



Prediction of Rabies Spread in Dogs and Humans in Muara Enim Regency Based on the SI–SEIR Model with Fourth-Order Runge–Kutta Numerical Solution

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ABSTRACT

Rabies is a fatal zoonotic disease that is generally transmitted through the bite of an infected dog. This research aims to predict the spread of rabies in dog and human populations in Muara Enim Regency using a combined SI–SEIR mathematical model. The system of nonlinear differential equations was solved using the fourth-order Runge–Kutta (RK4) method. Rabies data on dogs and humans were obtained from the Lampung Veterinary Center for the period 2021–2024. The simulation results show that the number of infected dogs increased significantly at the beginning of the period, peaked in the third year, and then declined as the population of susceptible dogs decreased. In the human population, the increase in exposed and infected individuals occurred after the surge in the dog population, indicating cross-species transmission. This model provides a quantitative overview of rabies dynamics and can be used as a basis for rabies control strategies in Muara Enim Regency.

Keywords: Rabies, SI–SEIR Model, The fourth-order Runge–Kutta, Numerical simulation, Epidemiology

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1. Introduction

Rabies is a deadly zoonotic disease that attacks the central nervous system and almost always causes death after clinical symptoms appear [1]. The WHO reports that approximately 59,000 human deaths are caused by rabies each year, and 99% of these are transmitted through dog bites [2]. In Indonesia, rabies remains a public health problem, including in Muara Enim Regency, which annually reports cases of bites from rabies-infecting animals [3]. To better understand rabies transmission, numerous studies have employed mathematical models to investigate infection dynamics in animal and human populations [4–7]. Compartmental models such as SIR, SEIR, and their variants have been widely used to quantify disease transmission and evaluate intervention strategies [8]. Recent studies have demonstrated that these models can capture the effects of contact rates, environmental factors,

and cross-species transmission on rabies dynamics in dogs and humans [9]. However, most existing studies focus primarily on theoretical analysis, stability properties, or generalized epidemiological scenarios, while relatively few incorporate regional epidemiological data to predict rabies transmission between dogs and humans in specific endemic areas [10]. Furthermore, studies integrating an SI model for dogs with an SEIR model for humans using local surveillance data remain limited, particularly for Muara Enim Regency. Therefore, developing a location-specific SI–SEIR model is important to provide more realistic predictions that can support evidence-based rabies control policies [9].

In the context of rabies with animal-to-human transmission, using combined models such as SI for dogs and SEIR for humans provides a more representative approach [11]. These models allow for a more detailed analysis of how infection in the primary reservoir (dogs) can drive increases in human cases. Nonlinear differential models with dog and human compartments can describe outbreak patterns and identify the most influential parameters in rabies transmission dynamics [12]. Epidemiological models such as the SI–SEIR system generally produce nonlinear ordinary differential equations that rarely admit closed-form analytical solutions, making numerical approximation essential. Among the available numerical methods, the fourth-order Runge–Kutta (RK4) method was selected because it provides significantly higher accuracy than the Euler and Heun methods while maintaining reasonable computational efficiency [13]. Unlike the Euler method, whose first-order accuracy often requires very small step sizes to achieve acceptable precision, RK4 achieves fourth-order accuracy and substantially reduces truncation errors. Compared with the Heun method, RK4 also offers better numerical stability for nonlinear epidemic models, making it more suitable for simulating long-term rabies transmission dynamics. Previous studies have demonstrated that RK4 produces reliable numerical solutions for SEIR-type epidemiological models with stable compartment trajectories [14].

This study proposes a data-driven SI–SEIR mathematical model combined with the fourth-order Runge–Kutta numerical method to simulate rabies transmission between dogs and humans in Muara Enim Regency. Unlike previous studies that primarily emphasize theoretical analysis or generalized epidemic settings, this research incorporates regional surveillance data to generate location-specific predictions of rabies dynamics. The proposed approach is expected to provide a more realistic representation of local transmission patterns and to support evidence-based decision making for rabies prevention and control. The main contribution of this study lies in integrating an SI model for the dog population and an SEIR model for the human population with locally calibrated epidemiological data and solving the resulting nonlinear system using the fourth-order Runge–Kutta method to obtain quantitative predictions for Muara Enim Regency. The remainder of this paper is organized as follows. Section 2 describes the research methodology, data sources, and the development of the SI–SEIR mathematical model. Section 3 presents the numerical solution using the fourth-order Runge–Kutta method and discusses the simulation results. Finally, Section 4 summarizes the main findings and outlines future research directions.

2. Research Methods

This study employed secondary surveillance data on rabies cases in dogs and humans in Muara Enim Regency covering the period from 2021 to 2024, obtained from the Lampung Veterinary Center (Balai Veteriner Lampung). The dataset consisted of annual epidemiological records reported in Table 1 and Table 2. For the human population, the dataset included the total population size (N), the numbers of susceptible (S), exposed (E), infected (I), recovered (R), and rabies-related deaths (D). For the dog population, the dataset contained the total dog population (N), susceptible dogs (S), infected dogs (I), and rabies-related deaths (D). Overall, the dataset comprised eight annual epidemiological observations, including four yearly records for the human population and four yearly records for the dog population. No missing values or inconsistent records were identified during the validation process.

These data were used to estimate the initial conditions and epidemiological parameters required for the SI–SEIR mathematical model.

This data served as the basis for constructing a combined SI–SEIR mathematical model. The dog population was modeled using the SI model because infected dogs cannot recover. In contrast, the human population was modeled using the SEIR model to describe the stages of vulnerability, exposure, infection, and recovery. The resulting system of nonlinear differential equations was then solved numerically using the fourth-order Runge–Kutta (RK4) method with a time step of one year and a simulation duration of five years. This numerical approach was chosen because it provides stable, accurate results for simulating the dynamics of rabies spread in both dog and human populations. The research procedure adopted in this study is illustrated in Figure 1. The workflow consists of six sequential stages, beginning with data acquisition and ending with numerical simulation and interpretation of the results [15].

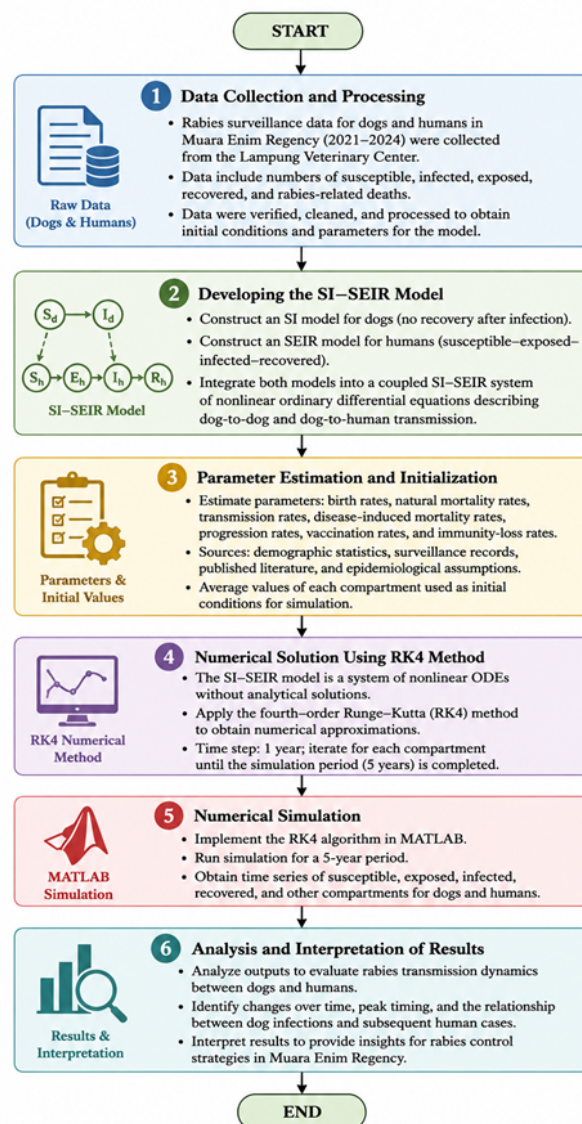


Figure 1. Research Procedures

3. Result and Discussion

The results of the SI–SEIR model simulation applied to the dog and human populations in Muara Enim Regency provide a quantitative picture. The simulation was conducted using the fourth-order Runge–Kutta (RK4) method with a one-year time step, allowing periodic population changes in each compartment. Overall, the results indicate a strong relationship between increasing infections in dogs, the primary reservoir, and the emergence of cases in humans, the secondary host.

3.1. SI-SEIR Mathematical Model

The SI-SEIR model for rabies spread described in this study consists of six subpopulations with the following assumptions:

1. Population assumption. The SI-SEIR model assumes an open population for both dogs and humans. The total population is not constant over time because individuals may enter the population through birth and leave through natural mortality. In the human population, additional losses may occur through rabies-induced mortality. Similarly, the dog population may change due to birth, natural mortality, and rabies-induced mortality. Consequently, the total populations of dogs and humans are dynamic and are not constrained to remain constant throughout the simulation period.
2. Subpopulation (S_d) is the number of susceptible dogs. Subpopulation (S_d) increases due to births but decreases due to rabies transmission and natural deaths.
3. Subpopulation (I_d) is the number of infected dogs. Subpopulation (I_d) increases due to transmission and decreases due to natural deaths and deaths from rabies.
4. Subpopulation (S_h) is the number of susceptible humans. Subpopulation (S_h) increases due to births and loss of immunity from recovered humans, but decreases due to infection from infected dogs and natural deaths.
5. Subpopulation (E_h) represents the number of exposed humans. The model assumes an open human population in which the total population is not constant over time. The (E_h) subpopulation increases through contact with infected dogs and decreases through progression to the infectious compartment and natural mortality. The overall human population may change through births, natural mortality, and rabies-induced mortality.
6. Subpopulation (I_h) is the number of infected humans. The subpopulation (I_h) increases as clinical symptoms emerge from the previously exposed phase. It decreases due to treatment or vaccination, as well as natural deaths and deaths from rabies diseases.
7. Subpopulation (R_h) is the number of recovered humans. The subpopulation (R_h) increases due to vaccination but can decrease due to loss of immunity, leading to re-susceptibility and natural death.

Based on this assumption, the spread of rabies can be presented schematically in a compartment diagram, as shown in Figure 2

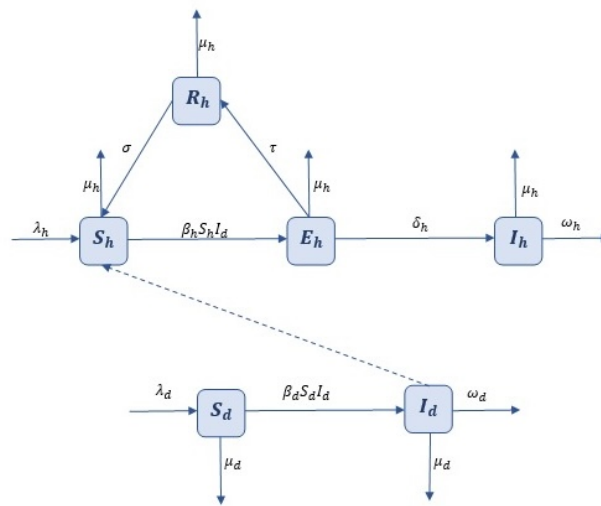


Figure 2. SI-SEIR Model Compartment Diagram

Based on the compartment diagram above, a mathematical model of rabies spread is formulated as a system of differential equations.

$$\frac{dS_d}{dt} = \lambda_d - \beta_d S_d I_d - \mu_d S_d, \quad (1)$$

$$\frac{dI_d}{dt} = \beta_d S_d I_d - (\mu_d + \omega_d) I_d, \quad (2)$$

$$\frac{dS_h}{dt} = \lambda_h + \sigma R_h - \beta_h S_h I_d - \mu_h S_h. \quad (3)$$

The term $\mu_h S_h$ represents natural mortality among susceptible humans. The natural mortality rate μ_h is consistently applied to all human compartments S_h, E_h, I_h, R_h , whereas the rabies-induced mortality rate ω_h is applied only to I_h . Therefore, susceptible humans are subject to natural mortality but not to rabies-induced mortality.

$$\frac{dE_h}{dt} = \beta_h S_h I_d - (\delta_h + \tau + \mu_h) E_h, \quad (4)$$

$$\frac{dI_h}{dt} = \delta_h E_h - (\omega_h + \mu_h) I_h, \quad (5)$$

$$\frac{dR_h}{dt} = \tau E_h - (\sigma + \mu_h)R_h. \quad (6)$$

where:

S_d : average number of susceptible dogs,

I_d : average number of infected dogs,

S_h : average number of susceptible humans,

E_h : average number of exposed humans,

I_h : average number of infected humans,

- R_h : average number of recovered humans,
- λ_d : dog population growth rate,
- β_d : rabies transmission rate between dogs,
- μ_d : natural death rate of dogs,
- ω_d : rabies-induced death rate of dogs,
- λ_h : human population growth rate,
- σ : human immunity loss rate,
- δ_h : progression rate from exposed to infected humans,
- β_h : dog-to-human transmission rate,
- τ : rate of exposed humans entering the recovered compartment through treatment or vaccination,
- ω_h : rabies-induced death rate of humans,
- μ_h : natural death rate of humans.

3.2. Equilibrium Points and Stability Analysis

To determine the equilibrium point it is determined that

$$\frac{dS_d}{dt} = 0; \frac{dI_d}{dt} = 0; \frac{dS_h}{dt} = 0; \frac{dE_h}{dt} = 0; \frac{dI_h}{dt} = 0; \frac{dR_h}{dt} = 0.$$

Let disease-free equilibrium point $I_d^* = 0; E_h^* = 0; I_h^* = 0$. Since there are no infected dogs then Equation 1 becomes $S_d^* = \frac{\lambda_d}{\mu_d}$. For human, since $I_d^* = 0; E_h^* = 0; I_h^* = 0$ then Equation 3 becomes $S_h^* = \frac{\lambda_h}{\mu_h}$. Thus, disease free equilibrium is

$$E_0 = (S_d, I_d, S_h, E_h, I_h, R_h) = \left(\frac{\lambda_d}{\mu_d}, 0, \frac{\lambda_h}{\mu_h}, 0, 0, 0\right).$$

In endemic equilibrium, the infection persists so that $I_d^* > 0$. From Equation 2 we get $S_d^* = \frac{\mu_d + \omega_d}{\beta_d}$. Thus,

$$I_d^* = \frac{\lambda_d - \mu_d S_d^*}{\mu_d + \omega_d}.$$

Since $S_d^* = \frac{\mu_d + \omega_d}{\beta_d}$ then

$$I_d^* = \frac{\lambda_d}{\mu_d + \omega_d} - \frac{\mu_d}{\omega_d}.$$

That equation can be used as the basic reproduction number. For the dog section,

$$R_{0d} = \frac{\beta_d \lambda_d}{\mu_d(\mu_d + \omega_d)}$$

and

$$I_d^* = \frac{\mu_d}{\beta_d}(R_{0d} - 1).$$

Thus, the endemic equilibrium is positive only if $R_{0d} > 1$. The Jacobian matrix is obtained as follows.

$$J = \begin{bmatrix} -\beta_d I_d - \mu_d & -\beta_d S_d & 0 & 0 & 0 & 0 \\ \beta_d I_d & \beta_d S_d - (\mu_d + \omega_d) & 0 & 0 & 0 & 0 \\ 0 & -\beta_h S_h & -\beta_h I_d - \mu_h & 0 & 0 & \sigma \\ 0 & \beta_h S_h & \beta_h I_d & -(\delta_h + \tau + \mu_h) & 0 & 0 \\ 0 & 0 & 0 & \delta_h & -(\omega_h + \mu_h) & 0 \\ 0 & 0 & 0 & \tau & 0 & -(\sigma + \mu_h) \end{bmatrix}.$$

To determine stability at disease-free equilibrium, substitute $E_0 = \left(\frac{\lambda_d}{\mu_d}, 0, \frac{\lambda_h}{\mu_h}, 0, 0, 0\right)$ to the Jacobian. The eigenvalues (EV) relating to the dynamics of dogs become:

1. $EV_1 = -\mu_d$;
2. $EV_2 = (\mu_d + \omega_d)(R_{0d} - 1)$ where $R_{0d} = \frac{\beta_d \lambda_d}{\mu_d(\mu_d + \omega_d)}$;
3. $EV_3 = -\mu_h$;
4. $EV_4 = -(\delta_h + \tau + \mu_h)$;
5. $EV_5 = -(\omega_h + \mu_h)$;
6. $EV_6 = -(\sigma + \mu_h)$.

Thus, the disease-free equilibrium is locally asymptotically stable when $R_{0d} < 1$, whereas it becomes unstable when $R_{0d} > 1$.

3.3. Solving the SI-SEIR Model Using the fourth-order Runge-Kutta Method

The fourth-order Runge-Kutta method provides high accuracy for solving ordinary differential equation. This high accuracy enables reliable, precise solutions. The fourth-order Runge-Kutta method is known for its numerical stability. This method is suitable for solving differential equations where other methods may fail or require very small step sizes. Based on the System of Equations in Equations (1) to (6), this method is solved using the Fourth-Order Runge-Kutta method as shown in the following Equation: $Y_k = Y_k + (k_1 + 2k_2 + 2k_3 + k_4)$. The rabies disease spread model to be solved is already a system of equations, so it does not need to be modified. The system of equations consisting of Equation (1) to Equation (6) is substituted into the fourth-order Runge-Kutta equations to obtain Equations (7) to Equation (12). To numerically solve the nonlinear SI-SEIR system, the fourth-order Runge-Kutta (RK4) method was applied. For each compartment, four intermediate function evaluations, denoted by f_1, f_2, f_3 , and f_4 were calculated at each iteration. These evaluations correspond to the initial point, two intermediate points, and the endpoint of the time step, respectively.

$$S_d(i+1) = S_d(i) + \left(\frac{h}{6}\right) (f_{1,S_d} + 2f_{2,S_d} + 2f_{3,S_d} + f_{4,S_d}), \quad (7)$$

$$I_d(i+1) = I_d(i) + \left(\frac{h}{6}\right) (f_{1,I_d} + 2f_{2,I_d} + 2f_{3,I_d} + f_{4,I_d}), \quad (8)$$

$$S_h(i+1) = S_h(i) + \left(\frac{h}{6}\right) (f_{1,S_h} + 2f_{2,S_h} + 2f_{3,S_h} + f_{4,S_h}), \quad (9)$$

$$E_h(i+1) = E_h(i) + \left(\frac{h}{6}\right) (f_{1,E_h} + 2f_{2,E_h} + 2f_{3,E_h} + f_{4,E_h}), \quad (10)$$

$$I_h(i+1) = I_h(i) + \left(\frac{h}{6}\right) (f_{1,I_h} + 2f_{2,I_h} + 2f_{3,I_h} + f_{4,I_h}), \quad (11)$$

$$R_h(i+1) = R_h(i) + \left(\frac{h}{6}\right) (f_{1,R_h} + 2f_{2,R_h} + 2f_{3,R_h} + f_{4,R_h}). \quad (12)$$

where:

$$f_{1,S_d} = \lambda_d - \beta_d S_d I_d - \mu_d S_d$$

$$f_{1,I_d} = \beta_d S_d I_d - (\mu_d + \omega_d) I_d$$

$$f_{1,S_h} = \lambda_h + \sigma R_h - \beta_h S_h I_d + \mu_d S_d$$

$$f_{1,E_h} = \beta_h S_h I_d - (\delta + \tau + \mu_h) E_h$$

$$\begin{aligned}
f_{1,I_h} &= \delta E_h - (\omega_h + \mu_h) I_h \\
f_{1,R_h} &= \tau E_h - (\sigma + \mu_h) R_h \\
f_{2,S_d} &= \lambda_d - \beta_d \left(S_d + \frac{1}{2} h f_{1,S_d} \right) \left(I_d + \frac{1}{2} h f_{1,I_d} \right) - \mu_d \left(S_d + \frac{1}{2} h f_{1,S_d} \right) \\
f_{2,I_d} &= \beta_d \left(S_d + \frac{1}{2} h f_{1,S_d} \right) \left(I_d + \frac{1}{2} h f_{1,I_d} \right) - (\mu_d + \omega_d) \left(I_d + \frac{1}{2} h f_{1,I_d} \right) \\
f_{2,S_h} &= \lambda_h + \sigma \left(R_h + \frac{1}{2} h f_{1,R_h} \right) - \beta_h \left(S_h + \frac{1}{2} h f_{1,S_h} \right) \left(I_d + \frac{1}{2} h f_{1,I_d} \right) - \mu_h \left(S_h + \frac{1}{2} h f_{1,S_h} \right) \\
f_{2,E_h} &= \beta_h \left(S_h + \frac{1}{2} h f_{1,S_h} \right) \left(I_d + \frac{1}{2} h f_{1,I_d} \right) - (\delta + \tau + \mu_h) \left(E_h + \frac{1}{2} h f_{1,E_h} \right) \\
f_{2,I_h} &= \delta \left(E_h + \frac{1}{2} h f_{1,E_h} \right) - (\omega_h + \mu_h) \left(I_h + \frac{1}{2} h f_{1,I_h} \right) \\
f_{2,R_h} &= \tau \left(E_h + \frac{1}{2} h f_{1,E_h} \right) - (\sigma + \mu_h) \left(R_h + \frac{1}{2} h f_{1,R_h} \right) \\
f_{3,S_d} &= \lambda_d - \beta_d \left(S_d + \frac{1}{2} h f_{2,S_d} \right) \left(I_d + \frac{1}{2} h f_{2,I_d} \right) - \mu_d \left(S_d + \frac{1}{2} h f_{2,S_d} \right) \\
f_{3,I_d} &= \beta_d \left(S_d + \frac{1}{2} h f_{2,S_d} \right) \left(I_d + \frac{1}{2} h f_{2,I_d} \right) - (\mu_d + \omega_d) \left(I_d + \frac{1}{2} h f_{2,I_d} \right) \\
f_{3,S_h} &= \lambda_h + \sigma \left(R_h + \frac{1}{2} h f_{2,R_h} \right) - \beta_h \left(S_h + \frac{1}{2} h f_{2,S_h} \right) \left(I_d + \frac{1}{2} h f_{2,I_d} \right) - \mu_h \left(S_h + \frac{1}{2} h f_{2,S_h} \right) \\
f_{3,E_h} &= \beta_h \left(S_h + \frac{1}{2} h f_{2,S_h} \right) \left(I_d + \frac{1}{2} h f_{2,I_d} \right) - (\delta + \tau + \mu_h) \left(E_h + \frac{1}{2} h f_{2,E_h} \right) \\
f_{3,I_h} &= \delta \left(E_h + \frac{1}{2} h f_{2,E_h} \right) - (\omega_h + \mu_h) \left(I_h + \frac{1}{2} h f_{2,I_h} \right) \\
f_{3,R_h} &= \tau \left(E_h + \frac{1}{2} h f_{2,E_h} \right) - (\sigma + \mu_h) \left(R_h + \frac{1}{2} h f_{2,R_h} \right) \\
f_{4,S_d} &= \lambda_d - \beta_d (S_d + h f_{3,S_d}) (I_d + h f_{3,I_d}) - \mu_d (S_d + h f_{3,S_d}) \\
f_{4,I_d} &= \beta_d (S_d + h f_{3,S_d}) (I_d + h f_{3,I_d}) - (\mu_d + \omega_d) (I_d + h f_{3,I_d}) \\
f_{4,S_h} &= \lambda_h + \sigma (R_h + h f_{3,R_h}) - \beta_h (S_h + h f_{3,S_h}) (I_d + h f_{3,I_d}) - \mu_h (S_h + h f_{3,S_h}) \\
f_{4,E_h} &= \beta_h (S_h + h f_{3,S_h}) (I_d + h f_{3,I_d}) - (\delta + \tau + \mu_h) (E_h + h f_{3,E_h}) \\
f_{4,I_h} &= \delta (E_h + h f_{3,E_h}) - (\omega_h + \mu_h) (I_h + h f_{3,I_h}) \\
f_{4,R_h} &= \tau (E_h + h f_{3,E_h}) - (\sigma + \mu_h) (R_h + h f_{3,R_h})
\end{aligned}$$

3.4. Numerical Simulation Using the fourth-order Runge-Kutta Method

The SEIR model was simulated using the fourth-order Runge-Kutta method. This simulation was performed by substituting the initial values with secondary data and predetermined parameter values. The data used in the simulation are presented in Tables 1 and 2 below.

Table 1. Distribution Data of Rabies Disease in Dogs

Year	N_d	S_d	I_d	D_d
2021	5,528	5,518	5	5
2022	14,989	14,979	5	5
2023	934	928	6	6
2024	660	656	4	4
Total	22,111	22,081	20	20
Average	5,528	5,520	5	5

In Table 1 and Table 2, N_d and N_h denote the total dog and human populations, respectively, while D_d and D_h denote the numbers of rabies-related deaths among dogs and humans, respectively. The N_d and N_h values represent the total populations reported for the corresponding year, whereas D_d and D_h represent annual rabies-related death records. Therefore, D_d and D_h are reported separately from the epidemiological subpopulations used in the SI–SEIR model.

Table 2. Distribution Data of Rabies Disease in Humans

Year	N_h	S_h	E_h	I_h	R_h	D_h
2021	617,190	617,180	5	0	5	0
2022	624,000	624,000	2	0	2	0
2023	640,200	640,157	20	3	17	3
2024	653,731	653,717	7	3	4	1
Total	2,535,121	2,535,054	34	6	28	4
Average	633,780	633,764	9	2	7	1

Based on data obtained from 2021 to 2024, the average population of subpopulation S_d is 5,520 dogs, subpopulation I_d is 5 dogs, subpopulation S_h is 633,764 people, subpopulation E_h is 9 people, subpopulation I_h is 2 people, and subpopulation R_h is 7, and subpopulation D_h is 1 person. Thus, the initial values are as shown in Table 3.

Table 3. Initial Values

Variable	Value	Description
S_d	5,520	Average number of the dog population susceptible to rabies
I_d	5	Average number of dogs in the population infected with rabies
S_h	633,764	Average number of human population susceptible to rabies
E_h	9	Average number of people exposed to rabies
I_h	2	Average number of people infected with rabies
R_h	7	Average number of human population recovered from rabies

Dogs generally enter the senior phase between 7 and 11 years of age, with a median age at death of around 12 years [16]. Therefore, the average lifespan of a dog can be taken as 8 years as a conservative estimate for a stray dog population at higher risk of death due to environmental factors and disease. The natural mortality rate of dogs (μ_d) in the mathematical model can be calculated using the formula:

$$\mu_d = \frac{1}{\text{average dog age}}$$

with an average dog age of 8 years, then:

$$\mu_d = \frac{1}{8} = 0.125.$$

Meanwhile, the rabies mortality rate in dogs is calculated based on data on dogs that died from rabies infection. Based on the data obtained, it is known that 5 dogs have died from rabies infection in the last 4 years. Therefore, the human rabies mortality rate is:

$$\omega_d = \frac{\text{average number of rabies-related dog deaths}}{\text{average number of infected dogs}}$$

to obtain

$$\omega_d = \frac{5}{5} = 1.$$

Although this value appears high, it is biologically consistent with the clinical progression of rabies. Rabies is a progressive neurological disease that is almost invariably fatal in animals once clinical signs appear. Therefore, the value $\omega_d = 1$ reflects the observed mortality pattern in the local surveillance data and is consistent with the near-100% fatality of clinical rabies in animals.

We can calculate the natural human mortality rate from the population's life expectancy in Muara Enim Regency. According to data from the Central Statistics Agency of Muara Enim Regency, the life expectancy of the population of Muara Enim Regency is 68.11. Therefore, the natural human mortality rate is:

$$\mu_h = \frac{1}{\text{life expectancy}}$$

to obtain

$$\mu_h = \frac{1}{68.11} = 0.01468.$$

Meanwhile, the human rabies mortality rate is calculated based on data on humans who died from rabies infection. Based on the data obtained, it is known that 4 humans have died from rabies infection in the last 4 years. Thus, the number of deaths due to rabies in humans is:

$$\omega_h = \frac{\text{average number of rabies-related deaths}}{\text{average number of infected individuals}}$$

thus, we obtain

$$\omega_h = \frac{1}{2} = 0.5.$$

The rate of recovered and resuscitated humans (σ) is determined based on the duration of antibody persistence after rabies vaccination. According to research [17], protective antibodies remain detectable for up to 3 years after vaccination. Therefore, the rate of loss of immunity is calculated using the formula.

$$\sigma = \frac{1}{\text{duration of immunity persistence (in years)}}$$

we obtain

$$\sigma = \frac{1}{3} = 0.33.$$

From the data in Tables 1 and 2, the parameter values can be obtained as follows:

$$\lambda_d = \frac{N(t+1) - N_t}{N_t}.$$

$$\text{For } 2021 \rightarrow 2022, \lambda_d = \frac{(14,989 - 5,528)}{5,528} = 1.71147.$$

$$\text{For } 2022 \rightarrow 2023, \lambda_d = \frac{(934 - 14,989)}{14,989} = -0.93769.$$

$$\text{For } 2023 \rightarrow 2024, \lambda_d = \frac{(660 - 934)}{934} = -0.29336.$$

Average dog birth rate is:

$$\lambda_d = \frac{(1.71147 + (-0.93769) + (-0.29336))}{3} = \frac{0.48042}{3} = 0.16014.$$

where $\lambda_h = \frac{N(t+1) - N_t}{N_t}$.

For $2021 \rightarrow 2022$, $\lambda_h = \frac{(624,000 - 617,190)}{617,190} = 0.01103$.

For $2022 \rightarrow 2023$, $\lambda_h = \frac{(640,200 - 624,000)}{624,000} = 0.02596$.

For $2023 \rightarrow 2024$, $\lambda_h = \frac{(653,731 - 640,200)}{640,200} = 0.02114$.

Average human birth rate is:

$$\lambda_h = \frac{(0.01103 + 0.02596 + 0.02114)}{3} = \frac{0.058}{3} = 0.01938.$$

Next, the other parameter values are obtained as follows:

$$\beta_d = \frac{I_d}{N_d} = \frac{5}{5,528} = 0.000904.$$

$$\beta_h = \frac{I_h}{N_h} = \frac{2}{633,780} = 0.00000315.$$

$$\delta = \frac{I_h}{E_h} = \frac{2}{9} = 0.222.$$

$$\tau = \frac{R_h}{E_h} = \frac{7}{9} = 0.778.$$

The parameter values are then presented in Table 4 below.

Table 4. Parameter Values

Parameter	Definition	Value
λ_d	Dog Birth Rate	0.16014
μ_d	Dog Natural Death Rate	0.125
β_d	Dog Rabies Transmission Rate between Dogs	0.000904
ω_d	Dog Death Rate due to Rabies	1
λ_h	Human Birth Rate	0.01938
μ_h	Human Natural Death Rate	0.01468
β_h	Dog Human Transmission Rate	0.00000315
δ	Rate of Humans Exposed and Infected	0.222
τ	Rate of Humans Exposed and Recovered	0.778
ω_h	The Rate of Human Deaths Due to Rabies	0.5
σ	Immunity loss rate	0.33

The simulation was performed by substituting the initial values and parameter values into Equations 7 and 12, which represent the numerical solution of the rabies disease model using the fourth-order Runge-Kutta method. The numerical solution of the rabies disease spread model using the fourth-order Runge-Kutta method was obtained as follows.

$$\begin{aligned}
 S_d(i+1) &= S_d(i) + \frac{h}{6}(f_{1,S_d} + 2f_{2,S_d} + 2f_{3,S_d} + f_{4,S_d}) \\
 &= 5,520 + \frac{1}{6}(-714.79026 + 2(-713.5962038) + 2(-789.7824537) + (-1,081.603896)) \\
 &\approx 4,719.474755.
 \end{aligned}$$

$$\begin{aligned}
I_d(i+1) &= I_d(i) + \frac{h}{6}(f_{1,I_d} + 2f_{2,I_d} + 2f_{3,I_d} + f_{4,I_d}) \\
&= 5 + \frac{1}{6}(19.3254 + 2(51.93520626) + 2(109.703803) + 361.4450648) \\
&\approx 122.3414139.
\end{aligned}$$

$$\begin{aligned}
S_h(i+1) &= S_h(i) + \frac{h}{6}(f_{1,S_h} + 2f_{2,S_h} + 2f_{3,S_h} + f_{4,S_h}) \\
&= 633,764 + \frac{1}{6}(-9,311.307683 + 2(-9,261.28086) + 2(-9,294.021396) + (-9,386.750989)) \\
&\approx 624,462.5561.
\end{aligned}$$

$$\begin{aligned}
E_h(i+1) &= E_h(i) + \frac{h}{6}(f_{1,E_h} + 2f_{2,E_h} + 2f_{3,E_h} + f_{4,E_h}) \\
&= 9 + \frac{1}{6}(0.849663 + 2(19.49375685) + 2(42.34665301) + 173.5313093) \\
&\approx 58.67696534.
\end{aligned}$$

$$\begin{aligned}
I_h(i+1) &= I_h(i) + \frac{h}{6}(f_{1,I_h} + 2f_{2,I_h} + 2f_{3,I_h} + f_{4,I_h}) \\
&= 2 + \frac{1}{6}(0.96864 + 2(0.813682775) + 2(2.923476978) + 8.864914551) \\
&\approx 4.884645676.
\end{aligned}$$

$$\begin{aligned}
R_h(i+1) &= R_h(i) + \frac{h}{6}(f_{1,R_h} + 2f_{2,R_h} + 2f_{3,R_h} + f_{4,R_h}) \\
&= 7 + \frac{1}{6}(4.58924 + 2(4.221795485) + 2(11.42582678) + 33.59658644) \\
&\approx 18.5801785.
\end{aligned}$$

Thus, we obtain for $i = 0$, $(S_d) = 4,719.474755$, $(I_d) = 122.3414139$, $(S_h) = 624,462,5561$, $(E_h) = 58.67696534$, $(I_h) = 4.884645676$ and $(R_h) = 18.5801785$. Then, the same procedure is performed for the next iteration with $Sd_i, Id_i, Sh_i, Eh_i, Ih_i, Rh_i$ where $i = 0, 1, 2, \dots, t$.

The iteration of the numerical solution of the rabies disease spread model using the fourth-order Runge-Kutta method up to $t = 5$ years is presented in the Figure 3, Figure 4, and Table 5.

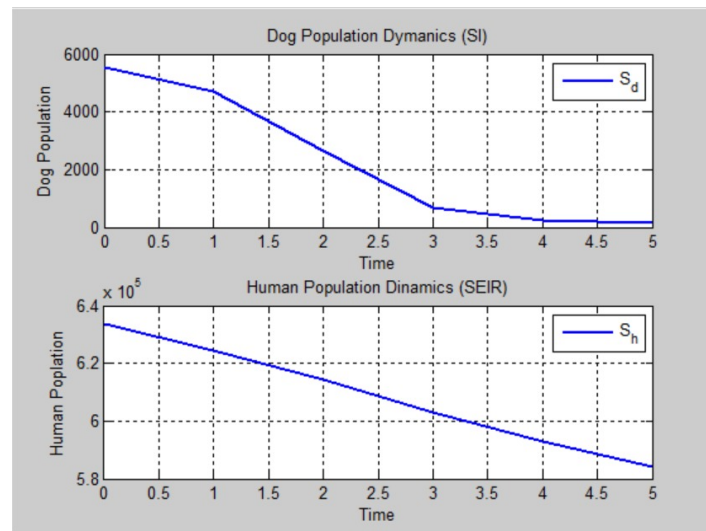


Figure 3. Graph of Results of Susceptible Dog Population (S_d) and Susceptible Human Population (S_h)

According to Figure 3 the susceptible dog population declined sharply from around 6,000 to nearly zero in the fifth year. This decline occurred because more dogs were infected through direct contact, reducing the number of susceptible dogs. This pattern indicates that rabies transmission in the dog population was rapid at the beginning of the simulation, then slowed once most dogs had become infected.

Meanwhile, the susceptible human population declined more slowly, from around 640,000 to 580,000 in five years. This decline reflects the movement of a small proportion of individuals into the exposed compartment due to indirect contact with infected dogs. Comparing the two graphs confirms that dogs are the primary vectors of rabies transmission, while human transmission occurs secondary to animal infection.

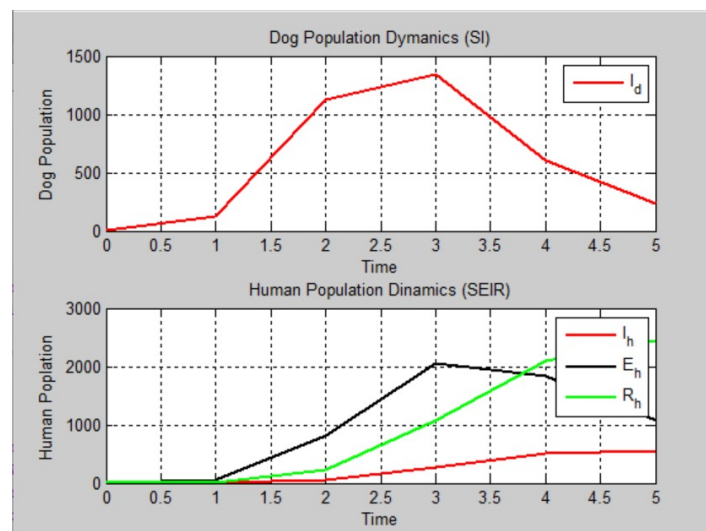


Figure 4. Graph of Results of Infected Dogs (I_d), Exposed Humans (E_h), Infected Humans (I_h), and Recovered Humans (R_h)

Figure 4 shows that the number of dogs infected with rabies increased sharply from the start of the simulation and peaked in the third year, reaching nearly 1,400. This spike was caused by the high rate of transmission between dogs through direct contact, particularly bites. After reaching the peak, the number of infected dogs gradually declined as susceptible dogs decreased and deaths increased, both natural and due to rabies. This pattern illustrates the peak phase of an outbreak, followed by a decline as the remaining population of potential carriers is depleted.

In the human population, the SEIR graph shows that the number of exposed humans (E_h) increased as cases in dogs increased, peaking in the third year. The number of infected humans (I_h) increased more slowly and peaked near the end of the simulation period because rabies transmission from dogs to humans requires direct contact, such as a bite. Meanwhile, the number of recovered humans (R_h) continues to increase through the end of the simulation due to transitions between the exposed and infected phases, as well as the effectiveness of vaccination and treatment.

Overall, the relationship between the two graphs demonstrates a strong link between rabies dynamics in dogs and humans. Human cases have consistently followed the trend seen in dogs, confirming that human infections are secondary to animal transmission. These findings suggest that controlling rabies in dogs through mass vaccination and monitoring stray dog populations is a key step to reducing the risk of transmission to humans.

Table 5. Numerical Simulation Results

Year	S_d	I_d	S_h	E_h	I_h	R_h
0	5,520	5	$6.3376 \cdot 10^5$	9	2	7
1	4,719	122	$6.2446 \cdot 10^5$	59	5	19
2	2,634	1,122	$6.1435 \cdot 10^5$	809	59	219
3	667	1,341	$6.0298 \cdot 10^5$	2,058	276	1,070
4	251	606	$5.9289 \cdot 10^5$	1,847	515	2,087
5	155	237	$5.8428 \cdot 10^5$	1,078	560	2,439

4. Conclusions

The SI–SEIR model simulations indicate that rabies transmission in the dog population plays an important role in the emergence of human rabies cases in Muara Enim Regency. The number of infected dogs increases sharply at the beginning of the simulation and subsequently declines as the susceptible dog population decreases, indicating the occurrence of an infection peak. In the human population, the increase in exposed and infected individuals occurs after the increase in infected dogs, suggesting a temporal relationship in which human cases arise following rabies transmission from infected dogs. The number of recovered humans also increases over time, reflecting the role of post-bite treatment and vaccination in the modeled population.

This study contributes to the application of a coupled SI–SEIR mathematical model to describe rabies transmission between dogs and humans at the regional level. By integrating dog-to-dog and dog-to-human transmission dynamics and solving the resulting nonlinear system using the fourth-order Runge–Kutta method, the study provides a quantitative representation of the temporal relationship between rabies transmission in dogs and the emergence of human cases. The results provide a mathematical basis for emphasizing dog-focused rabies control and surveillance as an important component of human rabies prevention in Muara Enim Regency.

This study has several limitations. First, the analysis is based on rabies surveillance data from 2021–2024, which limits the length of the observation period and the availability of detailed temporal information. Second, several model parameters were estimated from available surveillance data, demographic information, published references, and epidemiological assumptions; therefore, parameter

uncertainty may affect the simulation results. Third, the model uses a deterministic compartmental framework and does not explicitly incorporate spatial heterogeneity, stochastic effects, seasonal variation, or differences in vaccination coverage among locations. In addition, the model does not evaluate alternative intervention scenarios quantitatively.

Future research could extend the model by incorporating more detailed temporal and spatial data, vaccination coverage and vaccination effectiveness, and uncertainty or sensitivity analysis of the model parameters. Intervention-based simulations could also be conducted to compare the effectiveness of different vaccination and surveillance strategies in reducing rabies transmission from dogs to humans. Such extensions would provide a more comprehensive basis for evaluating rabies control policies in Muara Enim Regency.

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