Review Article

Mathematical Analysis of Rabies Infection

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A mathematical model is proposed to study the dynamics of the transmission of rabies, incorporating predation of dogs by humans. The model is shown to have a unique disease-free equilibrium which is globally asymptotically stable whenever $\mathscr{R}_0 \leq 1$. Local sensitivity analysis suggests that the disease can be controlled through reducing contact with infected dogs, increasing immunization of dogs, screening recruited dogs, culling of infected dogs, and use of dog meat as a delicacy.

1. Introduction

Rabies is a zoonotic viral disease that is usually transmitted by an infected animal (such as foxes, raccoons, cats, coyotes, bats, skunks, and dogs) through bites or scratch that introduces the virus into the blood of another animal or a human [1, 2]. Once the virus enters the body, it either migrates directly to the brain via the peripheral nervous system to replicate there or stays in the muscles to replicate before migrating to the brain via the neuromuscular junctions. The infection becomes fatal upon reaching the brain since it produces acute inflammations of the brain, leading to coma and eventually death. The infection usually goes through five distinct phases of incubation (3 to 12 weeks), prodome (2 to 10 days, with worsening symptoms over time), acute neurologic period, coma, and death. Early symptoms of rabies infection are general body weakness, fever, and headache, which are also peculiar with common flu and other viral diseases

[3]. Rabies is preventable and treatment is possible at early stages of infection. Antirabies vaccination of all pets and domestic animals and humans, screening of imported animals, and awareness creation are some pre-exposure prophylaxis strategies that governments can embark on to prevent rabies. Individuals can also prevent the disease by vaccinating their pets and keeping away from possible infection sources. Postexposure prophylaxis strategies including washing the bitten area with soapy water for some time and administering a series of shots of rabies immune globulin at the early stages are often effective.

All mammals are possible reservoirs of the rabies virus, but small rodents such as rats and rabbits are less likely to cause transfers. A survey conducted in Ghana by Addy [4] revealed that, of 1,514 exposed domestic animals examined, dogs, cats, and cattle are responsible for 98.7%, 0.07%, and 0.70% infections, respectively. Rural areas of Southeast Asia and Africa bear the brunt of rabies

with tens of thousands contracting the disease annually. Most of these infections are caused by dog bites due to the fact that dogs are closest in contact with humans since they are used as pets, as guards, for hunting purposes, and even as delicacies.

The impact of mathematics continues to increase over the years as more evidence is found to support the fact that mathematical modeling helps to increase our understanding of the dynamics of infectious diseases. This has led to increased use of mathematics to model many infectious disease (see [5–18], and references therein).

Zoonotic diseases have received their fair share of attention by mathematicians. Specifically, Wang and Lou [19] developed an ODE model to study the impact of combinations of culling and vaccination on the control of rabies transmission and observed that vaccination alone was a better strategy whilst culling was worst in the control of rabies transmission. The work of Wang and Lou [19] is however contradicted by that of Carroll et al. [20] that suggested that culling is more effective than vaccination, arguing that adding immunocontraceptives on animal populations could help improve the effectiveness of vaccination. To analyze the strategies for optimal distribution of vaccine baits in order to minimize the spread of the disease and the cost of carrying out the control, Ding et al. [21] formulated a model to describe rabies in raccoons with discrete time and spatial features. Hou et al. [12] suggested that the control of rabies needs to include awareness creation about rabies, increased domestic dog vaccination, and reduction in the number of stray dogs. Asamoah et al. [5] also suggested that vaccination of pets and use of preexposure and postexposure prophylaxis could help in the control of rabies spread. In China and some parts of Africa (for example, the Upper East and West regions of Ghana), dog meat is a delicacy. It is noteworthy that while so much research has been carried out on the transmission and control of rabies, the impact of predation of dogs by humans has not been studied in the transmission of rabies. This current work seeks to incorporate consumption of dog meat by humans into a dog-human rabies model.

The rest of the paper is organized as follows. In Section 2, the mathematical model under consideration is derived, while Section 3 discusses some basic qualitative properties of the model. In Section 4, numerical simulation of the model is carried out to study the impact of various factors on the transmission of rabies. The findings and conclusions are presented in Section 5.

2. Model Formulation

We consider the transmission of rabies from a dog population to a dog-predator human population. Each of the dog and human populations is divided into three compartments of Susceptibles (S), Exposed (E), and Infected (I). We use subscripts d and h to denote dogs and humans, respectively, so that Susceptible, Exposed, and Infected dogs are represented by S_d, E_d, and I_d, respectively, while Susceptible, Exposed, and Infected humans are represented by S_h, E_h, and I_h, respectively. The total dog and human populations are therefore given by $W_d(t) = S_d(t) + E_d(t) + I_d(t)$ and $W_h(t) = S_h(t) + S_h(t) + I_h(t)$. Human-to-human transmission of the rabies virus is assumed to be nonexistent or insignificant so that only direct dog-to-dog and dog-to-human transmission is considered. We assume that all recruitments of dogs and humans are made into the susceptible classes $S_d(t)$ and $S_h(t)$, at constant rates P_d and P_h, respectively. Dogs and humans contract the disease through effective contact with infected dogs, with transmission probabilities β_d and β_h, respectively. Rabies transmission is assumed to be reduced through vaccination of susceptible dogs and humans at rates δ_d and δ_h, respectively. Upon effective contact with infected dogs, dogs and humans are moved to the exposed classes before being moved (if postexposure prophylaxis is ineffective) to the infected classes at rates β_{DD} and β_H for dogs and humans, respectively. A successful treatment at rate, θ, of exposed and infected humans is assumed while successful culling at rate C_d is applied to exposed and infected dogs. Dogs and humans are assumed to suffer rabies-induced death at rates σ_d and σ_h, respectively. Parameters μ_d and μ_h are taken to be the natural death rates in dogs and humans, respectively. We assume that dogs suffer predation from humans at rate γ_1. Since dogs in the infected class are assumed to be culled, we exclude the infected dog population from the predation.

With these assumptions, the mathematical model describing the dynamics of dog-human rabies transmission is represented by the set of differential equations in equation (1). Table 1 shows the descriptions of the parameters used in the model.

TABLE 1: Description of parameters of the model.

Parameter	Description
P_d	Recruitment into the susceptible dog population (birth and immigration)
P_h	Recruitment into the susceptible human population (birth and immigration)
γ_1	Rate of human predation of dogs
$\gamma \ll \gamma_1$	Rate at which consumed dogs are converted into susceptible humans
β_d	Rate of disease transmission within the dog population
$\beta_{DD} < \sigma_d$	Rate at which exposed dogs develop clinical rabies
β_h	Rate of dog-to-human disease transmission
$\beta_H < \sigma_h$	Rate at which exposed humans develop clinical rabies
$\mu_d < \beta_{DD}$	Natural death rate of dogs
μ_h	Natural death rate of humans
C_d	Death rate of dogs due to culling
σ_d	Disease induce death rate within the dog population
σ_h	Disease-induced death rate within the human population
δ_d	Rate of immunization of dogs
δ_h	Rate of immunization of humans
θ	Rate of treatment of infected human population

$$
\begin{cases}
\dfrac{dS_d}{dt} = P_d - \beta_d S_d (E_d + I_d)(1 - \delta_d) - (\mu_d + \gamma_1(S_h + E_h))S_d, \\[2mm]
\dfrac{dE_d}{dt} = \beta_d S_d (E_d + I_d)(1 - \delta_d) - (\mu_d + \gamma_1(S_h + E_h) + C_d + \beta_{DD})E_d, \\[2mm]
\dfrac{dI_d}{dt} = \beta_{DD} E_d - (\mu_d + \gamma_1(S_h + E_h) + \sigma_d + C_d)I_d, \\[2mm]
\dfrac{dS_h}{dt} = P_h + \gamma(S_h + E_h)(S_d + E_d + I_d) + \theta(I_h + E_h) - [\beta_h(I_d + E_d)(1 - \delta_h) + \mu_h]S_h, \\[2mm]
\dfrac{dE_h}{dt} = \beta_h S_h (I_d + E_d)(1 - \delta_h) - (\theta + \mu_h + \beta_H)E_h, \\[2mm]
\dfrac{dI_h}{dt} = \beta_H E_h - (\mu_h + \sigma_h + \theta)I_h, \\[2mm]
\text{with initial conditions} \quad S_d(0) \geq 0, E_d(0) \geq 0, I_d(0) \geq 0, \\[2mm]
\qquad\qquad\qquad\qquad\quad S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0.
\end{cases}
\tag{1}
$$

For convenience, we make the following substitutions

$$
\begin{aligned}
\psi_1 &= \beta_d(1 - \delta_d), \\
\psi_2 &= \beta_h(1 - \delta_h), \\
\psi_3 &= \mu_d + C_d + \beta_{DD} \\
\psi_4 &= \mu_d + \sigma_d + CC_d, \\
\psi_5 &= \theta + \mu_h + \beta_H, \\
\psi_6 &= \mu_h + \sigma_h + \theta.
\end{aligned}
\tag{2}
$$

In the next section, we discuss some basic properties of the model.

3. Basic Properties of the Model

3.1. Positivity of Solutions. Model (1) is an epidemiological model, and hence, it is necessary that the associated population sizes be positive. Model (1) should be considered in a feasible region where such property (nonnegative) is preserved. This is provided in Theorem 1.

Theorem 1. *If positive conditions initial conditions are provided for (1), then all its solutions remain positive for t > 0.*

Proof. From the first equation, if at some point $t > 0$, we have $S_d(t) = 0$ and then, we have $(dS_d/dt) = P_d > 0$. This shows that $S_d(t) > 0$.

Similar arguments can be used to show that the other state variables have nonnegative solutions for all $t > 0$, hence completing the proof. □

Theorem 1 indicates that the model is epidemiologically meaningful and mathematically well posed.

3.2. Boundedness of Solution of the Model

Theorem 2 (Boundedness of the Model). *All solutions of system (1) starting in $\mathbb{R}^6_+$ are bounded.*

Proof. Consider $\quad W_d(t) = S_d(t) + E_d(t) + I_d(t) \quad$ and $W_h(t) = S_h(t) + E_h(t) + I_h(t)$.

Then,

$$\frac{dW_d}{dt} = P_d - (\mu_d + \gamma_1(S_h + E_h))S_d$$

$$- (\mu_d + \gamma_1(S_h + E_h) + C_d)E_d - (\mu_d + \gamma_1(S_h + E_h) + \sigma_d)I_d$$

$$= P_d - (\mu_d + \gamma_1(S_h + E_h))(S_d + E_d + I_d) - C_d E_d - \sigma_d I_d,$$

$$\frac{dW_h}{dt} = P_h + \gamma(S_h + E_h)(S_d + E_d + I_d) - \mu_h S_h - \mu_h E_h - I_h(\mu_h + \sigma_h)$$

$$= P_h + \gamma(S_h + E_h)(S_d + E_d + I_d) - (S_h + E_h + I_h)\mu_h - I_h\sigma_h.$$
(3)

From the above equations, we have

$$\frac{dW_d}{dt} \leq P_d - \gamma_1(S_h + E_h)(S_d + E_d + I_d) - \mu_d W_d,$$

$$\frac{dW_h}{dt} \leq P_h + \gamma(S_h + E_h)(S_d + E_d + I_d) - \mu_h W_h.$$
(4)

If we set $W = W_h + W_d$ and $P = P_d + P_h$, then,

$$\frac{dW}{dt} \leq P - \min\{\mu_d, \mu_h\}W,$$
(5)

so that

$$W(t) \leq W(0)e^{-Pt} + \frac{P}{\min\{\mu_d, \mu_h\}}\left(1 - e^{-Pt}\right).$$
(6)

The feasible region for the model is thus given by

$$\Omega = \left\{(S_d, E_d, I_d, S_h, E_h, I_h) \in \mathbb{R}^6 : 0 \leq W \leq \frac{P}{\min\{\mu_d, \mu_h\}}\right\}.$$
(7)

System (1) can therefore be conveniently studied in Ω. □

3.3. Equilibrium Points of the Model.
The model exhibits a biologically reasonable disease-free equilibrium $\varepsilon_0 = (S_{d0}, 0, 0, S_{h0}, 0, 0)$, where $S_{d0} = (P_d/(\mu_d + \gamma_1 S_{h0}))$ and S_{h0} satisfy the following quadratic equation:

$$\gamma_1\mu_h S_{h0}^2 - (P_d\gamma + P_h\gamma_1 - \mu_h\mu_d)S_{h0} - \mu_d P_h = 0.$$
(8)

Hence,

$$S_{h0} = \frac{(P_d\gamma + P_h\gamma_1 - \mu_h\mu_d) + \sqrt{(P_d\gamma + P_h\gamma_1 - \mu_h\mu_d)^2 + 4\gamma_1\mu_h\mu_d P_h}}{2\gamma_1\mu_h},$$

$$S_{d0} = \frac{2\mu_h P_d}{P_d\gamma + P_h\gamma_1 + \mu_h\mu_d + \sqrt{(P_d\gamma + P_h\gamma_1 - \mu_h\mu_d)^2 + 4\gamma_1\mu_h\mu_d P_h}}.$$
(9)

Remark 1. Model (1) has a unique realistic DFE.

Proof. Since the coefficient of S_{h0}^2 and the constant term in (8) have different signs, Descartes' rule of sign shows that equation (8) has only one positive solution. This concludes the proof of the remark.

The basic reproduction ratio, $\mathcal{R}_0$, is the average number of secondary infections that are caused by one infectious individual, which is introduced into an initially disease-free population, during its infectious period. Using the next-generation method of Diekmann et al. [22], we compute $\mathcal{R}_0$ as

$$\mathcal{R}_0 = \frac{S_{d0}\psi_1(\gamma_1 S_{h0} + \beta_{DD} + \psi_4)}{(\gamma_1 S_{h0} + \psi_4)(\gamma_1 S_{h0} + \psi_3)}.$$
(10)

In the presence of Infectives, model (1) is said to be exhibiting an endemic equilibrium point $\varepsilon_* = (S_d^*, E_d^*, I_d^*, S_h^*, E_h^*, I_h^*)$, where S_d^*, E_d^*, I_d^*, and I_h^* are given by

$$S_d^* = \frac{(\psi_3 + \gamma_1(S_h^* + E_h^*))(\psi_4 + \gamma_1(S_h^* + E_h^*))}{\psi_1[\psi_4 + \gamma_1(S_h^* + E_h^*) + \beta_{DD}]},$$

$$E_d^* = \frac{\psi_5 E_h^*(\psi_4 + \gamma_1(S_h^* + E_h^*))}{\psi_2 S_h^*[\psi_4 + \gamma_1(S_h^* + E_h^*) + \beta_{DD}]},$$

$$I_d^* = \frac{\beta_{DD}\psi_5 E_h^*}{\psi_2 S_h^*[\psi_4 + \gamma_1(S_h^* + E_h^*) + \beta_{DD}]}$$

$$I_h^* = \frac{\beta_H E_h^*}{\psi_6},$$

$$\tag{11}$$

and S_h^* and E_h^* satisfy the following set of equations (see Appendix for derivation):

$$\mathbb{S}_h \mathcal{G} \mathbb{E}_h^T + \mathbf{b}_1^T \mathbb{E}_h^T = 0,$$
$$\mathbb{S}_h \mathcal{H} \mathbb{E}_h^T + \mathbf{b}_2^T \mathbb{E}_h^T = 0.$$
$$\tag{12}$$

Here,

$$\mathbb{S}_h = \left[(S_h^*)^4, (S_h^*)^3, (S_h^*)^2, S_h^* \right],$$

$$\mathbb{E}_h = \left[(E_h^*)^3, (E_h^*)^2, E_h^*, 1 \right],$$

$$\mathcal{G} = \begin{bmatrix} \mathcal{G}_{11} & 0 & 0 & 0 \\ G_{21} & 3\mathcal{G}_{11} & 0 & 0 \\ \mathcal{G}_{31} & 2\mathcal{G}_{21} + \gamma_1^2\psi_5\psi_1 & 3\mathcal{G}_{11} & 0 \\ \mathcal{G}_{41} & \mathcal{G}_{31} + \gamma_1(\psi_3 + \psi_4)\psi_1\psi_5 & \mathcal{G}_{21} + 2\gamma_1^2\psi_5\psi_1 & G_{11} \end{bmatrix},$$

$$\mathbf{b}_1 = \begin{bmatrix} 0 \\ \psi_5\psi_1\psi_3\psi_4 \\ \gamma_1\psi_1\psi_5(\psi_3 + \psi_4) \\ \gamma_1^2\psi_5\psi_1 \end{bmatrix},$$

$$\mathcal{H} = \begin{bmatrix} \mathcal{H}_{11} & 0 & 0 & 0 \\ \mathcal{H}_{21} & 3\mathcal{H}_{11} & 0 & 0 \\ \mathcal{H}_{31} & \mathcal{H}_{21} + \varepsilon_1 & 3\mathcal{H}_{11} & 0 \\ \mathcal{H}_{41} & \mathcal{H}_{31} + \varepsilon_3 & \mathcal{H}_{21} + 2\varepsilon_1 + \varepsilon_2 & \mathcal{H}_{11} \end{bmatrix},$$

$$\mathbf{b}_2 = \begin{bmatrix} 0 \\ 0 \\ \gamma\psi_6\psi_5\psi_1(\psi_4 + \beta_{DD}) \\ \gamma\psi_6\psi_5\psi_1\gamma_1 \end{bmatrix},$$

$$\tag{13}$$

where

$$\mathcal{G}_{11} = \psi_2\gamma_1^3,$$

$$\mathcal{G}_{31} = \psi_2\gamma_1[(\psi_3 + \psi_4)\mu_d - P_d\psi_1 + \psi_3\psi_4],$$

$$\mathcal{H}_{11} = \gamma\psi_6\psi_2\gamma_1^2,$$

$$\mathcal{H}_{41} = P_h\psi_1(\psi_4 + \beta_{DD})\psi_2\psi_6,$$

$$\mathcal{H}_{31} = \psi_2\psi_6(P_h\psi_1\gamma_1 + \gamma\psi_3\psi_4) - \psi_1\psi_2\psi_6\mu_h(\psi_4 + \beta_{DD}),$$

$$\varepsilon_1 = (\gamma\psi_6(\psi_2\psi_3 + \psi_2\psi_4 + \psi_1\psi_5) + \theta\psi_1\psi_2(\psi_6 + \beta_H)$$
$$- \psi_1\psi_2\psi_6\psi_5)\gamma_1,$$

$$\varepsilon_3 = \psi_1(\psi_4 + \beta_{DD})(\theta\psi_2(\psi_6 + \beta_H) + \psi_6\psi_5(\gamma - \psi_2)$$
$$+ \psi_2\psi_6\mu_h),$$

$$\mathcal{G}_{21} = \gamma_1^2\psi_2(\psi_3 + \psi_4 + \mu_d),$$

$$\mathcal{G}_{41} = \psi_2[\psi_3\psi_4\mu_d - P_d\psi_1(\psi_4 + \beta_{DD})],$$

$$\mathcal{H}_{21} = \psi_2\psi_6\gamma_1(\gamma(\psi_3 + \psi_4) - \mu_h\psi_1),$$

$$\tag{14}$$

where S_h^* and E_h^* can be found by solving equation (12). Even though the solution can be done numerically, the nature of the problem suggests that an algebraic solution may be obtainable. The problem falls in the category of Open Problem 1. □

Open Problem 1

Let $\mathbf{b} = [b_1, b_2, \ldots, b_n], \mathbf{c} = [c_1, c_2, \ldots, c_n],$ $\mathbf{x} = [x^n, x^{n-1}, \ldots x], \mathbf{y} = [y^{n-1}, x^{n-2}, \ldots y, 1],$ and $\mathbb{A}$ and $\mathbb{B}$ be n–dimensional lower triangular square matrices. Is there an algebraic technique that be used to determine $\mathbf{x}$ and $\mathbf{y}$ satisfying the following equations?

$$\mathbf{x}\mathbb{A}\mathbf{y}^T + \mathbf{b}\mathbf{y}^T = 0,$$
$$\mathbf{x}\mathbb{B}\mathbf{y}^T + \mathbf{c}\mathbf{y}^T = 0.$$
$$\tag{15}$$

In the next section, the local stability of the disease-free equilibrium is discussed.

3.4. Local Stability of the Disease-Free Equilibrium Point. We use the Lyapunov indirect method to study the local stability of the disease-free equilibrium point of the model. An equilibrium point is said to be locally asymptotically stable if all eigenvalues of the Jacobian model evaluated at the given equilibrium point have negative real parts.

The Jacobian matrix of the model evaluated at ε_0 is given by

$$J(\varepsilon_0) = \begin{bmatrix} -\mu_d - \gamma_1 S_{h0} & -\psi_1 S_{d0} & -\psi_1 S_{d0} & -\gamma_1 S_{d0} & -\gamma_1 S_{d0} & 0 \\ 0 & \psi_1 S_{d0} - \psi_3 - \gamma_1 S_{h0} & \psi_1 S_{d0} & 0 & 0 & 0 \\ 0 & \beta_{DD} & -\psi_4 - \gamma_1 S_{h0} & 0 & 0 & 0 \\ \gamma S_{h0} & (\gamma - \psi_2) S_{h0} & (\gamma - \psi_2) S_{h0} & \gamma S_{d0} - \mu_h & \gamma S_{d0} + \theta & \theta \\ 0 & \psi_2 S_{h0} & \psi_2 S_{h0} & 0 & -\psi_5 & 0 \\ 0 & 0 & 0 & 0 & \beta_H & -\psi_6 \end{bmatrix}. \tag{16}$$

Two of the eigenvalues ($-\psi_5$ and $-\psi_6$) of $J(\varepsilon_0)$ are clearly negative and the remaining eigenvalues are the zeros of $\mathscr{P}_1(\lambda)\mathscr{P}_2(\lambda) = 0$, where

$$\left.\begin{aligned} \mathscr{P}_1(\lambda) &= \lambda^2 + (2\gamma_1 S_{h0} + \psi_3 + \psi_4 - \psi_1 S_{d0})\lambda \\ &\quad + (\psi_4 + \gamma_1 S_{h0})(\psi_3 + \gamma_1 S_{h0})(1 - \mathscr{R}_0) = 0, \\ \mathscr{P}_2(\lambda) &= \lambda^2 + (\mu_h - \gamma S_{d0} + \gamma_1 S_{h0} + \mu_d)\lambda \\ &\quad + \mu_h \gamma_1 S_{h0} + \mu_h \mu_d - \gamma S_{d0} \mu_d = 0. \end{aligned}\right\} \tag{17}$$

The second equation of (8) has both zeros being negative if (remembering that $S_{d0} = (P_d/(\mu_d + \gamma_1 S_{h0}))$)

$$\begin{aligned} \mu_h(\gamma_1 S_{h0} + \mu_d) - \frac{\gamma P_d \mu_d}{\mu_d + \gamma_1 S_{h0}} &> 0, \\ \gamma_1 S_{h0} + \mu_d + \mu_h - \frac{P_d \gamma}{\mu_d + \gamma_1 S_{h0}} &> 0. \end{aligned} \tag{18}$$

These conditions are easy to establish using (8).

Also, the first equation in (17) has zeros with negative real parts if

$$\begin{aligned} (\psi_4 + \gamma_1 S_{h0})(\psi_3 + \gamma_1 S_{h0})(1 - \mathscr{R}_0) &> 0, \\ 2\gamma_1 S_{h0} + \psi_3 + \psi_4 - \psi_1 S_{d0} &> 0. \end{aligned} \tag{19}$$

The first condition is true whenever $\mathscr{R}_0 < 1$ whilst the second condition can be written as $((\psi_4 + \psi_3 + 2\gamma_1 S_{h0})(\psi_4 + \gamma_1 S_{h0} + \beta_{DD}))/((\psi_4 + \gamma_1 S_{h0})(\psi_3 + \gamma_1 S_{h0})) - \mathscr{R}_0 > 0$, which holds if $\mathscr{R}_0 < 1$. The following result is therefore established.

Theorem 3. *The disease-free equilibrium state, ε_0, of model (1) is locally asymptotically stable whenever $\mathscr{R}_0 < 1$ and unstable otherwise.*

3.5. Local Stability Analysis of Endemic Equilibrium. The stability of the endemic equilibriums of the model is similarly established by finding the eigenvalues of the Jacobian matrix of the model evaluated at the positive solutions of (12). The Jacobian model evaluated at ε^* if it exists is given by

$$\mathscr{J}(\varepsilon^*) = \begin{bmatrix} \mathscr{J}_{11} & -S_d^* \psi_1 & -S_d^* \psi_1 & -\gamma_1 S_d^* & -\gamma_1 S_d^* & 0 \\ \psi_1 EI_d & \mathscr{J}_{22} & S_d^* \psi_1 & -\gamma_1 E_d^* & -\gamma_1 E_d^* & 0 \\ 0 & \beta_{DD} & \mathscr{J}_{33} & -\gamma_1 I_d^* & -\gamma_1 I_d^* & 0 \\ \gamma SE_h & \gamma SE_h - \psi_2 S_h^* & \gamma SE_h - \psi_2 S_h^* & \mathscr{J}_{44} & \gamma SEI_d + \theta & \theta \\ 0 & \psi_2 S_h^* & \psi_2 S_h^* & \psi_2 EI_d & -\psi_5 & 0 \\ 0 & 0 & 0 & 0 & \beta_H & -\psi_6 \end{bmatrix}, \tag{20}$$

where

$$\begin{aligned} \mathscr{J}_{11} &= -(\psi_1 EI_d + SE_h \gamma_1 + \mu_d), \\ \mathscr{J}_{33} &= -(SE_h \gamma_1 + \psi_4), \\ EI_d &= E_d^* + I_d^*, \\ SEI_d &= S_d^* + E_d^* + I_d^*, \\ \mathscr{J}_{22} &= S_d \psi_1 - SE_h \gamma_1 - \psi_3, \\ \mathscr{J}_{44} &= \gamma SEI_d - \psi_2 EI_d - \mu_h, \\ SE_h &= S_h^* + E_h^*. \end{aligned} \tag{21}$$

The Jacobian matrix $\mathscr{J}(\varepsilon^*)$ has a characteristic polynomial given by

$$\sum_{n=0}^{6} \Lambda_n \lambda^n = 0, \tag{22}$$

where

$$\Lambda_6 = 1,$$

$$\Lambda_5 = \psi_6 + \psi_5 + \frac{\gamma E_h S E I_d}{S_h} + \frac{P_d}{S_d} + \frac{\theta(E_h + I_h) + P_h}{S_h} + \frac{S_d \psi_1 I_d}{E_d} + \frac{\beta_{DD} E_d}{I_d},$$

$$\Lambda_4 = \left(\frac{\psi_6}{S_d} + \frac{P_h}{S_h S_d}\right) P_d + \left(\frac{\psi_6}{S_h} + \frac{\psi_5}{S_h}\right) P_h + \left(\psi_5 + \frac{\gamma S E I_d E_h}{S_h} + \frac{\theta(E_h + I_h)}{S_h} + \frac{\beta_{DD} E_d}{I_d} + \frac{S_d \psi_1 I_d}{E_d}\right)\left(\frac{P_d}{S_d} + \psi_6\right)$$

$$+ \left(\frac{\theta I_h}{S_h} + \frac{\beta_{DD} E_d}{I_d} + \frac{S_d \psi_1 I_d}{E_d}\right)\psi_5 + \gamma \gamma_1 S E I_d S E_h + E I_d \psi_1^2 S_d +$$

$$\left(\frac{\gamma S E I_d E_h}{S_h} + \frac{P_h}{S_h} + \frac{\theta(E_h + I_h)}{S_h}\right)\left(\frac{S_d \psi_1 I_d}{E_d} + \frac{\beta_{DD} E_d}{I_d}\right),$$

$$\Lambda_3 = \left(\gamma S E_h \gamma_1 + \frac{E I_d \psi_1^2 S_d}{E_d}\right)\beta_{DD} E_d + \left[\psi_5\left(\frac{P_h}{S_h} + \frac{\beta_{DD} E_d}{I_d} + \frac{S_d \psi_1 I_d}{E_d} + \frac{P_d}{S_d}\right) + \left(\frac{\gamma S E I_d E_h}{S_h} + \frac{\theta E_h}{S_h}\right)\left(\frac{\beta_{DD} E_d}{I_d} + \frac{S_d \psi_1 I_d}{E_d} + \frac{P_d}{S_d}\right)\right]$$

$$\psi_6 \quad + \left[\gamma \gamma_1 S E I_d S E_h + E I_d \psi_1^2 S_d + \left(\frac{P_h}{S_h} + \frac{\theta I_h}{S_h}\right)\left(\frac{\beta_{DD} E_d}{I_d} + \frac{S_d \psi_1 I_d}{E_d} + \frac{P_d}{S_d}\right) + \frac{\beta_{DD} E_d P_d}{S_d I_d} + \frac{\psi_1 I_d P_d}{E_d}\right](\psi_6 + \psi_5)$$

$$+ \left[S_d \psi_1 S E_h I_d + \frac{S E_h E I_d P_d}{S_d} + \left(\frac{(E_d + S_d)\beta_{DD} E_d}{I_d} + \frac{(I_d + S_d)S_d \psi_1 I_d}{E_d}\right) S E_h\right]\gamma \gamma_1$$

$$+ \left(S_d \psi_1^2 \left(\frac{\gamma S E I_d E_h}{S_h} + \frac{P_h}{S_h} + \frac{\theta(E_h + I_h)}{S_h}\right) + \frac{\psi_1^2 S_d \beta_{DD} E_d}{I_d}\right) E I_d$$

$$+ \left(\frac{\beta_{DD} E_d P_d}{S_d I_d} + \frac{\psi_1 I_d P_d}{E_d}\right)\left(\frac{\gamma S E I_d E_h}{S_h} + \frac{P_h}{S_h} + \frac{\theta(E_h + I_h)}{S_h}\right) + \gamma E I_d S E I_d S E_h \gamma_1 \psi_2.$$

$$(23)$$

Clearly, $\Lambda_n > 0, \forall n = 3, 4, 5, 6$, and Λ_0, Λ_1, and Λ_2 can similarly be shown to be positive. By Descartes' rule of signs, the following result is thus established.

Theorem 4. *The endemic equilibrium ε^* (whenever it exists) is locally asymptotically stable.*

3.6. Global Stability of Disease-Free Equilibrium Point. To study the global stability of $\mathscr{E}_0$, we employ the technique of Castillo-Chavez et al. [23] as follows:

If $X_G = (S_d, S_h)$ and $Z_G = (E_d, I_d, E_h, I_h)$, then model (1) can be written as

$$\left.\begin{array}{l} \dfrac{dX_G}{dt} = F(X_G, Z_G), \\[2mm] \dfrac{dZ_G}{dt} = G(X_G, Z_G), G(X_G, 0) = 0. \end{array}\right\} \quad (24)$$

By the theorem of Castillo-Chavez et al. [23], ε_0 is globally asymptotically stable if the following conditions are satisfied:

GS1: ε_0 is locally asymptotically stable

GS2: X_G^* is globally stable for $(dX_G/dt) = G(X, 0)$

GS3: $G(X_G, Z_G) = A_G Z_G - \widehat{G}(X_G, Z_G)$, $\widehat{G}(X_G, Z_G) \geq 0$, where $A_G = D_{Z_G}(X_G^*, 0)$ is an M-Matrix

The first condition GS1 has been established for $\mathscr{R}_0 < 1$ in Theorem 3.

For the second condition, we note that $F(X_G, 0) = \begin{pmatrix} P_d - \mu_d S_d - \gamma_1 S_h S_d \\ P_h + \gamma S_h S_d - \mu_h S_h \end{pmatrix}$ is a limiting function of $(dX_G/dt) = F(X_G, Z_G)$. Therefore, the second condition is satisfied.

As for the third condition, we observe that A_G is given by

$$A_G = \begin{bmatrix} S_{d0}\psi_1 - \psi_3 - \gamma_1 S_{h0} & S_{d0}\psi_1 & 0 & 0 \\ \beta_{DD} & -(\psi_4 + \gamma_1 S_{h0}) & 0 & 0 \\ S_{h0}\psi_2 & S_{h0}\psi_2 & -\psi_5 & 0 \\ 0 & 0 & \beta_H & -\psi_6 \end{bmatrix},$$

$$(25)$$

which is an M−matrix (i.e., all nondiagonal entries are nonnegative).

Furthermore, the matrix $\widehat{G}(X_G, Z_G)$ is given by

$$\widehat{G}(X_G, Z_G) = \begin{pmatrix} \psi_1 (S_{d0} - S_d)(E_d + I_d) + E_d \gamma_1 (S_h + E_h - S_{h0}) \\ I_d \gamma_1 (S_h + E_h - S_{h0}) \\ \psi_2 (S_{h0} - S_h)(E_d + I_d) \\ 0 \end{pmatrix}. \tag{26}$$

From $(\mathrm{d}S_d/\mathrm{d}t)$, we have

$$S_d(t) = \frac{\vartheta(t_0) S_d(t_0) + P_d t}{\vartheta(t)}, \tag{27}$$

where

$$\vartheta(t) = \exp\left\{\int (\mu_d + \gamma_1 (S_h + E_h) + (E_d + I_d)\psi_1) \mathrm{d}t\right\}. \tag{28}$$

Hence, $S_{d0} \geq S_d(t)$. Similarly, $S_h(t) \geq S_{h0}$ and hence, $\widehat{G} \geq 0$, satisfies condition GS3. Now, since all three conditions are satisfied, the following result is established.

Theorem 5. *The disease-free equilibrium point, ε_0, is globally asymptotically stable whenever $\mathscr{R}_0 \leq 1$.*

For simulation purposes, we use the following parameter values:

$$\left.\begin{array}{l} P_d = 0.45, \\ P_h = 0.1, \\ \beta_d = 0.062, \\ \delta_d = 0.5 \\ \beta_{DD} = 0.6, \\ \sigma_d = 0.8, \\ \beta_h = 0.0229, \\ \delta_h = 0.005, \\ \beta_H = 0.0001, \\ \sigma_h = 0.0001, \\ \gamma_1 = 0.01, \\ \gamma = 0.0001, \\ \mu_d = 0.06, \\ C_d = 0.05, \\ \theta = 0.9, \\ \mu_h = 0.04. \end{array}\right\} \tag{29}$$

3.7. Sensitivity Analysis.

To determine the impact of model parameters on the spread of rabies, we employed global sensitivity analysis technique of Marino et al. [24] to calculate the partial rank correlation coefficients of the model parameters on which the basic reproduction number depends. The result of sensitivity analysis informs which model parameters are most important in determining the output variable (in this case, $\mathscr{R}_0$) and hence should be given much attention in terms of measurement for accuracy. The PRCC of results are shown in Figure 1.

Local sensitivity analysis discusses the impact of small changes in parameter values on the spread of the disease. To study the sensitivity of the disease spread to parameter changes, the basic reproduction number $\mathscr{R}_0$ is used, since it determines the persistence or possible eradication of the disease. The normalized forward sensitivity index is used to perform the analysis. It is defined as follows.

Let $\mathscr{R}_0$ be a differentiable function x_i. Then, the normalized forward sensitivity index of R_0 relative to x_i is given by

$$\eta_{\mathscr{R}_0}^{x_i} = \frac{\partial \mathscr{R}_0}{\partial x_i} \times \frac{x_i}{\mathscr{R}_0}. \tag{30}$$

Local sensitivity indexes are presented in Table 2. This index measures the relative percentage change in $\mathscr{R}_0$ due to a percentage change in x_i. The sensitivity indexes of the parameters determining $\mathscr{R}_0$ are calculated and presented in Table 2. A positive sensitivity index of a parameter implies that an increase (decrease) in the value of the parameter will result in an increase (decrease) in $\mathscr{R}_0$. On the other hand, a negative sensitivity index implies that an increase (decrease) in the value of the parameter will result in a decrease (an increase) in $\mathscr{R}_0$.

3.8. Bifurcation Analysis.

To study the stability of endemic equilibrium points, the center manifold theory as described in Theorem 4.1 of [25] is used as an alternative to the Lyapunov indirect method.

Choosing $\beta_d = \beta_d^* = ((\psi_3 + \gamma_1 S_{h0})(\psi_4 + \gamma_1 S_{h0}))/((1 - \delta_d) S_{d0}(\gamma_1 S_{h0} + \sigma_d + \psi_3))$ as a bifurcation parameter leads the Jacobian model to have a zero simple eigenvalue. The right and left eigenvectors associated with this zero eigenvalue are given by $\mathbf{w} = w_2 [\eta_1, 1, \eta_3, \eta_4, \eta_5, \eta_6]^T$ and $\mathbf{v} = [0, v_2, \zeta_3 v_2, 0, 0, 0]$ with $(\eta_3 \zeta_3 + 1) w_2 v_2 = 1$, where

$$\eta_1 = \frac{S_{d0}(\varphi_1 \gamma_1 - \varphi_2 \gamma) + \mu_h \varphi_2}{S_{d0} \mu_d - \mu_h (\mu_d + \gamma_1 S_{h0})},$$

$$\eta_3 = \frac{\beta_{DD}}{\psi_4 + \gamma_1 S_{h0}},$$

$$\eta_4 = \frac{\varphi_2 \gamma S_{h0} - \varphi_1 (\mu_d + \gamma_1 S_{h0})}{S_{d0} \mu_d - \mu_h (\mu_d + \gamma_1 S_{h0})},$$

$$\eta_5 = \frac{\psi_2 S_{h0} (\gamma_1 S_{h0} + \psi_4 + \beta_{DD})}{(\psi_4 + \gamma_1 S_{h0})\psi_5},$$

$$\eta_6 = \frac{\beta_H \psi_2 S_{h0} (\gamma_1 S_{h0} + \psi_4 + \beta_{DD})}{(\psi_4 + \gamma_1 S_{h0})\psi_5 \psi_6}, \tag{31}$$

$$\zeta_3 = \frac{(\gamma_1 S_{h0} + \psi_3)}{\gamma_1 S_{h0} + \psi_4 + \beta_{DD}},$$

$$\varphi_1 = S_{d0}(\psi_1 (1 + \eta_3) + \gamma_1 \eta_5),$$

$$\varphi_2 = S_{h0}(1 + \eta_3)(\gamma - \psi_2) + (\gamma S_{d0} + \theta)\eta_5 + \theta \eta_6.$$
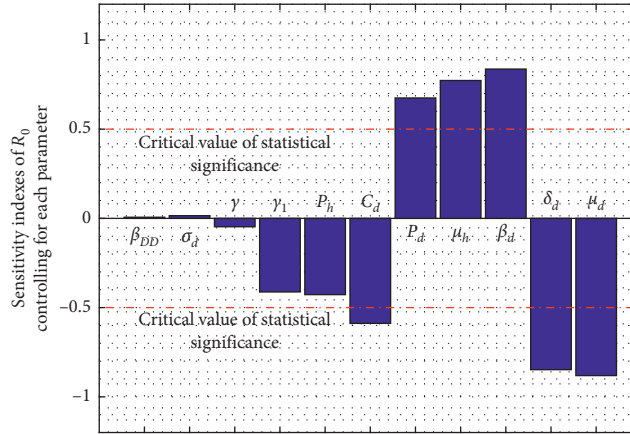
The bifurcation coefficients are thus given by

FIGURE 1: Plot of PRCC of $\mathscr{R}_0$ with respect to model parameters.

TABLE 2: Sensitivity indexes of $\mathscr{R}_0$ with respect to model parameters.

C_d	P_d	P_h	β_{DD}	β_d	δ_d	μ_d	μ_h	σ_d	γ	γ_1
−0.009	−2.010	−0.0143	−0.012	1.000	−1.000	1.056	3.024	−0.011	−3.967	0.943

$$\mathbf{a} = 2w_2 \left(p^* - 1 \right),$$

$$\mathbf{b} = \frac{S_{d0} \left(1 - \delta_d \right) \left(1 + \eta_3 \right)}{1 + \eta_3 \zeta_3}, \tag{32}$$

where $p^* = (\beta_d^* \eta_1 (1 - \delta_d)(1 + \eta_3))/(\gamma_1 (\eta_4 + \eta_5) (1 + \eta_3 \zeta_3))$.

Clearly, $\mathbf{b}$ is always positive and the sign of $\mathbf{a}$ is dependent on p^*. The following result therefore easily follows [25].

Theorem 6. *Model (1) undergoes a backward bifurcation at $\mathscr{R}_0 = 1$ when $p^* > 1$ and a forward bifurcation when $p^* < 1$ in which case the endemic equilibrium will be locally asymptotically stable.*

We note that since $\mathscr{E}_0$ is GAS whenever $\mathscr{R}_0 \leq 1$, then backward bifurcation may only occur whenever $\mathscr{R}$ approaches unity from above.

4. Numerical Simulation

We performed numerical simulation in this section to study the impact of various parameters on the spread of the rabies infection. To perform these experiments, the following initial values are used: $S_d(0) = 2.0$, $E_d(0) = 1.5$, $I_d(0) = 1.0$, $S_h(0) = 2.5$, $E_h(0) = 0.5$, and $I_h(0) = 0.5$ and the baseline parameter values are taken from equation (29).

To illustrate the local stability of the disease-free equilibrium, model (1) is solved for various initial values of the state variables and the results are plotted on the same graph. This procedure is done for the case when $\mathscr{R}_0 < 1$ and when $\mathscr{R}_0 > 1$. The fact that the DFE is LAS whenever $\mathscr{R}_0 < 1$ as in Theorem 3 is demonstrated in Figure 2.

To illustrate the result in Theorem 1, we solved model (1) for two different scenarios: (a) when initial conditions are less than $(P_h + P_d)/\min\{\mu_h, \mu_d\}$ and (b) when initial

conditions are greater than $(P_h + P_d)/\min\{\mu_h, \mu_d\}$. Figure 3 shows that the feasible region Ω is an invariant attractor of system (1).

We also solved the model for different values of the most influential model parameters in order to simulate the effect of those variables. The results are shown in Figures 4–8.

From Figure 4, it is observed that increasing the dog-dog rabies transmission rate, β_d leads to a decline (an increase) in the Susceptible dog population (Exposed and Infected dog populations) and a similar (but slight) effect is observed for the Susceptible human population (Exposed and Infected human populations). The smaller impact on human population could be attributable to the fact that infected dogs will often be culled, leading to reduced infection among humans.

From Figure 5, it is observed that increasing the rate of immunization of dogs, δ_d, leads to an increase (a decrease) in the Susceptible dog population (Exposed and Infected dog populations) and a similar effect is observed for the Susceptible human population (Exposed and Infected human populations). It is however observed that for higher rates of immunization, the Susceptible dog populations first peak before declining. This could be attributable to the fact that immunization may not confer permanent reduction in transmissibility of rabies from dogs. We also observe that irrespective of the rates of immunization, a steady state is reached, confirming the global stability of the disease-free equilibrium.

5. Findings

The following are the findings of the study:

  (i) The rabies-free equilibrium point is globally asymptotically stable whenever the basic reproduction number $\mathscr{R}_0$ is less than unity.
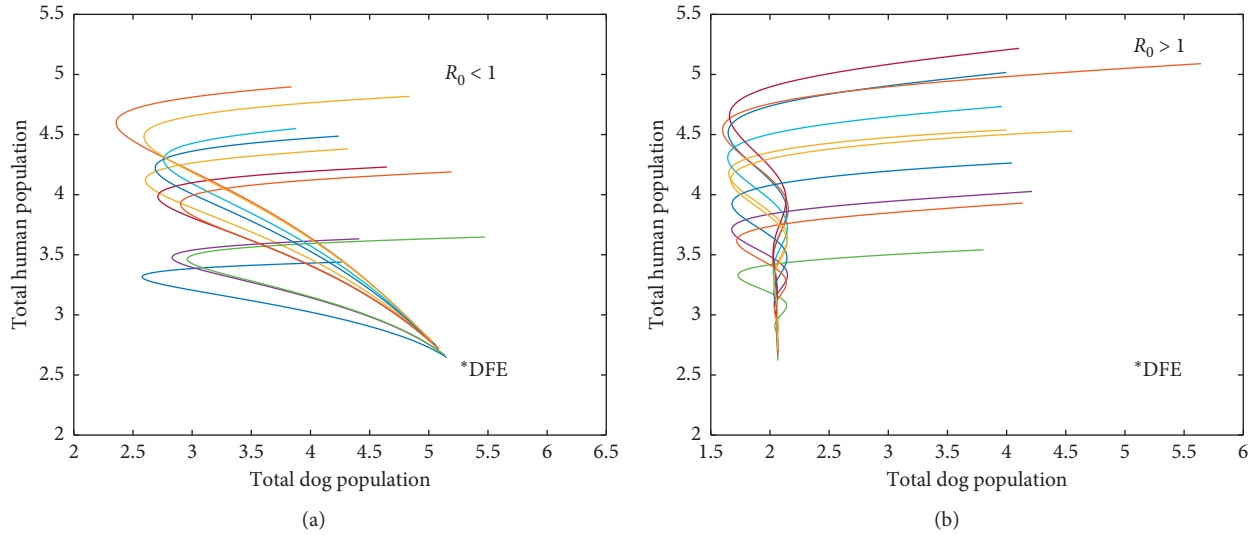
(a)

(b)

FIGURE 2: Simulation of model (1) showing the local asymptotic stability of the $\mathscr{E}_0$ (a) for $\mathscr{R}_0 < 1$ and instability (b) for $\mathscr{R}_0 > 1$.
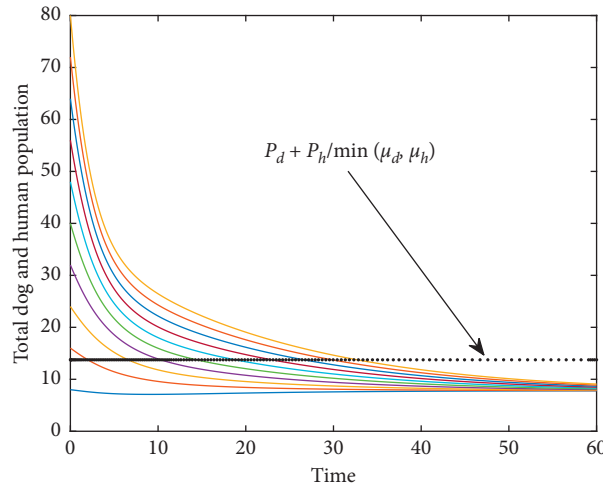

FIGURE 3: Simulation of the model illustrating the boundedness of solutions of model (1) (Theorem 1).

(ii) While the spread of disease is positively correlated with the dog-to-dog transmission rate β_d, recruitment rate of dogs P_d, rate at which exposed dogs develop clinical rabies β_{DD}, and disease-induced death rate among σ_d, and natural death rate of humans μ_h, it is negatively correlated with the natural death rate of dogs μ_d, immunization of dogs δ_d, culling rate C_d, rate of consumption of dogs γ_1, as indicated in the sensitivity indexes of $\mathscr{R}_0$.

(iii) Efforts at controlling the spread of rabies should focus more on dog population than on humans.

(iv) Under certain conditions, the model may exhibit either forward or backward bifurcation near $\mathscr{R}_0 = 1$. The possibility of forward bifurcation suggests the existence of locally asymptotically stable endemic equilibrium when $\mathscr{R}_0 > 1$.

6. Discussion and Conclusion

In this paper, a nonlinear mathematical model has been proposed to study the dynamics of rabies infection among dogs and in a human population that is exposed to dog bites. It is observed that the model has a unique disease-free equilibrium point which is locally asymptotically stable whenever the basic reproduction number is less than unity. The disease-free equilibrium point, $\mathscr{E}_0$, is also globally asymptotically stable whenever $\mathscr{R}_0 \leq 1$. It is also observed that the most influential factors on the spread of the disease are probability of infection upon contact among dogs β_d, rate of immunization of dogs δ_d, rate of recruitment of dogs P_d, rate at which exposed dogs develop clinical rabies β_{DD}, disease-induced death rate within the dog population σ_d, and natural mortality rate of dogs, μ_d. If the rate of immunization in the dog population is increased, it will have an increasing effect on the susceptible human population as shown in Figure 5. If
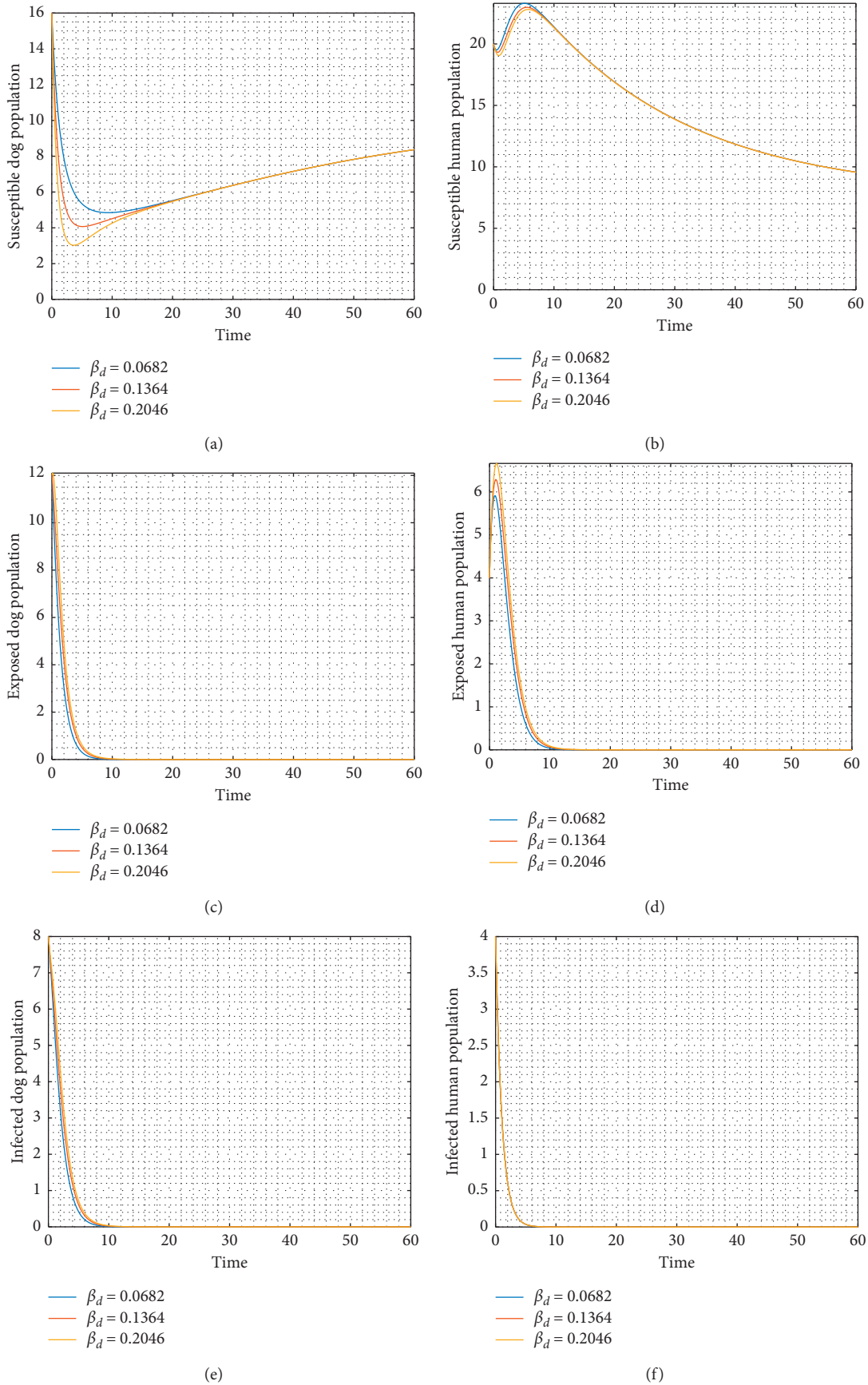
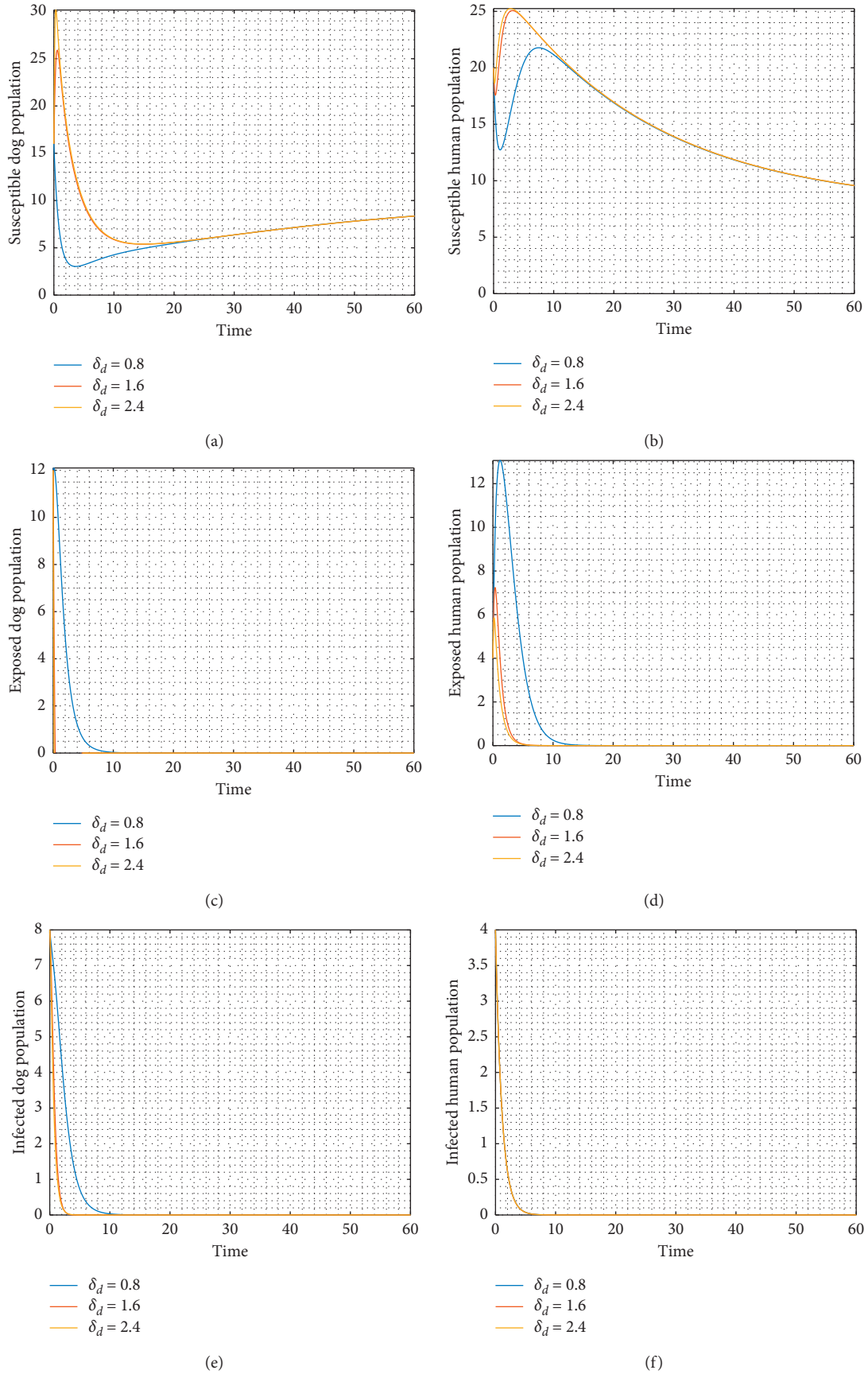FIGURE 4: Solution curves of model (1) showing the effect of β_d.

(a)

(b)

(c)

(d)

(e)

(f)

FIGURE 5: Solution curves of model (1) showing the effect of δ_d.

Figure 6: Solution curves of model (1) showing the effect of σ_d.

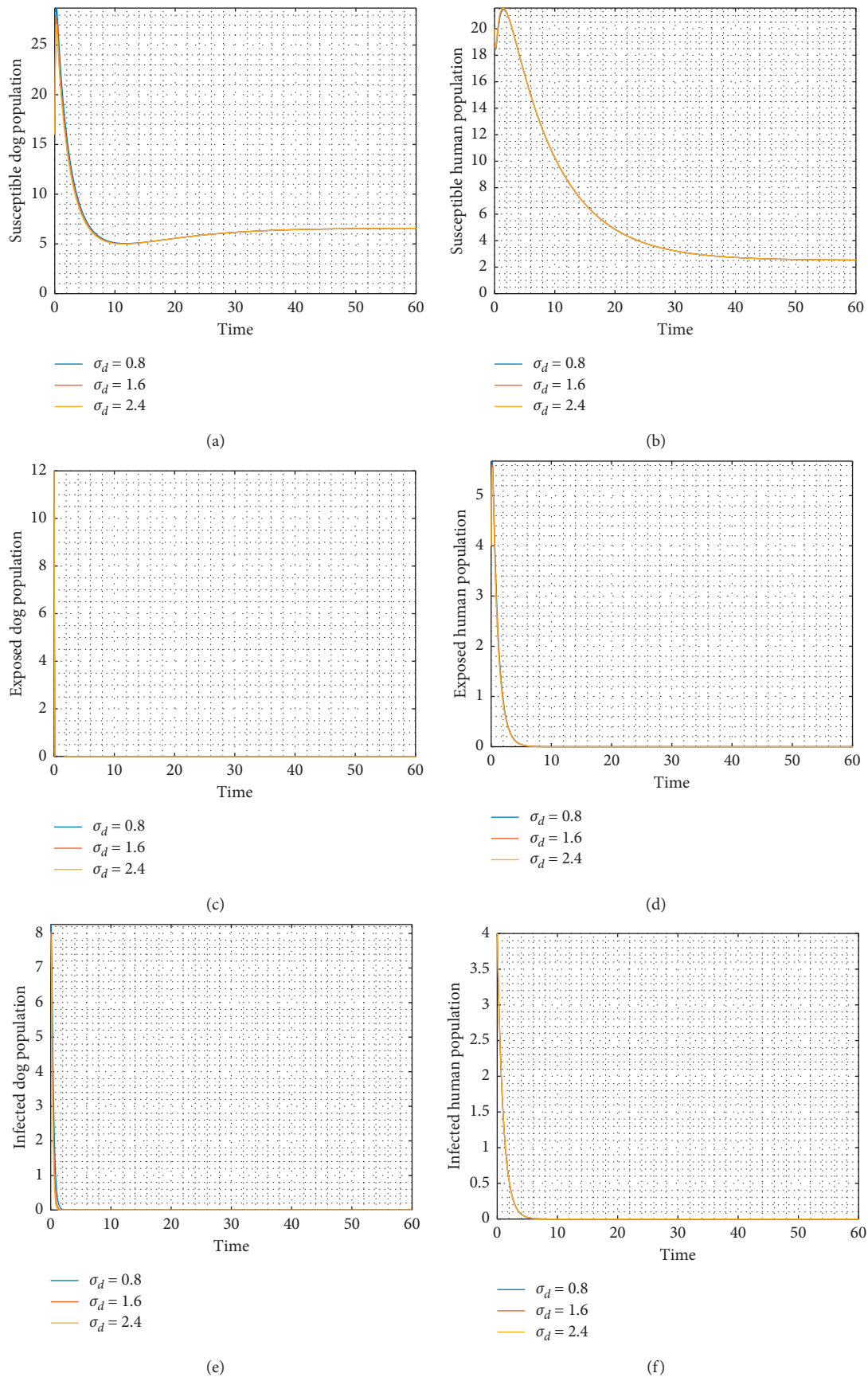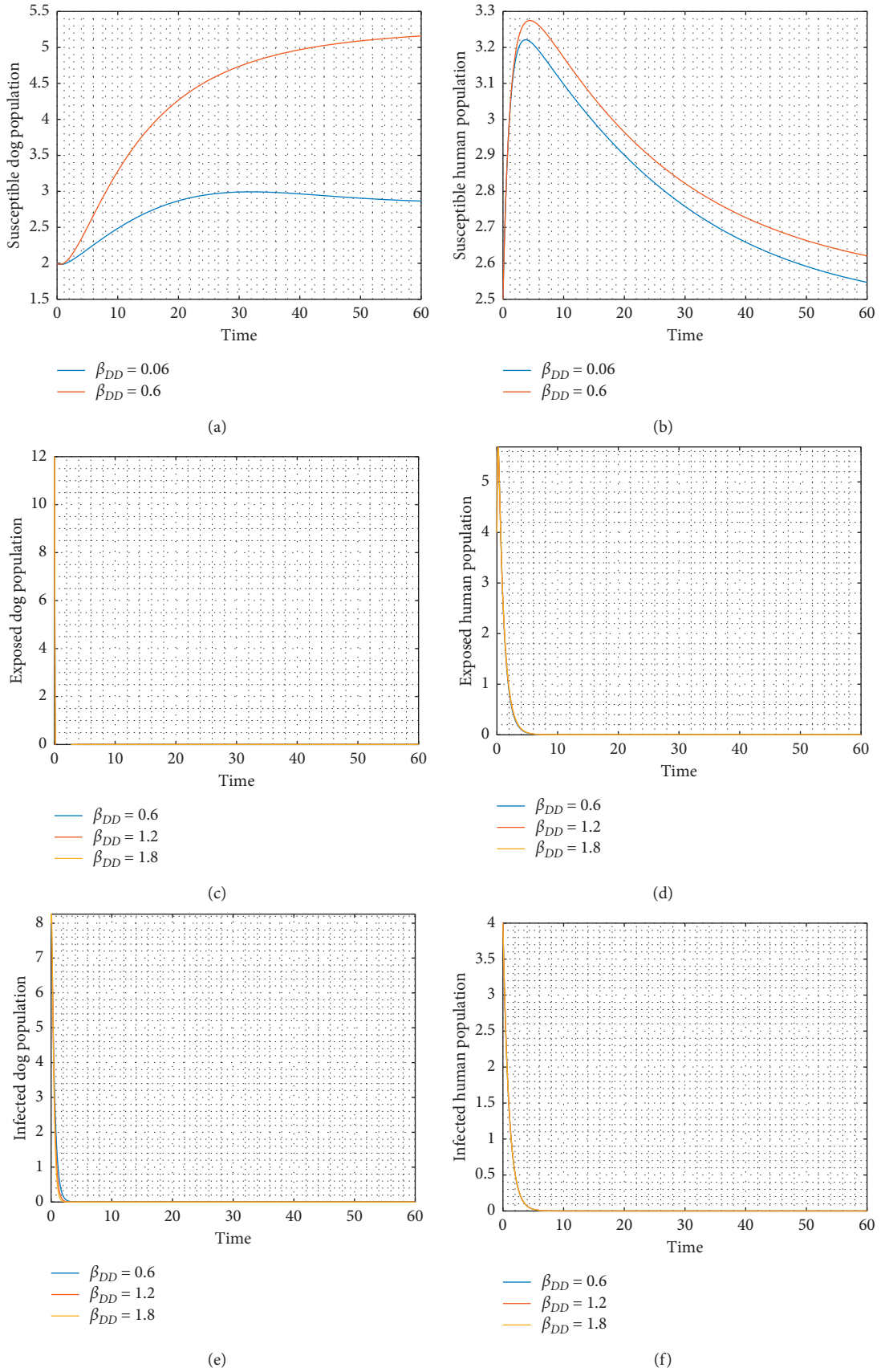(a)

(b)

(c)

(d)

(e)

(f)

FIGURE 7: Solution curves of model (1) showing the effect of β_{DD}.
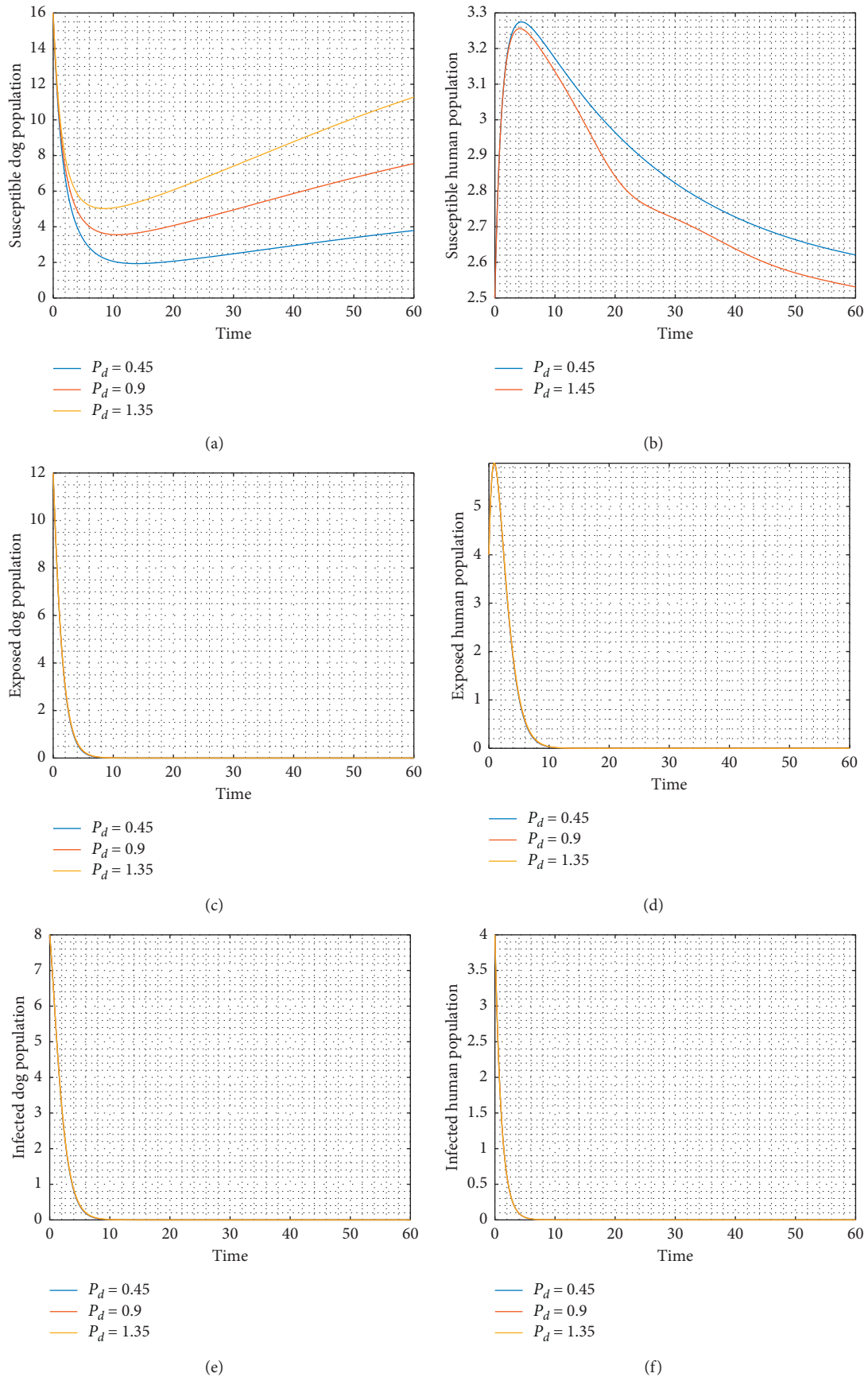
Figure 8: Solution curves of model (1) showing the effect of P_d.

the recruitment rate within the dog population, disease-induced death rate of dogs, rate of contact among dogs, and the rate at which exposed dogs develop clinical rabies each is reduced, with all other factors remaining constant, there will be a positive effect on the susceptible human population as shown in Figures 4 and 6–8. These actions will lead to a reduction in the reproduction number. However, even though it has been accepted that the eradication of infectious diseases in a community should target the reduction of $\mathscr{R}_0$ to less than unity, this may not be the only case for this system. It has also been observed that under certain conditions, backward bifurcation may occur around $\mathscr{R}_0 = 1$. From the analysis, we noted that since ε_0 is GAS whenever $\mathscr{R}_0 \leq 1$, then backward bifurcation may only occur whenever $\mathscr{R}_0$ approaches unity from above. This has a major implication for the spread of the disease once the basis of reducing $\mathscr{R}_0$ below the threshold point, $\mathscr{R}_0 = 1$, may not just be enough for effectively controlling the spread of the disease because whenever $\mathscr{R}_0$ approaches unity from above, there is the likelihood of coexistence of endemic equilibrium which may result in reinfection of the rabies virus. The conclusions, however, of Asamoah et al. [5]; Hou et al. [12]; and Wang and Lou [19] are that vaccination, which may lead to immunity, as a better control method of rabies infection in dogs will still be relevant in the presence of backward bifurcation.

Appendix

Derivation of Equation (12)

The equilibrium points of the model are found by equating the right-hand sides to zero to obtain

$$P_d - S_d (E_d + I_d)\psi_1 - (\mu_d + \gamma_1 (S_h + E_h))S_d = 0, \quad \text{(A.1)}$$

$$S_d (E_d + I_d)\psi_1 - (\psi_3 + \gamma_1 (S_h + E_h))E_d = 0, \quad \text{(A.2)}$$

$$\beta_{DD} E_d - (\psi_4 + \gamma_1 (S_h + E_h))I_d = 0, \quad \text{(A.3)}$$

$$P_h + \gamma (S_h + E_h)(N_d) + \theta(E_h + I_h) - ((E_d + I_d)\psi_2 + \mu_h)S_h = 0, \quad \text{(A.4)}$$

$$S_h (E_d + I_d)\psi_2 - \psi_5 E_h = 0, \quad \text{(A.5)}$$

$$\beta_H E_h - \psi_6 I_h = 0. \quad \text{(A.6)}$$

Let the endemic equilibrium be $\varepsilon_* = (S_d^*, E_d^*, I_d^*, S_h^*, E_h^*, I_h^*)$.

From equation (A.1), we have $I_h^* = (\beta_H E_h^*)/\psi_6$.

Also from (A.3) and (A.5), we get $I_d^* = (\beta_{DD} E_d^*)/(\psi_4 + \gamma_1 (S_h^* + E_h^*))$ and $E_d^* + I_d^* = ((\psi_5 E_h^*)/(S_h^* \psi_2))$ respectively, which can be used to show that $E_d^* = (\psi_5 E_h^* (\psi_4 + \gamma_1 (S_h^* + E_h^*)))/(S_h^* \psi_2 (\psi_4 + \gamma_1 (S_h^* + E_h^*) + \beta_{DD}))$ and $I_d^* = (\beta_{DD} \psi_5 E_h^*)/S_h^* \psi_2 (\psi_4 + \gamma_1 (S_h^* + E_h^*) + \beta_{DD})$.

From (A.2), we get $S_d = ((\psi_3 + \gamma_1 (S_h^* + E_h^*))E_d)/(\psi_1 (E_d + I_d))$ and upon substituting the expression for E_d^*, we obtain $S_d = (\psi_3 + \gamma_1 (S_h^* + E_h^*))(\psi_4 + \gamma_1 (S_h^* + E_h))/(\psi_1 (\psi_4 + \gamma_1 (S_h^* + E_h^*) + \beta_{DD}))$.

Thus, S_d^*, E_d^*, I_d^*, and I_h^* have been expressed in terms of S_h^* and E_h^*.

Equations (A.1) and (A.2) can then be used to find S_h^* and E_h^*.

Substituting S_d^*, E_d^*, I_d^*, and I_h^* into (A.1) and (A.2) (after some long algebraic simplification) gives the following equations:

$$\psi_2 \gamma_1^3 S_h^{*4} + \left(3\psi_2 \gamma_1^3 E_h^* + \gamma_1^2 \psi_2 (\psi_3 + \psi_4 + \mu_d)\right) S_h^{*3}$$
$$+ \left\{3\psi_2 \gamma_1^3 E_h^{*2} + \left(2\gamma_1^2 (\psi_3 + \psi_4)\psi_2 + \gamma_1^2 (\psi_1 \psi_5 + 2\psi_2 \mu_d)\right) E_h^* - \psi_2 \gamma_1 (P_d \psi_1 - \psi_3 \psi_4) + \gamma_1 (\psi_3 + \psi_4)\psi_2 \mu_d\right\} S_h^{*2}$$
$$+ \left\{\psi_2 \gamma_1^3 E_h^{*3} + \left(\gamma_1^2 (\psi_3 + \psi_4)\psi_2 + \gamma_1^2 (2\psi_1 \psi_5 + \psi_2 \mu_d)\right) E_h^{*2} + [(\gamma_1 \psi_1 \psi_5 + \gamma_1 \psi_2 \mu_d)(\psi_3 + \psi_4) - \psi_2 \gamma_1 (P_d \psi_1 - \psi_3 \psi_4)] E_h^*$$
$$- P_d \psi_2 (\psi_4 + \beta_{DD})\psi_1 + \psi_3 \psi_4 \psi_2 \mu_d\right\}$$
$$S_h^* + \gamma_1^2 \psi_5 \psi_1 E_h^{*3} + \gamma_1 (\psi_3 + \psi_4)\psi_1 \psi_5 E_h^{*2} + \psi_5 E_h^* \psi_1 \psi_3 \psi_4 = 0, \quad \text{(A.7)}$$

$$\gamma \psi_5 \psi_2 \gamma_1^2 S_h^{*4} + \left(\gamma \psi_6 \psi_2 \gamma_1 \psi_4 + \gamma \psi_6 \psi_2 \psi_3 \gamma_1 - \psi_1 \psi_2 \psi_6 \mu_h \gamma_1 + 3\gamma \psi_6 \psi_2 \gamma_1^2 E_h\right) S_h^{*3}$$
$$+ \left\{3\gamma \psi_6 \psi_2 \gamma_1^2 E_h^{*2} + [2\gamma \psi_6 \psi_2 \psi_3 \gamma_1 + \theta \psi_1 \psi_2 \beta_h^* \gamma_1 + 2\gamma \psi_6 \psi_2 \gamma_1 \psi_4 - \psi_1 \psi_2 \psi_6 \mu_h^* \gamma_1 + \theta \psi_1 \psi_2 \psi_6 \gamma_1 + \gamma \psi_6 \psi_5 \psi_1 \gamma_1 - \psi_1 \psi_2 \psi_6 \psi_5 \gamma_1] E_h^*\right.$$
$$- \psi_1 \psi_2 \psi_6 \mu_h^* \psi_4 - \psi_1 \psi_2 \psi_6 \mu_h^* \beta_{DD} + P_h^* \psi_1 \psi_2 \psi_6 \gamma_1 + \gamma \psi_6 \psi_2 \psi_3 \psi_4\right\} S_h^{*2}$$
$$+ \left\{\gamma \psi_6 \psi_2 \gamma_1^2 E_h^{*3} + [\gamma \psi_6 \psi_2 \psi_3 \gamma_1 + \gamma \psi_6 \psi_2 \gamma_1 \psi_4 + 2\gamma \psi_6 \psi_5 \psi_1 \gamma_1 + \theta \psi_1 \psi_2 \psi_6 \gamma_1 + \theta \psi_1 \psi_2 \beta_H \gamma_1 - \psi_1 \psi_2 \psi_6 \psi_5 \gamma_1] E_h^{*2}\right.$$
$$+ [\psi_6 \gamma \psi_1 \psi_5 \psi_4 + \psi_6 \gamma \psi_1 \psi_5 \beta_{DD} + \gamma \psi_6 \psi_2 \psi_3 \psi_4 - \psi_1 \psi_2 \psi_6 \psi_5 \psi_4 - \psi_1 \psi_2 \psi_6 \psi_5 \beta_{DD} + \theta \psi_1 \psi_2 \psi_6 \psi_4 + \theta \psi_1 \psi_2 \psi_6 \beta_{DD} + \theta \psi_1 \psi_2 \beta_h^* \psi_4$$
$$+ \theta \psi_1 \psi_2 \beta_h^* \beta_{DD} + P_h^* \psi_1 \psi_2 \psi_6 \gamma_1] E_h^* + P_h^* \psi_1 \psi_2 \psi_6 \beta_{DD} + P_h^* \psi_1 \psi_2 \psi_6 \psi_4\right\}$$
$$S_h^* + \gamma \psi_6 \psi_5 \psi_1 \gamma_1 E_h^{*3} + (\psi_6 \gamma \psi_1 \psi_5 \beta_{DD} + \psi_6 \gamma \psi_1 \psi_5 \psi_4) E_h^{*2} = 0. \quad \text{(A.8)}$$

Equations (A.7) and (A.8) can then be rewritten as in equation (12).

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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