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A Compartmental Model for the Transmission Dynamics of Rabies Disease in Dog Population

(Suatu Model Petak untuk Transmisi Dinamik Penyakit Rabies dalam Populasi Anjing)

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ABSTRACT

Dogs are the main source of more than 90% of human rabies infections that pose a significant threat to public health, primarily in Africa and Asia. However, it is also one of the viral diseases that can be prevented by vaccination that affects both warm-blooded animals and humans. There are two types of rabies vaccines: pre-exposure prophylaxis and post-exposure prophylaxis (PEP). Mathematical models can be valuable tools for predicting and controlling the spread of rabies disease. Thus, we introduce an SEIV (Susceptible-Exposed-Infected-Vaccinated) model incorporate vaccination control strategy to examine the transmission dynamics of rabies disease in dog population. The basic reproduction number, R_0, positively invariant and attracting region, steady states, and the stability analysis of the model are investigated. We find that there are two equilibria exist in the model, i.e., disease-free and endemic equilibria. To prove the global stability of disease-free and endemic equilibria, the theory of asymptotic autonomous system and geometric approach have been applied, respectively. Hence, we find that the disease-free and endemic equilibria are globally asymptotically stable if $R_0 < 1$ and $R_0 < 1$, respectively. Numerical simulations are performed to depict the dynamics of the model. As a conclusion, we will be able to control the disease effectively if the vaccination rate is sufficiently large.

Keywords: Nonlinear ordinary differential equation; numerical simulation; rabies disease; stability analysis; steady state

ABSTRAK

Anjing merupakan punca utama lebih daripada 90% jangkitan rabies manusia yang menimbulkan ancaman ketara kepada kesihatan awam, terutamanya di Afrika dan Asia. Walau bagaimanapun, ia juga merupakan salah satu penyakit virus yang boleh dicegah dengan vaksinasi yang memberi kesan kepada haiwan berdarah panas dan manusia. Terdapat dua jenis vaksin rabies: profilaksis pra-pendedahan dan profilaksis pasca-pendedahan (PPP). Model matematik boleh menjadi alat yang berguna untuk meramal dan mengawal penyebaran penyakit rabies. Oleh itu, kami memperkenalkan suatu model SEIV (Rentan-Terdedah-Dijangkiti-Diberi vaksin) yang menggabungkan strategi kawalan vaksinasi untuk mengkaji dinamik penularan penyakit rabies dalam populasi anjing. Nombor reproduksi asas, R_0, rantau invarian positif dan menarik, keadaan pegun dan analisis kestabilan model telah dikaji. Kami mendapati bahawa terdapat dua titik keseimbangan yang wujud dalam model, iaitu, titik keseimbangan bebas penyakit dan endemik. Untuk membuktikan kestabilan global bagi titik keseimbangan bebas penyakit dan endemik, teori sistem autonomi asimptotik dan pendekatan geometri masing-masing telah diaplikasi. Oleh itu, kami mendapati bahawa titik keseimbangan bebas penyakit dan endemik masing-masing mencapai kestabilan secara asimptotik secara global jika $R_0 < 1$ dan $R_0 < 1$. Simulasi berangka dijalankan untuk menggambarkan kedinamikan model. Kesimpulannya, kita akan dapat mengawal penyakit ini dengan berkesan sekiranya kadar vaksinasi cukup besar.

Kata kunci: Analisis kestabilan; keadaan pegun; penyakit rabies; persamaan pembezaan biasa tak linear; simulasi berangka

INTRODUCTION

Rabies disease is a severe viral illness that attacks the central nervous system of mammals (animals and humans), leading to neurological disease in the brain and ultimately resulting in fatality (CDC 2023). The primary mode of rabies transmission usually occurs via a bite from an infected animal. However, contracting rabies disease is also feasible through scratches, scrapes or open wounds that come into contact with saliva or other potentially infectious substances from a rabid animal (CDC 2023; Gold et al. 2021). Moreover, the virus enters the nerve pathways to reach the spinal cord and brain, resulting in inflammation and, eventually, fatality (CDC 2023). The earliest symptoms of rabies disease are similar to the signs of the flu, including weakness or pain, fever, or headache. There could also be soreness, prickling or itching at the bite site (CDC 2023; Gold et al. 2021). The symptoms lead to cognitive impairment, anxiety, confusion, and agitation (CDC 2023; Gold et al. 2021, 2020). The person may develop delirium, strange behavior, hallucinations, hydrophobia (fear of water), and insomnia as the disease worsens. The acute phase of the sickness usually lasts between 2 and 10 days. When clinical symptoms of rabies develop, the disease is generally fatal (CDC 2023; Gold et al. 2020). Wildlife such as raccoons, skunks, foxes, and bats are the main carriers of the virus and they can transfer the virus to other animals or humans (Musa et al. 2020). However, the majority of rabies cases in underdeveloped nations are caused by stray dogs or domestic dogs (Colombi et al. 2020; WHO 2023b).

The World Health Organization (WHO) reported that rabies has spread widely to more than 150 nations and territories (Colombi et al. 2020; Nagarajan & Rupprecht 2020). Thousands of people are killed by dog-mediated rabies annually, and most of the victims are children. Approximately 95% of human fatalities occur in Africa and Asia, where control measures for dog rabies are inadequately implemented, particularly in impoverished rural communities with limited or no access to appropriate post-exposure prophylaxis (PEP) (CDC 2023; Colombi et al. 2020; Nagarajan & Rupprecht 2020). In Africa, approximately 24,000 people die from rabies annually, which accounts for almost half of the total rabies deaths worldwide each year (CDC 2023; Nagarajan & Rupprecht 2020). Secondly, in Asia, India holds the top position for the highest number of human rabies deaths, contributing to approximately 59.9% of such fatalities globally. Following India, China is significantly affected by rabies, where it ranks third in terms of deaths among the 39 notifiable infectious diseases in the country. Poverty, lack of infrastructure, social and cultural hurdles, and restricted access to healthcare play a part in the spreading and persistence of rabies disease in these regions (WHO 2023b). The implementation of effective surveillance and control measures is essential to reducing the global burden of this deadly disease. Recognizing the severity of the issue, WHO and its partners have launched a global campaign to eliminate human deaths caused by dog-mediated rabies by 2030 (WHO 2023a, 2023b). The campaign aims to raise awareness about rabies disease, improve access to pre-and post-exposure prophylaxis (PEP) for vulnerable populations in remote rural areas, conduct mass dog vaccination programs, enhance rabies surveillance through veterinary services, expand oral vaccination programs for wildlife, promote dog registration, and carry out education campaigns in affected communities.

Mathematical modelling plays a crucial role in understanding and controlling rabies by providing insights into its transmission dynamics, control strategies, dog movements, and predicting possible future outbreaks. For instance, Renald, Kuznetsov and Kreppel (2020) formulated an Susceptible-Exposed-Infected-Vaccinated (SEIV) mathematical model to investigate the effectiveness of rabies control methods, that is, mass-culling and vaccination of dogs, in Arusha, Tanzania. They classified the dog population into three subgroups, namely domestic, stray, and Maasai dogs. They computed the basic reproduction number, R_0 (without considering any control strategies), and effective reproduction number, R_e (with control strategies). This model was fit to the survey data (i.e., dog bites, rabies death, and vaccination coverage) of the dog population in Arusha that spanned over five years which was recorded by a local non-governmental organization and the Ministry of Agriculture, Livestock Development and Fisheries of the United Republic of Tanzania. They found that R_e with the control strategies of vaccination and culling dogs combination is the most effective control measure to reduce the transmission rate of rabies. Hence, they employed R_e (vaccination and the culling) in proving the stability of the model. In addition, they proved that the disease-free and endemic equilibria achieved local and global asymptotic stabilities if $R_e < 1$ and R_e>1, respectively. They concluded that the most effective control method in combating the transmission of rabies disease in Arusha town is the combination of vaccination and culling dog strategy. Eze et al. (2020) employed Susceptible-Exposed-Infected-Recovered (SEIR) and SEIV models to study the transmission dynamics of rabies in dog and human populations, respectively. They computed the basic reproduction number and identified the existence of equilibrium points. Moreover, the stability of the model is analyzed. They discovered that the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$. Otherwise, the endemic equilibrium achieves local asymptotic stability. To validate the theoretical results, numerical simulations have been performed and they found that both results are well agreed. In addition, they discovered that, by employing pre-and post-exposure prophylaxis in the human population and strengthening the vaccination control strategy in the dog population, these control measures have shown a great influence in eliminating rabies disease. They highly recommended

that vaccination is one of the effective control strategies in preventing the spreading of rabies disease.

Abdulmajid and Hassan (2021) formulated a Susceptible-Infected-Vaccinated (SIV) delay differential equation model to investigate the rabies transmission dynamics between dogs and humans. This model integrated three interventions: vaccination of dogs and humans, and birth control of newborn puppies. They considered the incubation period of 2 months as the time delay in the model. They showed that the disease-free and endemic equilibria are locally and globally asymptotically stable if $R_0 < 1$ and $R_0 < 1$, respectively. They found that the rate of dog vaccination and birth control of puppies have the most significant impact on rabies transmission among dog and human populations. Moreover, they discovered, by increasing the time delay, the numbers of infected human and dogs are decreasing and eradication of rabies disease in 10 years time is possible if the annual birth of puppies (using immunocontraception) is decreasing and the vaccination rates are increasing. They concluded that the prevalence of rabies disease can be decreased in the future if the incubation time is increased with effective control strategies.

Rabies disease remains a significant public health problem in many parts of the world, particularly in developing countries, despite being preventable with vaccines, where access to healthcare and vaccines may be limited (WHO 2023a, 2023b). Every year, tens of thousands of people die from rabies, with the majority of cases occurring in Asia and Africa. The persistence of rabies disease is a multifaceted issue, which includes issues related to animal control, access to vaccines and healthcare, public education, and community engagement (WHO 2023a, 2023b). Thus, it is necessary for us to have a strong collaboration among government agencies, healthcare providers, veterinarians, and the general public to combat the disease. Not only that, mathematical models play a crucial role in our efforts to eliminate diseases and assist in assessing effective control strategies (Eze et al. 2020; Renald, Kuznetsov & Kreppel 2020). Hence, developing a robust mathematical model to examine the transmission dynamics of rabies disease will aid policy makers and public health officials in taking action to control and prevent the spread of this deadly disease effectively. In this study, we propose a mathematical model with vaccination control measures to investigate the transmission dynamics of rabies disease in dog populations. Moreover, we are looking forward to finding the basic reproduction number, identifying the steady states, analysing the stability, and conducting the numerical simulation of the model to depict the spreading dynamics of rabies disease in the dog population. The paper is structured as follows: In the next section, we introduce a Susceptible-Exposed-Infected-Vaccinated (SEIV) epidemic model, where the incubation period and vaccination intervention are considered, to examine the spreading of rabies disease in dog population. Moreover, we examine the well-posedness of the model by finding the positively invariant and attracting region, the existence of disease-free and endemic equilibria, and compute the basic reproduction number of the model. The stability analysis and numerical simulations of the model are discussed in the subsequent sections. Finally, in the last section, we provide concluding remarks and discussion.

MATHEMATICAL MODEL

We propose a deterministic model with a vaccination control strategy to investigate the transmission dynamics of rabies disease in dog population. The dog population is categorized into four subclasses: susceptible, exposed, infected, and vaccinated. These subclasses are denoted by $S(t)$, $E(t)$, $I(t)$ and $V(t)$, respectively, where t represents time. Our rabies model is defined as follows:

$$\begin{aligned} S'(t) &= \Lambda + \omega V - \beta SI - (\mu + \gamma)S, \\ E'(t) &= \beta SI - (\mu + \eta + \sigma)E, \\ I'(t) &= \sigma E - (\mu + \xi + \phi)I, \\ V'(t) &= \gamma S + \eta E - (\mu + \omega)V. \end{aligned} \tag{1}$$

The total population of dogs is $N(t) = S(t) + I(t) + E(t) + V(t)$, whereas the description of associated parameters of model (1) is listed in Table 1. There are some assumptions of our proposed model: the recruitment rate of dogs is constant and the vaccinated dogs will become susceptible when the loss of immunity occurs.

Next, we aim to determine in which the solutions of model (1) hold from both biological and mathematical perspectives. This involves identifying the region Γ where the solution of model (1) remains positive and bounded for all time $t \geq 0$, discussed in Lemma *1*.

Lemma 1 The set Γ:$\left\{(S, E, I, V) \in \mathbb{R}_+^4 | 0 < S + E + I + V \leq \frac{\Lambda}{\mu}\right\}$ is a positively invariant and attracting region for model (1).

Proof Let $N(t) = S(t) + I(t) + E(t) + V(t)$ be the total population of dogs. Then we obtain

$$\begin{aligned} N' &= \Lambda - \mu N - (\xi + \phi)I, \\ &\leq \Lambda - \mu N \quad \text{since } -(\xi + \phi)I \leq 0. \end{aligned} \tag{2}$$

Next, we solve Equation (2) by using the integrating factor and we have

$$N(t) \leq \frac{\Lambda}{\mu} + \left[N(0) - \frac{\Lambda}{\mu}\right] e^{-\mu t}.$$

TABLE 1. Descriptions of parameters in model (1)

Parameter	Description
Λ	Recruitment rate of dogs
ω	The rate of vaccinated dogs lose the vaccine-based immunity
β	Disease transmission rate from infected dogs to susceptible dogs
μ	Natural death rate of dogs
γ	The vaccination rate of susceptible dogs
ξ	Disease-related death rate of dogs
η	The vaccination rate of exposed dogs
σ	The progression rate from exposure to infected dogs
ϕ	Risk factor of clinical outcome of exposed dogs

If $N(0) \leq \frac{\Lambda}{\mu}$, we get $N(t) \leq \frac{\Lambda}{\mu}$. Thus, $N(t) \leq \frac{\Lambda}{\mu}$ is a positively invariant region of model (1).

To prove $N(t) \leq \frac{\Lambda}{\mu}$ is an attracting region, let $\Phi = \frac{\Lambda}{\mu}$ and suppose $N(t) \leq \frac{\Lambda}{\mu}$ From (2), we have

$$\begin{aligned} N'(t) &\leq \Lambda - \mu N, \\ &= \mu(\Phi - N), \\ &< 0 \quad \text{because} \quad N > \frac{\Lambda}{\mu} \Rightarrow N > \Phi \Rightarrow \Phi - N < 0. \end{aligned}$$

Therefore, $\Gamma := \left\{(S,E,I,V) \in \mathbb{R}^4_+ \mid 0 < S+E+I+V \leq \frac{\Lambda}{\mu}\right\}$ is a positively invariant and attracting region of model (1). ■

We find that the disease-free equilibrium of model (1) is $E_d = (S_0, E_0, I_0, V_0)$, where

$$S_0 = \frac{\Lambda(\mu+\omega)}{\mu(\mu+\omega+\gamma)}, \quad E_0 = I_0 = 0, \quad \text{and} \quad V_0 = \frac{\Lambda\gamma}{\mu(\mu+\omega+\gamma)},$$

whereas the endemic equilibrium is $E_e = S^*, E^*, I^*, V^*$ where

$$S^* = \frac{(\mu+\xi+\phi)(\mu+\eta+\sigma)}{\sigma\beta},$$

$$E^* = \frac{\Lambda\sigma\beta(\mu+\omega) - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)(\mu+\omega+\gamma)}{\sigma\beta[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)]},$$

$$I^* = \frac{\Lambda\sigma\beta(\mu+\omega) - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)(\mu+\omega+\gamma)}{\beta(\mu+\xi+\phi)[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)]} \quad \text{and}$$

$$V^* = \frac{\gamma(\mu+\sigma)(\mu+\xi+\phi)(\mu+\eta+\sigma)+\eta[\Lambda\sigma\beta - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)]}{\sigma\beta[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)]}.$$

The basic reproduction number of model (1) can be determined by applying the next generation matrix approach (Diekmann, Heesterbeek & Metz 1990; Van den Driessche & Watmough 2002), which yields the following expression:

$$R_0 = \frac{\Lambda\sigma\beta(\mu+\omega)}{\mu(\mu+\omega+\gamma)(\mu+\xi+\phi)(\mu+\eta+\sigma)}.$$

Next, we would like to determine whether the existence of E_e has biological meaning in the following theorem.

Theorem 1 The E_e of model (1) exists and unique if $R_0 < 1$.

Proof The existence of E_e only has biological meaning if $S^*, E^*, I^*, V^* > 0$.Thus,letusconsider$R_0 < 1$whichimpliesthat $\Lambda\sigma\beta(\mu+\omega) > \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)(\mu+\omega+\gamma)$ since all associated parameters are positive. Then, we obtain

$$I^* = \frac{\Lambda\sigma\beta(\mu+\omega) - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)(\mu+\omega+\gamma)}{\beta(\mu+\xi+\phi)[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)} > 0,$$

$$E^* = \frac{\Lambda\sigma\beta(\mu+\omega) - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)(\mu+\omega+\gamma)}{\sigma\beta[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)} > 0 \quad \text{and}$$

$$V^* = \frac{\gamma(\mu+\sigma)(\mu+\xi+\phi)(\mu+\eta+\sigma)+\eta[\Lambda\sigma\beta - \mu(\mu+\xi+\phi)(\mu+\eta+\sigma)]}{\sigma\beta[\mu(\mu+\eta+\sigma)+\omega(\mu+\sigma)} > 0$$

whenever $R_0 < 1$. Moreover, $S^* = \frac{(\mu+\xi+\phi)(\mu+\eta+\sigma)}{\sigma\beta}$, which is always positive since all associated parameters are positive. Hence, the E_e of model (1) only exists and unique if $R_0 < 1$.■

STABILITY ANALYSIS OF THE MODEL

This section focuses on the stability analysis of model (1), which is essential in gaining a deeper understanding of the behaviour of transmission dynamics of model (1). We would like to identify under which condition(s) disease-free (E_d) and endemic equilibria (E_e) are locally and globally asymptotically stable. To determine the local stability of $E_d$and E_e, the linearization approach is employed. Before we show the local stability of $E_d$and E_e, let us define the Jacobian matrix of model (1) as follows:

$$J = \begin{bmatrix} -(\beta I+\mu+\gamma) & 0 & -\beta S & \omega \\ \beta I & -(\mu+\eta+\sigma) & \beta S & 0 \\ 0 & \sigma & -(\mu+\xi+\phi) & 0 \\ \gamma & \eta & 0 & -(\mu+\omega) \end{bmatrix}.$$

Theorem 2 The E_d of model (1) is locally asymptotically stable if $R_0 < 1$.

Proof Let λ be the eigenvalue and I be a 4×4 identity matrix. Then, at $E_d$we would like to solve $\det(J(E_d) - \lambda I) = 0$ for λ, which is shown as follows:

$$(\mu+\lambda)(\mu+\omega+\gamma+\lambda)\left[(\mu+\eta+\sigma+\lambda)(\mu+\xi+\phi+\lambda)-\frac{\Lambda\sigma\beta(\mu+\omega)}{\mu(\mu+\omega+\gamma)}\right]=0. \quad (3)$$

Since all associated parameters are positive, the trivial solutions of (3) are as follows:

$$\lambda=-\mu<0 \quad \text{and} \quad \lambda=-(\mu+\omega+\gamma)<0.$$

However, the nontrivial solutions of (3), $(\mu+\eta+\sigma+\lambda)(\mu+\xi+\phi+\lambda)-\frac{\Lambda\sigma\beta(\mu+\omega)}{\mu(\mu+\omega+\gamma)}=0$, can be rewritten as

$$A\lambda^2+B\lambda+C=0$$

where

$$A=\mu(\mu+\omega+\gamma), \quad B=\mu(\mu+\omega+\gamma)[(\mu+\eta+\sigma)+(\mu+\xi+\phi)]$$

and

$$C=\mu(\mu+\omega+\gamma)(\mu+\xi+\phi)(\mu+\eta+\sigma)-\Lambda\sigma\beta(\mu+\omega).$$

Then, by applying the quadratic formula, we obtain

$$B^2-4AC=\mu^2(\mu+\omega+\gamma)^2[(\mu+\eta+\sigma)+(\mu+\xi+\phi)]^2+4\Lambda\sigma\beta\mu(\mu+\omega)(\mu+\omega+\gamma),$$

$$>\mu^2(\mu+\omega+\gamma)^2[\eta+\sigma-(\xi+\phi)]^2,$$

$$\sqrt{B^2-4AC}>\mu(\mu+\omega+\gamma)[\eta+\sigma-(\xi+\phi)],$$

$$-\sqrt{B^2-4AC}<-\mu(\mu+\omega+\gamma)[\eta+\sigma-(\xi+\phi)].$$

Thus, we have

$$\lambda_-=\frac{-B-\sqrt{B^2-4AC}}{2A}<-(\mu+\eta+\sigma)<0.$$

Moreover,

$$B^2-4AC=B^2+4\mu(\mu+\omega+\gamma)[\Lambda\sigma\beta(\mu+\omega)-\mu(\mu+\omega+\gamma)(\mu+\xi+\phi)(\mu+\eta+\sigma)],$$

$$<B^2 \quad \text{if} \quad R_0<1,$$

$$\sqrt{B^2-4AC}<B.$$

$$\text{Then, we get } \lambda_+=\frac{-B+\sqrt{B^2-4AC}}{2A}<0.$$

Therefore, E_d achieves local asymptotic stability whenever $R_0<1$. ■

Subsequently, we will examine the local stability of E_e in the following theorem.

Theorem 3 The E_e of model (1) is locally asymptotically stable if $R_0<1$.

Proof By using a similar approach as in Theorem 2, we would like to solve $\det(J(E_e)-\lambda I=0)\ for\ \lambda$, where λ denotes the eigenvalue and I denotes a 4×4 identity matrix as follows:

$$\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0, \quad (4)$$

where

$$a_1=(\beta I+\mu+\gamma)+(\mu+\omega)+(\mu+\xi+\phi)+(\mu+\eta+\sigma),$$

$$a_2=\mu\gamma+(\mu+\omega)(\mu+\beta I)+(2\mu+\omega+\gamma+\beta I)[(\mu+\xi+\phi)+(\mu+\eta+\sigma)],$$

$$a_3=[\mu\gamma+(\mu+\omega)(\mu+\beta I)][(\mu+\xi+\phi)+(\mu+\eta+\sigma)]+\beta I(\mu+\xi+\phi)(\mu+\eta+\sigma)$$

and

$$a_4=\beta I(\mu+\xi+\phi)[\omega(\mu+\sigma)+\mu(\mu+\eta+\sigma)].$$

We apply the Routh-Hurwitz Criterion (DeJesus & Kaufman 1987; Ho, Datta & Bhattacharyya 1998; Khatwani 1981) to solve Equation (4) for eigenvalue λ. We discover that $a_1,a_2,a_3,a_4>0$ and $a_3(a_1a_2-a_3)-a_1^2a_4$ if $R_0<1$. By Routh-Hurwitz Criterion, we conclude that all eigenvalues of (3) are negative or have negative real parts. Therefore, E_e attains local asymptotic stability whenever $R_0<1$. ■

Next, we would like to explore the global stability of E_d and E_e. To demonstrate the global stability of E_d, we employ the comparison principle (Wolkowicz 1996) and the theory of asymptotic autonomous system (Thieme 1992). Meanwhile, to prove the global stability of E_e, we adopt the geometric approach introduced by Li and Muldowney (1996).

Theorem 4 The E_d of model (1) achieves global asymptotic stability whenever $R_0<1$ and $\eta\leq\gamma$.

Proof Since $S+E+I+V\to\frac{\Lambda}{\mu}$ as $t\to\infty$, we consider the last equation of model (1) is an asymptotically autonomous differential equation with the limit equation

$$\frac{dV}{dt}=\gamma\left(\frac{\Lambda}{\mu}-E-I-V\right)+\eta E-(\mu+\omega)V, \quad (5)$$

$$\leq\frac{\Lambda\gamma}{\mu}-(\mu+\omega+\gamma)V, \quad \text{if} \quad \eta\leq\gamma$$

since all associated variables and parameters are positive.

Then, by using the integrating factor, we get

$$\frac{d}{dt}\left[Ve^{(\mu+\omega+\gamma)t}\right]\leq\frac{\Lambda\gamma}{\mu}e^{(\mu+\omega+\gamma)t},$$

$$V(t)e^{(\mu+\omega+\gamma)t} \le V(0) + \frac{\Lambda\gamma}{\mu(\mu+\omega+\gamma)}\left[e^{(\mu+\omega+\gamma)t} - 1\right],$$

$$V(t) \le [V(0) - V_0]e^{-(\mu+\omega+\gamma)t} + V_0, \quad \text{where} \quad V_0 = \frac{\Lambda\gamma}{\mu(\mu+\omega+\gamma)}$$

$$V(t) \le V_0 + V(0)e^{-(\mu+\omega+\gamma)t}.$$

Let $\varepsilon = \frac{(\mu+\gamma)\varepsilon_1}{\omega}$. For every $\varepsilon > 0$, there exists a $t_1 > 0$ such that $V \le V_0 + \varepsilon$ for all $t > t_1$. Then, for all $t > t_1$,

$$\begin{aligned} \frac{dS}{dt} &= \Lambda + \omega V - \beta SI - (\mu+\gamma)S, \\ &\le \Lambda + \omega(V_0 + \varepsilon) - (\mu+\gamma)S. \end{aligned} \tag{6}$$

By using the integrating factor, we obtain

$$\frac{d}{dt}\left[Se^{(\mu+\gamma)t}\right] \le [\Lambda + \omega(V_0+\varepsilon)]e^{(\mu+\gamma)t},$$

$$S(t)e^{(\mu+\gamma)t} \le S(0) + \frac{\Lambda + \omega(V_0+\varepsilon)}{\mu+\gamma}\left[e^{(\mu+\gamma)t} - 1\right],$$

$$S(t) \le \left[S(0) - \frac{\Lambda + \omega(V_0+\varepsilon)}{\mu+\gamma}\right]e^{-(\mu+\gamma)t} + \frac{\Lambda + \omega(V_0+\varepsilon)}{\mu+\gamma},$$

$$\le \frac{\Lambda + \omega(V_0+\varepsilon)}{\mu+\gamma} + S(0)e^{-(\mu+\gamma)t},$$

$$= \frac{\Lambda + \omega\left(\frac{\Lambda}{\mu} - S_0\right)}{\mu+\gamma} + \frac{\omega\varepsilon}{\mu+\gamma} + S(0)e^{-(\mu+\gamma)t},$$

$$= S_0 + \frac{\omega\varepsilon}{\mu+\gamma} + S(0)e^{-(\mu+\gamma)t}.$$

Let $\varepsilon_1 = \frac{\omega\varepsilon}{\mu+\gamma}$. For every $\varepsilon_1 > 0$, there exists a $t_2 > 0$ such that $S \le S_0 + \varepsilon_1$ for all $t > t_2 > t_1$. Thus, we obtain the basic reproduction number of model (1) as follows:

$$R_0 = \frac{\sigma\beta S}{(\mu+\xi+\phi)(\mu+\eta+\sigma)} \le \frac{\sigma\beta(S_0+\varepsilon_1)}{(\mu+\xi+\phi)(\mu+\eta+\sigma)} = R_0 + \frac{\sigma\beta\varepsilon_1}{(\mu+\xi+\phi)(\mu+\eta+\sigma)}.$$

Next, we would like to consider the following system.

$$\begin{aligned} \frac{dE}{dt} &\le \beta(S_0+\varepsilon_1)I - (\mu+\eta+\sigma)E, \quad \text{since} \quad S \le S_0 + \varepsilon_1 \\ \frac{dI}{dt} &= \sigma E - (\mu+\xi+\phi)I. \end{aligned} \tag{7}$$

The corresponding linear system of (7) is given as follows:

$$\begin{aligned} \frac{d\hat{E}}{dt} &= \beta(S_0+\varepsilon_1)\hat{I} - (\mu+\eta+\sigma)\hat{E}, \\ \frac{d\hat{I}}{dt} &= \sigma\hat{E} - (\mu+\xi+\phi)\hat{I}. \end{aligned} \tag{8}$$

Let $\hat{\lambda}$ denote the eigenvalue and the characteristic Equation of (8) is defined as follows:

$$A\hat{\lambda}^2 + B\hat{\lambda} + C = 0\,,$$

where $A = 1, \quad B = (\mu+\eta+\sigma) + (\mu+\xi+\phi)$ and

$C = (\mu+\xi+\phi)(\mu+\eta+\sigma) - \sigma\beta(S_0+\varepsilon_1).$

$$B^2 - 4AC = B^2 + 4[\sigma\beta(S_0+\varepsilon_1) - (\mu+\xi+\phi)(\mu+\eta+\sigma)],$$

$$< B^2, \quad since \quad R_0 + \frac{\sigma\beta\varepsilon_1}{(\mu+\xi+\phi)(\mu+\eta+\sigma)} < 1$$

$$\sqrt{B^2 - 4AC} < B.$$

By using the quadratic formula, we get

$$\hat{\lambda}_+ = \frac{-B + \sqrt{B^2 - 4AC}}{2A} < 0.$$

Moreover, we have

$$\begin{aligned} B^2 - 4AC &= (\mu+\eta+\sigma)^2 - 2(\mu+\xi+\phi)(\mu+\eta+\sigma) \\ &\quad + (\mu+\xi+\phi)^2 + 4\sigma\beta\,(S_0+\varepsilon_1), \\ &> [\eta+\sigma-(\xi+\phi)]^2, \\ \sqrt{B^2 - 4AC} &< \eta+\sigma-(\xi+\phi) \end{aligned}$$

and

$$\hat{\lambda}_- = \frac{-B - \sqrt{B^2 - 4AC}}{2A} < -(\mu+\eta+\sigma) < 0.$$

Therefore, the general solution of Equation (8) is

$$x(t) = c_1u_1e^{\hat{\lambda}_+t} + c_2u_2e^{\hat{\lambda}_-t}$$

$$x(t) = c_1u_1e^{\hat{\lambda}_+t} + c_2u_2e^{\hat{\lambda}_-t}$$

where $x(t) = [\hat{E}(t),\ \hat{I}(t)]$, c_1 and c_2 are arbitrary constants, and u_1 and u_2 are the corresponding eigenvectors of eigenvalues $\hat{\lambda}_+$ and $\hat{\lambda}_-$, respectively. Furthermore, $x(t) \to 0$as $t \to \infty$. By applying the comparison principle (Wolkowicz 1996), $E, I \to 0$ as $t \to \infty$. Consequently, by the theory of asymptotic autonomous system (Thieme 1992), we obtain $V \to V_0$ and $S \to S_0$ as $t \to \infty$ from (5) and (6), respectively. Therefore, we conclude that E_d is globally attractive if $R_0 < 1$ and $\eta \le \gamma$.

In addition, we apply the geometric approach proposed by Li and Muldowney (1996) to prove the global stability of the endemic equilibrium (E_e) of the model (1). Before showing the proof, we provide a brief explanation of the geometric approach, which is summarized from Li and Muldowney (1996).

Consider the autonomous ordinary differential equation as follows:

$$\frac{dx}{dt} = f(x), \tag{9}$$

where $x \mapsto f(x) \in \mathbb{R}^n$ is a C^1 function for x in an open set $M \subset \mathbb{R}^n$. Here, x^*, x_0, and $x(t, x_0)$ represent an equilibrium point, initial point, and solution of (9) such that $x(0, x_0) = x_0$, respectively.
Assume the following hypotheses hold:

(A1) There exists a compact absorbing set $\widetilde{M} \subset M$.

(A2) Equation (9) has a unique equilibrium x^* in M.

Let $x \mapsto P(x)$ be a $\binom{n}{2} \times \binom{n}{2}$ matrix-valued function that is C^1 for $x \in M$. Assume that $P^{-1}(x)$ exists and is continuous for $x \in \widetilde{M}$. A quantity q is defined as follows:

$$q = \lim_{t\to\infty} sup \sup_{x_0 \in \widetilde{M}} \frac{1}{t}\int_0^t \tilde{\sigma}\left(B\left(x(0,x_0)\right)\right) d\theta,$$

where $B = P_f P^{-1} + PJ^{[2]}P^{-1}$, the P_f is obtained by replacing each entry p of $P$by its derivative in the direction of f, and $\tilde{\sigma}(B)$ is the Lozinskiĭ measure of B with respect to a vector norm $\|\cdot\|$ in $\mathbb{R}^{\binom{n}{2}}$ (refer to Coppel & Vleck 1968), that is,

$$\tilde{\sigma}(B) = \lim_{h\to 0^+} \frac{\|I + h(B)\| - 1}{h}.$$

The following global stability result is proved in (Li & Muldowney 1996).
Lemma 2 (Li & Muldowney 1996) Assume that M is simply connected, and the hypotheses (A1) and (A2) hold. Then the unique equilibrium x^* of (9) is globally stable in M if $q < 0$.

Now we would like to apply Lemma 2 to prove the global stability of E_e.
Theorem 5 E_e of model (1) achieves global asymptotic stability if $R_0 < 1, \gamma < \eta + \sigma$

and

$$\gamma > \max\left\{\frac{\Lambda\beta}{\mu} - (2\mu + \eta + \sigma),\ \sigma - (2\mu + \xi + \phi + \omega)\right\}.$$

Proof Since $S + E + I + V \to \frac{\Lambda}{\mu}$ as $t \to \infty$, model (1) is a three-dimensional asymptotically autonomous system with the limit system as follows:

$$\begin{aligned} S' &= \Lambda + \omega\left(\frac{\Lambda}{\mu} - S - E - I\right) - \beta SI - (\mu + \gamma), \\ E' &= \beta SI - (\mu + \eta + \sigma)E, \\ I' &= \sigma E - (\mu + \xi + \phi)I. \end{aligned} \tag{10}$$

The Jacobian matrix of Equation (10) is

$$\hat{J} = \begin{bmatrix} -(\beta I + \mu + \gamma + \omega) & -\omega & -(\beta S + \omega) \\ \beta I & -(\mu + \eta + \sigma) & \beta S \\ 0 & \sigma & -(\mu + \xi + \phi) \end{bmatrix}.$$

From Theorem 1, it has been shown that E_e exists and unique whenever $R_0 < 1$. In order to show that E_e achieves global stability in Γ (see Lemma 1), from Lemma 2, the following conditions are required.

a. The existence of a compact absorbing set in the interior of Γ (i.e., hypothesis (A1)).
b. The uniqueness of E_e in $\tilde{\Gamma}$, the interior of Γ (i.e., hypothesis (A2)).
c. The requirement of Bendixson criterion (i.e., $q < 0$).

Under the assumption $R_0 < 1$, model (1) satisfies the hypotheses (A1) and (A2). In fact, E_d is unstable if $R_0 < 1$ and this implies the uniform persistence (Freedman, Ruan & Tang 1994). That is, there exists a constant $c > 0$ such that $\liminf_{t\to\infty} S(t) > c$, and $\liminf_{t\to\infty} V(t) > c$.
The boundedness of Γ and uniform persistence imply that model (1) has a compact absorbing set in $\tilde{\Gamma}$ (Hutson & Schmit 1992). Thus, hypothesis (A1) is satisfied. Moreover, from Theorem 1, it has been shown that $E_e \in \tilde{\Gamma}$ exists and unique whenever $R_0 < 1$, which has verified hypothesis (A2).
Now we remain to identify the condition of Bendixcon criterion, i.e, q. The second additive compound matrix $J^{[2]}$ of $\hat{J}$ is given by

$$J^{[2]} = \begin{bmatrix} -(j_1 + j_2) & \beta S & \beta S + \omega \\ \sigma & -(j_1 + j_3) & -\omega \\ 0 & \beta I & -(j_2 + j_3) \end{bmatrix},$$

where $j_1 = (\beta I + \mu + \gamma + \omega), j_2 = (\mu + \eta + \sigma)$ and $j_3 = (\mu + \xi + \phi)$.

Let $P = P(S, E, I) = \text{diag}\left\{\frac{S}{I}, \frac{S}{I}, \frac{S}{I}\right\}$. Then,

$P_f P^{-1} = \text{diag}\left\{\frac{S'}{S} - \frac{I'}{I}, \frac{S'}{S} - \frac{I'}{I}, \frac{S'}{S} - \frac{I'}{I}\right\}$ and the matrix

$B = P_f P^{-1} + PJ^{[2]}P^{-1}$ is given as follows:

$$B = \begin{bmatrix} \frac{S'}{S} - \frac{I'}{I} - (j_1 + j_2) & \beta S & \beta S + \omega \\ \sigma & \frac{S'}{S} - \frac{I'}{I} - (j_1 + j_3) & -\omega \\ 0 & \beta I & \frac{S'}{S} - \frac{I'}{I} - (j_2 + j_3) \end{bmatrix}.$$

Let $z = (z_1, z_2, z_3)$ be a vector in $\mathbb{R}^3 \cong \mathbb{R}^{\binom{3}{2}}$ and we choose a norm in $\mathbb{R}^3$ as $|(z_1, z_2, z_3)| = \max\{|z_1|, |z_2| + |z_3|\}$. In addition, we let $\tilde{\sigma}(B)$ be the Lozinskiĭ measure of B with respect to this norm. Then, by applying the logarithmic norm method as described in Martin Jr. (1974), we have

$$\tilde{\sigma}(B) \le \max\{g_1, g_2\}$$

where, for $i = 1,2,3$, $g_i = \tilde{\sigma}(B_{ii}) + \sum_{j\neq i,\ j=1}^{3} |B_{ij}|$, $|B_{ij}|$ are matrix norms with respect to the l_1 vector norm, and $\tilde{\sigma}$ denotes the Lozinskii measure with respect to the l_1 norm (refer to Li and Muldowney (1996) and Martin Jr. (1974) for further details). Thus, we have

$$\tilde{\sigma}(B_{11}) = \frac{S'}{S} - \frac{I'}{I} - (j_1 + j_2), \quad |B_{12}| = \beta S + \omega, \quad |B_{21}| = \sigma \quad \text{and}$$

$$\tilde{\sigma}(B_{22}) = \max\left\{\frac{S'}{S} - \frac{I'}{I} - [(j_1 + j_3) + \beta I], \quad \frac{S'}{S} - \frac{I'}{I} - [(j_2 + j_3) + \omega]\right\},$$

$$= \frac{S'}{S} - \frac{I'}{I} - (2\mu + \xi + \phi + \omega) + \max\{-\gamma, -(\eta + \sigma)\}.$$

Thus, we have

$$g_1 = \tilde{\sigma}(B_{11}) + |B_{12}|$$

$$= \frac{S'}{S} - \frac{I'}{I} - [(\beta I + \mu + \gamma) + (\mu + \eta + \sigma)] + \beta S,$$

$$\leq \frac{S'}{S} - \frac{I'}{I} - (2\mu + \gamma + \eta + \sigma) + \frac{\Lambda\beta}{\mu}, \quad \text{since} \quad S \leq N \leq \frac{\Lambda\beta}{\mu}.$$

$$g_2 = |B_{21}| + \widetilde{\sigma_1}(B_{22})$$

$$= \frac{S'}{S} - \frac{I'}{I} - (2\mu + \xi + \phi + \omega) + \sigma + \max\{-\gamma, -(\eta + \sigma)\},$$

Let $\tilde{a} = \min\left\{2\mu + \gamma + \eta + \sigma - \frac{\Lambda\beta}{\mu}, \ 2\mu + \xi + \phi + \omega + \gamma - \sigma\right\}$. Suppose $\gamma > \max\left\{\frac{\Lambda\beta}{\mu} - (2\mu + \eta + \sigma), \ \sigma - (2\mu + \xi + \phi + \omega)\right\}$. Thus, we have $\tilde{a} > 0$.

Then

$$g_1 \leq \frac{S'}{S} - \frac{I'}{I} - \tilde{a}, \quad \text{and} \quad g_2 = \frac{S'}{S} - \frac{I'}{I} - \tilde{a} \quad \text{if} \quad \gamma < \eta + \sigma.$$

For the solution $(S(t), E(t), I(t), V(t))$ of model (1) with arbitrary initial condition in $\tilde{\Gamma}$, we get

$$\frac{1}{t}\int_0^t g_1 \, d\theta, \quad \frac{1}{t}\int_0^t g_2 \, d\theta \leq -\tilde{a} + \frac{1}{t}\left\{\ln\left[\frac{S(t)}{S(0)}\right] - \ln\left[\frac{I(t)}{I(0)}\right]\right\}.$$

Let $x_0 = (S(0), E(0), I(0), V(0)) \in \tilde{\Gamma}$ and $\tilde{\sigma}(B) \leq \max\{g_1, g_2\}$. Thus, we obtain

$$\frac{1}{t}\int_0^t \tilde{\sigma}(B) \, d\theta \leq \max\left\{-\tilde{a} + \frac{1}{t}\left(\ln\left[\frac{S(t)}{S(0)}\right] - \ln\left[\frac{I(t)}{I(0)}\right]\right)\right\},$$

for all $t > 0$. Hence

$$\limsup_{t\to\infty} \sup_{x_0 \in \tilde{\Gamma}} \frac{1}{t}\int_0^t \tilde{\sigma}(B) \, d\theta \leq -\tilde{a} < 0 \quad \text{since} \quad \tilde{a} > 0.$$

Therefore, E_e achieves global stability.

NUMERICAL SIMULATIONS OF THE MODEL

In this section, we perform several numerical simulations of model (1) to illustrate the transmission dynamics of rabies disease in the dog population and validate the stability results of model (1). The numerical simulations of model (1) are conducted using the parameter values as stated in Table 2. The initial and equilibrium points of model (1) are represented by symbols ■ and ●, respectively.

We can see that, in Figure 1, all the trajectories of model (1) with arbitrary initial conditions are converging to the disease-free equilibrium, E_d, as $t \to \infty$ whenever the conditions $R_0 < 1$ and $\eta \leq \gamma$ as in Theorem 4 are satisfied. This result suggests that disease elimination is

TABLE 2. Parameter values to conduct the numerical simulation of equilibria E_d and E_e

Parameter	Parametervalue for E_d	Source	Parametervalue for E_e	Source	Unit
Λ	325	Bryce (2021)	325	Bryce (2021)	$\frac{\text{individual}}{\text{year}}$
β	0.00074	Assumed	0.0014	Assumed	$\frac{1}{\text{individual (year)}}$
μ	0.0833	Lv et al. (2023)	0.0833	Lv et al. (2023)	year $^{-1}$
σ	6	Ruan (2017)	3.8	Assumed	year $^{-1}$
ξ	1	Ruan (2017)	1	Ruan (2017)	year $^{-1}$
ω	0.5	Huang et al. (2018)	1	Ruan (2017)	year $^{-1}$
γ	0.5	Bornaa, Seidu & Daabo (2020)	0.9	Assumed	year $^{-1}$
η	0.09	Ruan (2017)	0.7	Assumed	year $^{-1}$
ϕ	0.6	Bornaa, Seidu & Daabo (2020)	0.8	Assumed	year $^{-1}$

likely to occur. Thus, by reducing the R_0 value such that it is less than the unity and by increasing the vaccination rate of susceptible dogs, the rabies disease is likely to die off equilibrium, E_d, whenever $R_0 < 1$ and $\eta \leq \gamma$

Moreover, from Figure 2, we observe that all the trajectories with arbitrary initial conditions are converging to the endemic equilibrium of model (1), i.e., $E_e$as t is sufficiently large if $R_0 < 1$ and all the conditions as stated in Theorem 5 are satisfied. This illustrates thatE_e, of model (1) achieves global asymptotic stability, which verifies the theoretical result of Theorem 5. In addition, this result also implies that the rabies disease persists in the dog population.

equilibrium E_e as $t \to \infty$ if the conditions of Theorem 5 are fulfilled

DISCUSSION AND CONCLUSION

In this paper, we considered an SEIV model with vaccination control strategy to examine the transmission dynamics of rabies disease in dog populations. This model is governed by a set of nonlinear ordinary differential equations. To ensure our proposed model (1) is well-posed, we identified the existence of positively invariant and attracting region, Γ(see

Lemma *1*). Moreover, we computed the basic reproduction number (R_0), identified the existence of disease-free and endemic equilibria, analyzed the stability of the model, and performed numerical simulations to depict the transmission dynamics of model (1) and validated the theoretical results. We found that by increasing the vaccination rate of susceptible dogs and reducing the R_0value such that it is below the unity, the rabies disease in dog population is

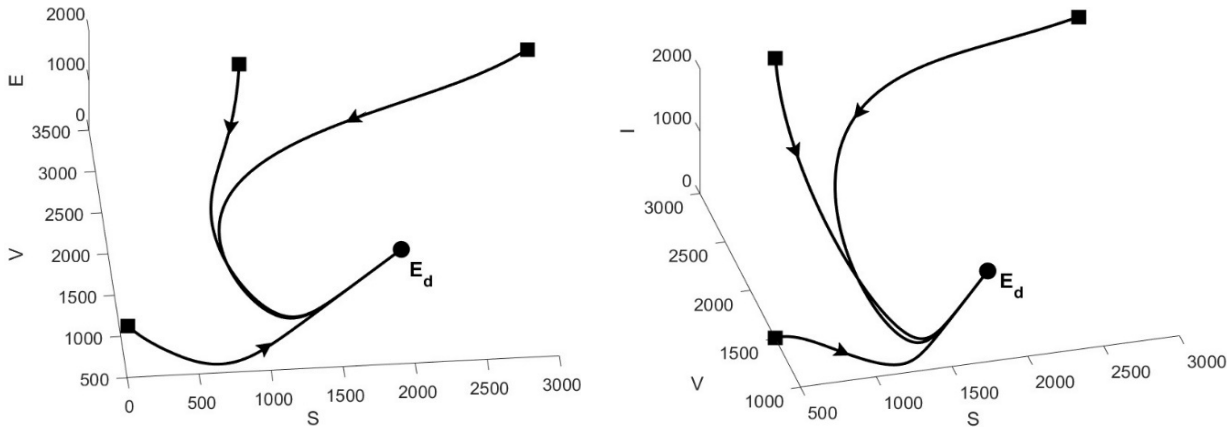

FIGURE 1. The transmission dynamics of rabies disease in dog population where all the solutions of model (1) with arbitrary initial conditions are approaching disease-free equilibrium E_d, whenever $R_0 < 1$ and $\eta \leq \gamma$

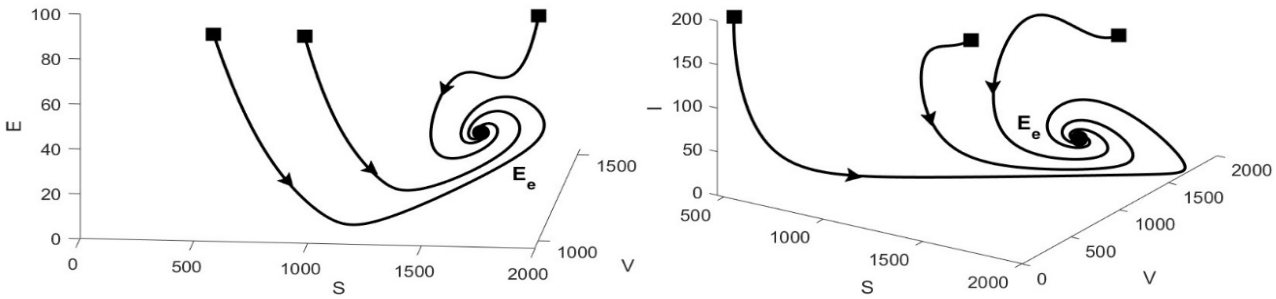

FIGURE 2. The transmission dynamics of rabies disease in dog population where all the trajectories of model (1) with arbitrary initial conditions are approaching endemic equilibrium E_e as $t \to \infty$ if the conditions of Theorem 5 are fulfilled

likely to die off. In addition, dog monitoring, surveillance, and public education programs are important to raise awareness about the dangers of rabies disease and educate communities on how to prevent themselves from getting infected, which is, ultimately leads to a reduction in the number of infected cases.

Furthermore, it is important to note that our model has some limitations. We assumed that the dog recruitment rate was constant, and the vaccinated dogs would become susceptible when the immunity declined. Moreover, we would like to suggest some insights for future work. For instance, it is important for us to know the impact of different vaccination strategies for both animal and human populations, by considering factors such as vaccine coverage, accessibility, and the effectiveness of the vaccines. Besides that, the temporal aspects of rabies outbreaks, which include seasonality and potential for periodic or episodic occurrences, and accounting for uncertainties in parameters are crucial for us to be more prepared if there is an outbreak occurs. Hence, future researchers could incorporate these factors into the model to improve its accuracy in predicting and controlling the transmission of rabies disease.

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