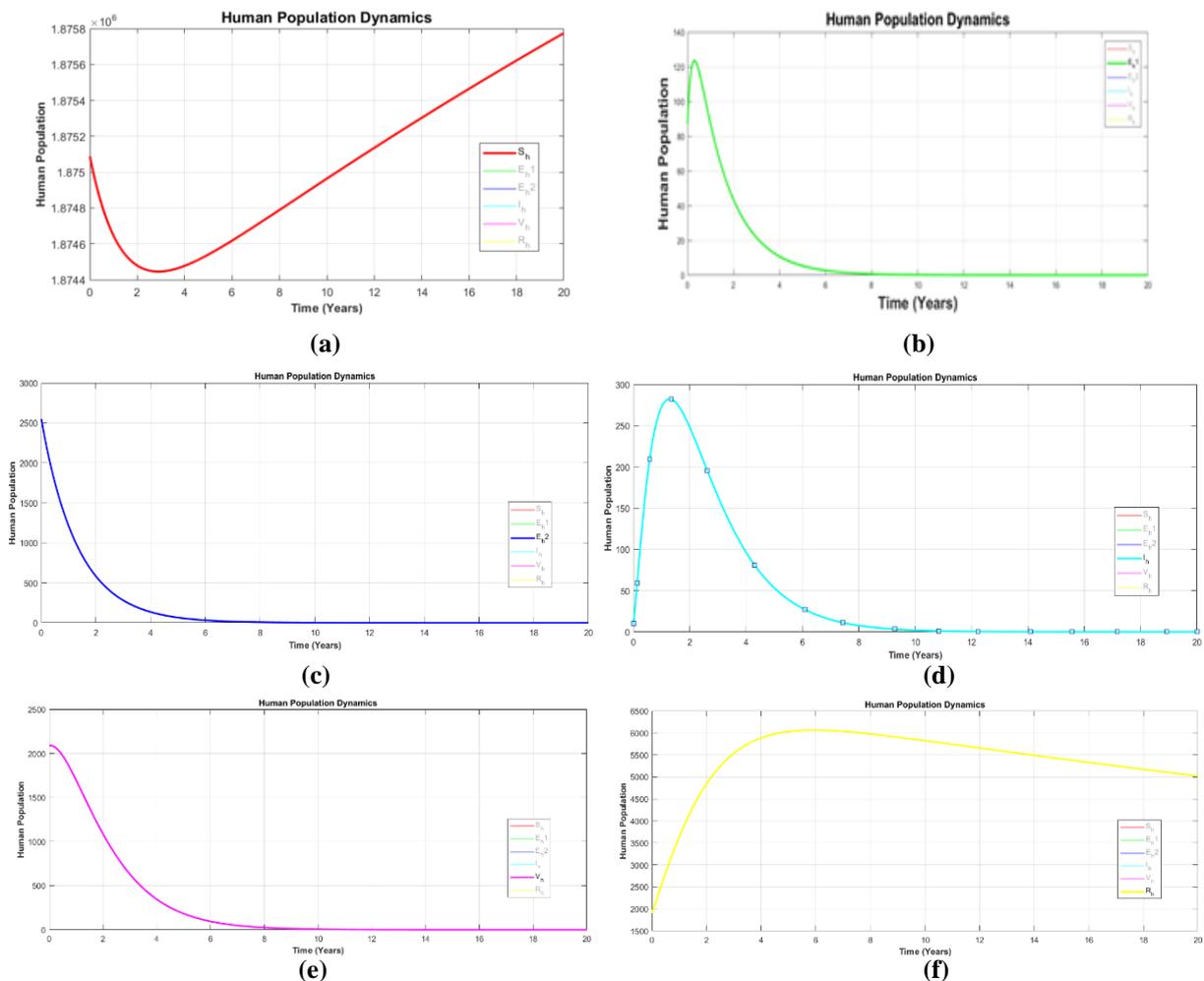


Figure 2. Graph of Disease-free Equilibrium Point of Individual Population
 (a) Subpopulation of Susceptible Individuals, (b) Subpopulation of Unaware Exposed Individuals,
 (c) Subpopulation of Aware Exposed Individuals, (d) Subpopulation of Infected Individuals,
 (e) Subpopulation of Vaccinated Individuals, (f) Subpopulation of Cured Individuals

Figure 2 shows the complex dynamics of rabies spread and the impact of vaccination in Maluku Province. Vulnerable and exposed populations significantly decreased, while infected individuals increased before declining. The vaccination program positively impacted the spread of rabies, as seen by the increased population of cured individuals.

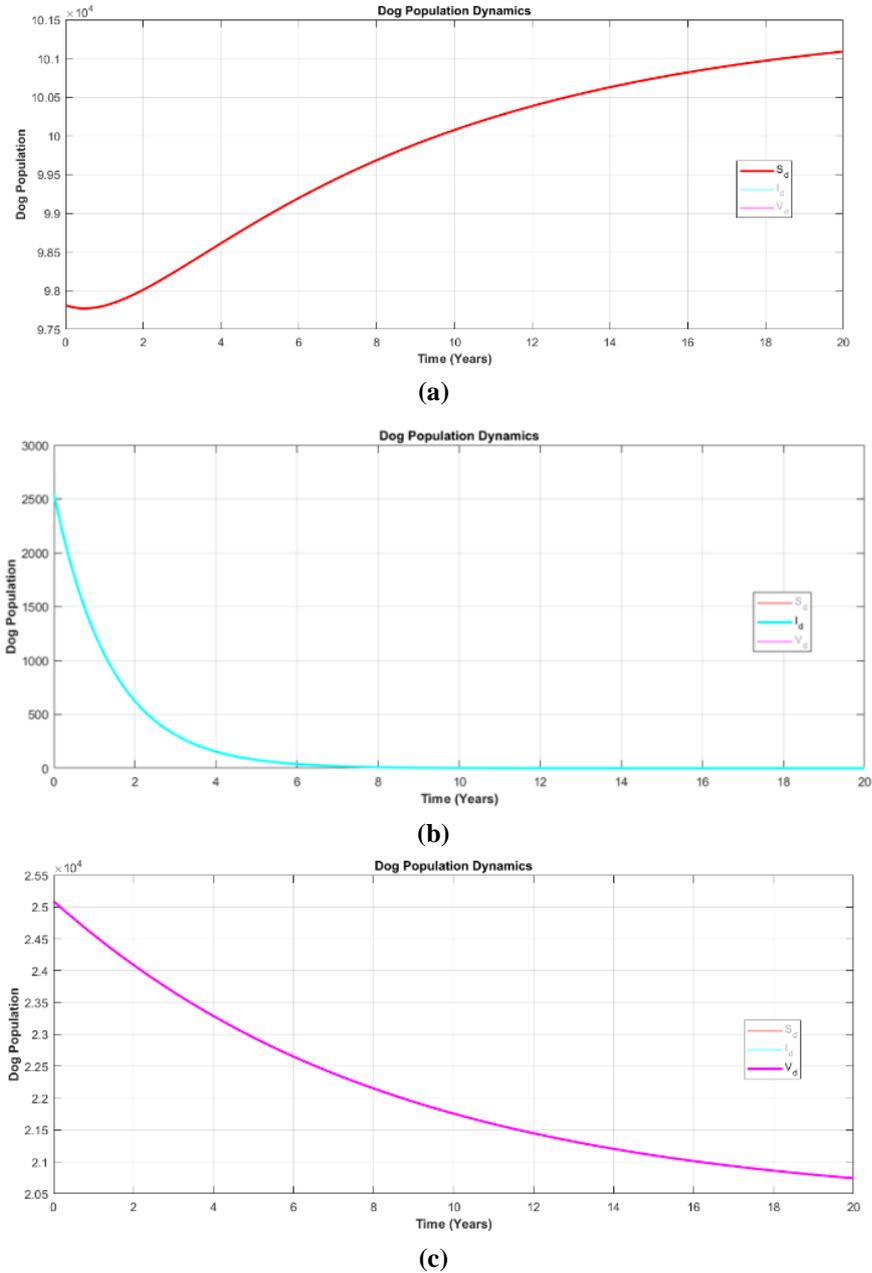


Figure 3. Graph of Disease-Free Equilibrium Point of Dog Population, (a) Subpopulation of Susceptible Dogs, (b) Subpopulation of Infected Dogs, (c) Subpopulation of Vaccinated Dogs

Figure 3 shows the population dynamics of dogs essential in spreading rabies and how interventions such as vaccination can affect these populations. Vulnerable dogs increased in population, while infected dogs decreased sharply due to control interventions. The dog vaccination program showed a positive impact in reducing the infected dog population and helped stabilize the outbreak situation.

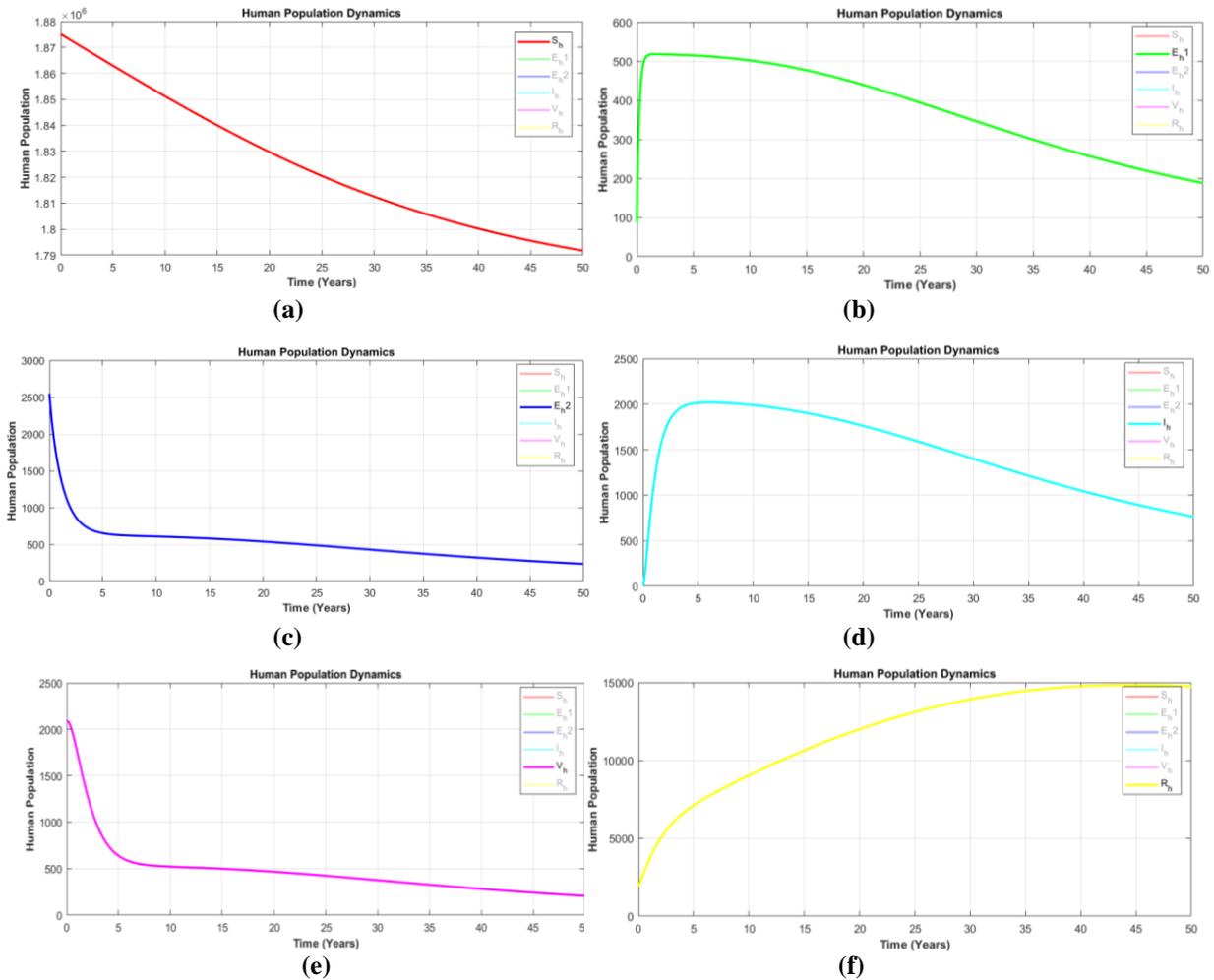
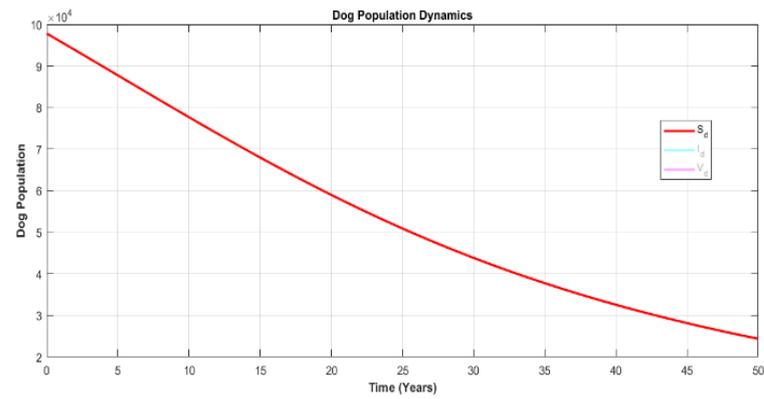
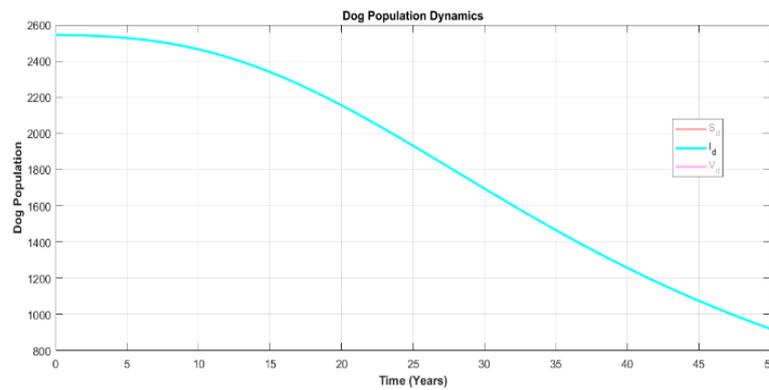


Figure 4: Graph of Endemic Equilibrium Point of Individual Population
 (a) Subpopulation of Susceptible Individuals, (b) Subpopulation of Unaware Exposed Individuals,
 (c) Subpopulation of Aware Exposed Individuals, (d) Subpopulation of Infected Individuals,
 (e) Subpopulation of Vaccinated Individuals, (f) Subpopulation of Cured Individuals

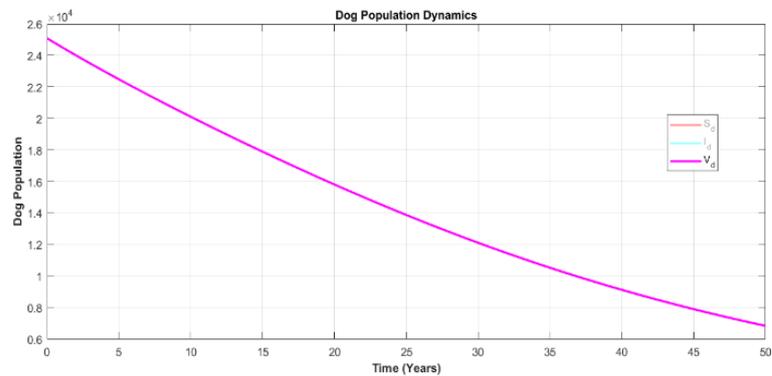
Figure 4 shows how each subpopulation changes over time in the rabies spread model. It shows the vital role of vaccination in reducing the number of susceptible and infected individuals and increasing the number of protected and cured individuals. Through this analysis, the disease dynamics and the effectiveness of vaccination interventions in controlling the spread of rabies can be understood.



(a)



(b)



(c)

Figure 5. Endemic Equilibrium Point Graph of Dog Population, (a) Subpopulation of Susceptible Dogs, (b) Subpopulation of Infected Dogs, (c) Subpopulation of Vaccinated Dogs

Figure 5 shows how each dog subpopulation changes over time in the rabies spread model. It has shown the critical role of vaccination in reducing the number of susceptible and infected dogs and increasing the number of protected dogs. The decrease in the number of susceptible and infected dogs and the increase in protected dogs demonstrate the effectiveness of vaccination interventions in controlling the spread of rabies. Vaccination programs are proven to reduce the prevalence of rabies in dog populations, which has a positive impact on controlling the spread of the disease.

4. CONCLUSIONS

Based on the results of the research that has been done, it can be concluded:

1. From the analysis of the model, it can be obtained that sub-populations (variables) of exposed individuals can be divided into two sub-populations, namely: sub-populations (variables) of exposed individuals who are not aware (E_{h2}) and exposed individuals who are aware (E_{h1}).

2. Based on the stability analysis of the mathematical model of rabies disease spread with vaccination in human and dog populations in Maluku Province, obtain that the disease-free stability analysis is locally asymptotically stable when $R_0 < 1$, and analysis of the stability of the locally asymptotically stable endemic, when $R_0 < 1$.
3. Simulations conducted in this study showed two main equilibrium points for the rabies transmission model: disease-free and endemic equilibrium. In the disease-free equilibrium, no individuals in the population are infected with rabies. This state is reached when the primary reproduction number R_0 is less than 1, indicating that each infected individual, on average, transmits the disease to fewer than one other individual. The infection will eventually go extinct, and the population will stabilize with no rabies cases. In contrast, endemic equilibrium occurs when the disease persists in the population, with the number of infected individuals remaining constant. This state occurs when R_0 is greater than 1, which means the infection will spread through the population. In this scenario, the disease becomes a regular part of the community, and ongoing efforts are required to manage and control its spread. The endemic equilibrium is characterized by a balance between new infections and recovery or death, leading to a steady state of disease prevalence.

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