

# The Mathematical Model for *Streptococcus suis* Infection in Pig-Human Population with Humidity Effect

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**Abstract:** In this paper, we developed a mathematical model for *Streptococcus suis*, which is an epidemic by considering the moisture that affects the infection. The disease is caused by *Streptococcus suis* infection found in pigs which can be transmitted to humans. The patients of *Streptococcus suis* were generally found in adults males and the elderly who contacted pigs or who ate uncooked pork. In human cases, the infection can cause a severe illness and death. This disease has an impact to the financial losses in the swine industry. In the development of models for this disease, we have divided the population into 7 related groups which are susceptible pig compartment, infected pig compartment, quarantined pig compartment, recovered pig compartment, susceptible human compartment, infected human compartment, and recovered human compartment. After that, we use this model and a quarantine strategy to analyze the spread of the infection. In addition, the basic reproduction number  $R_0$  is determined by using the next-generation matrix which can analyze the stability of the model. The numerical simulations of the proposed model are illustrated to confirm the results from theorems. The results showed that there is an effect from moisture to the disease transmission. When the moisture increases the disease infection also increases.

**Keywords:** *Streptococcus suis*; mathematical model; stability analysis; infectious disease; reproduction number

## 1 Introduction

Nowadays, one of the major problems in the world is zoonotic pathogen such as Rift Valley fever, SARS, pandemic influenza H1N1 2009, Yellow fever, Avian Influenza (H5N1) and (H7N9), West Nile virus [1], the Middle East respiratory syndrome coronavirus (MERS-CoV) [2], and novel coronavirus (COVID-19) [3]. *Streptococcus suis* is an infectious agent which is widely found in pigs around the world. This pathogen is generally found in the upper respiratory tract, genital, and alimentary tracts of the pigs [4,5]. *Streptococcus suis* is a Gram-positive bacterium and a facultative anaerobic bacterium which is a primary agent of sepsis and meningitis in pigs [6,7]. In pig farms, the disease can transmit



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from pig to pig rapidly. The reported rate of mortality in pigs from *Streptococcus suis* is about 20% [8]. It can classify at least 35 serotypes, for examples, serotypes 1 to 31, 33, and 1/2 [9,10]. Serotypes 1 to 9 and 14 are most commonly found in pigs around the world [11,12].

In 1968, the first human who was infected with *Streptococcus suis* was found in Denmark [4,5]. This situation confirms that *Streptococcus suis* can transmit from pig to human. Serotype 2 is reported that it is frequently found in human infections [13,14], but in some cases, the infections are caused by other serotypes. Kerdsin et al. reported that serotypes 2 and 14 are involved in human cases that found in Thailand [15,16]. Dutkiewicz et al. presented that disease found in human caused by *S. suis* serotypes 2, 4, 5, 14, 16, 21 and 24 [12]. *S. suis* serotype 9 which is the most common found in pigs in Europe was found the first case infection in human in May 2013 locating in northern of Thailand [17]. In addition, Hatrongjit et al. reported the first case of serotype 31 in human [18] in the central region of Thailand. The common symptoms of this disease in human are fever, headache, meningitis, septicemia, arthritis, pneumonia, and hearing loss [19].

To prevent the spread of the disease, the mathematical models become powerful tools for describing the dynamics of the disease. They can forecast the future behaviors of the disease with many assumptions. The solutions of the model can be simulated satisfying the given parameters from the hypothesis. Recently, many mathematical models have been used to describe the behavior of infectious disease [20–24]. Rahman et al. proposed the nonlinear SEITR fractional order model of tuberculosis disease by using Atangana-Baleanu derivative which consists of 5 population groups [25]. Arfan et al. [26] presented the fractional order model to predict the dynamic of tumor with drug intervention. The nonlinear fractional order mathematical model for predicting the dynamic of COVID-19 was created by Shah et al. [27]. Almuqrin et al. proposed the fractional model for forecasting the transmission of Ebola virus in bats population by using Atangana-Baleanu fractional derivative [28]. Srivastava et al. improved the mathematical model for studying the dynamic of diabetes patients [29]. Alzaid et al. [30] studied the dynamic of HIV-1 infection by using the mathematical model and showed that the mathematical model is efficient. The fractional order model of HIV with source term was proposed by Shah et al. to consider the behavior of CD4 + T-cells which depending on the concentration of the viral load [31]. The symmetry and asymmetry concepts can be linked to the epidemic model [32–34].

A number of research studied the disease from *Streptococcus suis* on epidemiology and medicine. However, a few pieces of the research proposed and studied the mathematical model for *Streptococcus suis*. Shen et al. [35] proposed the SIQRW model to explore the outbreaks of *S. Suis*. Giang et al. proposed the stochastic model and SEI model to predict the behavior of the disease and fitted the model parameters with collected data [9]. The proposed models consider the transmission on pig population only. However, to our knowledge, no research considering the disease transmission between pigs and humans for *Streptococcus suis*.

In this paper, we propose a mathematical model to describe the disease transmission of *Streptococcus suis* in humans and pigs. The novelty of this work is the improved mathematical model from one species to two species population. We classify the population of pigs and humans into 4 classes and 3 classes, respectively. This work contains the analysis of the model and numerical simulations to study the dynamic behavior of the disease. We also find the basic reproduction numbers to explain the rapid transmission and study the stability of the solution of the model.

This paper is constructed as follows; In Section 2, we present the mathematical model which expresses the *Streptococcus suis* transmission. The analysis of the proposed model is shown in Section 3 which includes a basic reproduction number, equilibrium points, and their stability. The numerical simulations are given in Section 4 and follow by a conclusion in Section 5.

## 2 Model Formulation

In this section, we describe the model formulation of *Streptococcus suis* disease transmission in humans and pigs. Based on the classical epidemiology model, we propose a new generalized model of *Streptococcus suis* infection which is SIQR-SIR model. There are some facts about the environmental factors such as temperature and relative humidity [36–38]. So, we consider the effect of the moisture in the air for disease transmission in the pig farm.

The investigated population is divided into two subpopulations. These are pig population and human population. Then, both subpopulations of pigs and human are separated into four classes and three classes, respectively. These are a pig susceptible class ( $S_p$ ), pig infectious class ( $I_p$ ), pig isolated class ( $Q_p$ ), pig recovery class ( $R_p$ ), human susceptible class ( $S_h$ ), human infectious class ( $I_h$ ), and human recovery class ( $R_h$ ). We assume that the total population at time  $t$  is  $N(t)$ . We have that

$$S_p(t) + I_p(t) + Q_p(t) + R_p(t) = N_p(t),$$

$$S_h(t) + I_h(t) + R_h(t) = N_h(t),$$

and

$$S_p(t) + I_p(t) + Q_p(t) + R_p(t) + S_h(t) + I_h(t) + R_h(t) = N_p(t) + N_h(t) = N(t),$$

where  $N_p(t)$  is the total population of pigs, and  $N_h(t)$  is the total population of human at time  $t$ .

We can write the dynamic equations for  $N_p(t)$  and  $N_h(t)$  as the following.

$$\frac{dN_p}{dt} = \frac{dS_p}{dt} + \frac{dI_p}{dt} + \frac{dQ_p}{dt} + \frac{dR_p}{dt},$$

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}.$$

The new infection in pigs can be described . where  $\beta_1$  is the transmission coefficient per unit of time per pig in the susceptible class contact with infectious class [39]. The new infection in human can be described by  $\beta_2 S_h I_p + \beta_3 S_h I_h$  where  $\beta_2$  is the transmission coefficient per unit of time per person in the susceptible class in contact with pig infectious class,  $\beta_3$  is the transmission coefficient per unit of time per person in the susceptible class contact with the infectious class. In our model, we investigate the relative humidity for considering the infection of this disease in pigs. Then, the new infection in pigs can be expressed by  $\beta_1 M S_p I_p$  instead of  $\beta_1 S_p I_p$ , where  $M$  is the moisture in the air (relative humidity). In addition, we assume that the disease cannot be transmitted from human to pigs. Throughout this paper, we assume that all parameters are positive constants.

The model of disease transmission by *Streptococcus suis* can be represented by the system of differential equations as follows:

$$\frac{dS_p}{dt} = N_1 - bS_p - \beta_1 M S_p I_p, \quad (1a)$$

$$\frac{dI_p}{dt} = \beta_1 M S_p I_p - aI_p - bI_p - \delta I_p, \quad (1b)$$

$$\frac{dQ_p}{dt} = \delta I_p - aQ_p - bQ_p - \epsilon Q_p, \quad (1c)$$

$$\frac{dR_p}{dt} = \epsilon Q_p - bR_p, \quad (1d)$$

$$\frac{dS_h}{dt} = N_2 - \beta_2 S_h I_p - \beta_3 S_h I_h - \mu S_h, \quad (1e)$$

$$\frac{dI_h}{dt} = \beta_2 S_h I_p + \beta_3 S_h I_h - \alpha I_h - \gamma I_h - \mu I_h, \quad (1f)$$

$$\frac{dR_h}{dt} = \gamma I_h - \mu R_h, \quad (1g)$$

where  $b$  is the pig removal rate,  $a$  is the pig death rate induced by the disease,  $\delta$  is the rate from infectious class to isolated class in pigs,  $e$  is the transition rate from isolated class to recovery class,  $\mu$  is the human natural death rate,  $\alpha$  is the death rate induced by disease, and  $\gamma$  is the transition rate from infectious class to recovery class. The model form is valid only if  $a \leq b$  and  $\alpha \leq \mu$ .

Eq. (1a) expresses the rate of change of the population of susceptible pig. The number of susceptible pigs increases only via birth  $N_1$  described in the first term on the right-hand side. On the other hand, the population of them decreases due to the removal rate of pig ( $b$ ), transmission of *Streptococcus suis* from infected pig to susceptible pig ( $\beta_1$ ) and moisture effect ( $M$ ).

Eq. (1b) expresses the rate of change of the population of the infected pig. On the right-hand side, the first term represents the number of infected pigs increases due to susceptible pig becoming infected pig with the rate  $\beta_1 M$  followed by the negative effects of disease death rate ( $a$ ), removal rate ( $b$ ), and isolated rate ( $\delta$ ).

Eq. (1c) expresses the rate of change of the population of the isolated pig. On the right-hand side, the first term represents the increase in the population of isolated pig due to the isolation of infected pig with the rate  $\delta$  followed by the negative effects of disease death rate ( $a$ ), removal rate ( $b$ ), and recovered rate ( $\epsilon$ ).

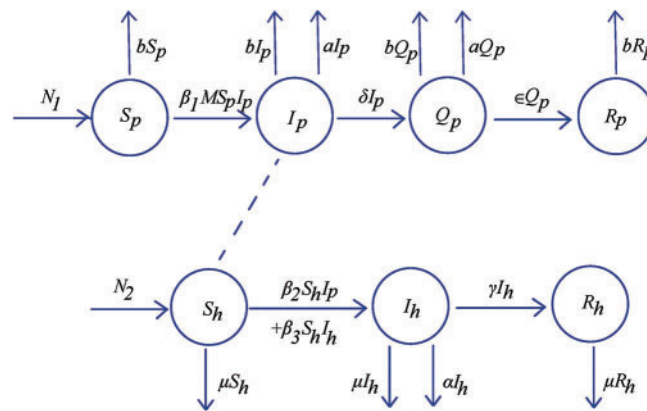
Eq. (1d) expresses the rate of change of the population of the recovered pig. On the right-hand side, the first term represents the increase in the population of the recovered pig due to treatment with the rate  $\epsilon$ , and the last term represents the removal rate ( $b$ ) of pigs.

Eq. (1e) expresses the rate of change of the population of susceptible human. The number of susceptible pigs increases only via birth  $N_2$  described in the first term on the right-hand side. On the other hand, the population of them decreases due to the transmission of *Streptococcus suis* from infected pig to susceptible human ( $\beta_2$ ) and infected human to susceptible human ( $\beta_3$ ). The last term represents the natural death rate ( $\mu$ ) of human.

Eq. (1f) expresses the rate of change of the population of the infected human. On the right-hand side, the first term and second term represent the number of the infected human increases due to susceptible human becoming infected human by contact with the infected pigs ( $\beta_2$ ) and the infected human ( $\beta_3$ ), respectively. The remaining terms are the negative effects of disease death rate ( $\alpha$ ), recovered rate ( $\gamma$ ), and natural death rate ( $\mu$ ).

Eq. (1g) expresses the rate of change of the population of recovered human. On the right-hand side, the first term represents the increase in the population of recovered human due to treatment with the rate ( $\gamma$ ), and the last term represents the natural death rate ( $\mu$ ) of human

Let  $S_p(0) = S_{p0}, I_p(0) = I_{p0}, Q_p(0) = Q_{p0}, R_p(0) = R_{p0}, S_h(0) = S_{h0}, I_h(0) = I_{h0}, R_h(0) = R_{h0}$ . A flowchart of the SIQR-SIR model of pigs and human which describe by the system (1) is shown in Fig 1.



**Figure 1:** Flowchart of SIQR-SIR (Susceptible-Infectious-Isolation-Recovery-Susceptible-Infectious-Recovery) model for human and pigs

The values of population classes  $S_p(t), I_p(t), Q_p(t), R_p(t), S_h(t), I_h(t), R_h(t)$  at time  $t$  is non-negative numbers.

Let

$$N(t) = S_p(t) + I_p(t) + Q_p(t) + R_p(t) + S_h(t) + I_h(t) + R_h(t),$$

and  $c = \min\{b, \mu\}$ . Then we obtain

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS_p}{dt} + \frac{dI_p}{dt} + \frac{dQ_p}{dt} + \frac{dR_p}{dt} + \frac{dS_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} \\ &= N_1 + N_2 - a(I_p + Q_p) - b(S_p + I_p + Q_p + R_p) - \alpha I_h - \mu(S_h + I_h + R_h) \\ &\leq N_1 + N_2 - cN, \end{aligned}$$

and

$$N(t) \leq \frac{N_1 + N_2}{c} + \left( N(0) - \frac{N_1 + N_2}{c} \right) e^{-ct}. \quad (2)$$

Therefore, the positive invariant of the system (1) is

$$\Omega = \left\{ (S_p, I_p, Q_p, R_p, S_h, I_h, R_h) \in R_+^7 : N \leq \frac{N_1 + N_2}{c} \right\}. \quad (3)$$

### 3 Model Analysis

#### 3.1 Equilibria

The equilibria are obtained by setting all equations of the system (1) to be zero.

$$N_1 - bS_p - \beta_1 MS_p I_p = 0,$$

$$\beta_1 MS_p I_p - aI_p - bI_p - \delta I_p = 0,$$

$$\delta I_p - aQ_p - bQ_p - \epsilon Q_p = 0,$$

$$\epsilon Q_p - bR_p = 0, \tag{4}$$

$$N_2 - \beta_2 S_h I_p - \beta_3 S_h I_h - \mu S_h = 0,$$

$$\beta_2 S_h I_p + \beta_3 S_h I_h - \alpha I_h - \gamma I_h - \mu I_h = 0,$$

$$\gamma I_h - \mu R_h = 0.$$

By solving the system (4), We get three equilibrium points:

i) Disease-free equilibrium

$$E_1 = \left( \frac{N_1}{b}, 0, 0, 0, \frac{N_2}{\mu}, 0, 0 \right).$$

ii) Pig disease free equilibrium

$$E_2 = \left( \frac{N_1}{b}, 0, 0, 0, \frac{A}{\beta_3}, \frac{\beta_3 N_2 - \mu A}{\beta_3 A}, \frac{\gamma}{\mu} \frac{\beta_3 N_2 - \mu A}{\beta_3 A} \right),$$

where  $A \equiv \alpha + \gamma + \mu$ .

Note that,  $E_2$  exists if  $\beta_3 N_2 - \mu A > 0$ .

iii) The endemic equilibrium

$$E^* = (S_p^*, I_p^*, Q_p^*, R_p^*, S_h^*, I_h^*, R_h^*),$$

where

$$S_p^* = \frac{B}{\beta_1 M},$$

$$I_p^* = \frac{\beta_1 M N_1 - bB}{\beta_1 B M},$$

$$Q_p^* = \frac{\delta I_p^*}{(a + b + \epsilon)},$$

$$R_p^* = \frac{\epsilon \delta I_p^*}{b(a + b + \epsilon)},$$

$$I_h^* = \frac{1}{2\beta_3 A} \left( X + \sqrt{X^2 + 4A\beta_2\beta_3 N_2 I_p^*} \right),$$

$$S_h^* = \frac{N_2}{\beta_2 I_p^* + \beta_3 I_h^* + \mu},$$

$$R_h^* = \frac{\gamma I_h^*}{\mu},$$

with  $X \equiv \beta_3 N_2 - A(\beta_2 I_p^* + \mu)$  and  $B \equiv a + b + \delta$ .

Note that,  $E^*$  exists if  $\beta_1 M N_1 - bB > 0$ .

### 3.2 Basic Reproduction Number

The basic reproduction number is the expected number of secondary cases produced by a single infection in a completely susceptible population. To compute the basic reproduction number and to study the local stability of the equilibrium, we use the next generation matrix method [40,41]. We define  $\mathbf{x}' = f(\mathbf{x}) - v(\mathbf{x})$ , where  $\mathbf{x} = [I_p, Q_p, I_h]^T$  where  $f(\mathbf{x})$  is the matrix of new infection terms, and  $v(\mathbf{x})$  is the matrix of transfer terms into compartment and out of compartment as:

$$f = \begin{bmatrix} \beta_1 M S_p I_p \\ 0 \\ \beta_2 S_h I_p + \beta_3 S_h I_h \end{bmatrix}, v = \begin{bmatrix} a I_p + b I_p + \delta I_p \\ \delta I_p + b Q_p + \epsilon Q_p \\ \alpha I_h + \gamma I_h + \mu I_h \end{bmatrix}.$$

The corresponding Jacobian matrices are

$$F = \begin{bmatrix} \beta_1 M S_p & 0 & 0 \\ 0 & 0 & 0 \\ \beta_2 S_h & 0 & \beta_3 S_h \end{bmatrix}, V = \begin{bmatrix} B & 0 & 0 \\ -\delta & a + b + \epsilon & 0 \\ 0 & 0 & A \end{bmatrix}.$$

Then, we obtain

$$\begin{aligned} FV^{-1} &= \begin{bmatrix} \frac{\beta_1 M S_p}{B} & 0 & 0 \\ 0 & 0 & 0 \\ \frac{\beta_2 S_h}{B} & 0 & \frac{\beta_3 S_h}{A} \end{bmatrix} \Big|_{E_1} \\ &= \begin{bmatrix} \frac{\beta_1 M N_1}{bB} & 0 & 0 \\ 0 & 0 & 0 \\ \frac{\beta_2 N_2}{\mu B} & 0 & \frac{\beta_3 N_2}{\mu A} \end{bmatrix} \end{aligned}$$

We obtain three eigenvalues of  $FV^{-1}$  as:

$$\lambda_1 = 0, \lambda_2 = \frac{\beta_3 N_2}{\mu A}, \lambda_3 = \frac{\beta_1 M N_1}{bB}.$$

Hence, the spectral radii are  $R_{01} = \frac{\beta_3 N_2}{\mu A}$ , and  $R_{02} = \frac{\beta_1 M N_1}{bB}$ .

### 3.3 The Stability of Disease-Free Equilibrium ( $E_1$ )

**Theorem 1.** The disease-free equilibrium point ( $E_1$ ) is locally asymptotically stable if  $R_{01} < 1$  and  $R_{02} < 1$ .

**Proof.** The model system (1) has the Jacobian matrix at the point  $E_1$  as:

$$J(E_1) = \begin{bmatrix} -b & -\frac{\beta_1 M N_1}{b} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_1 M N_1}{b} - B & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta & -(a+b+\epsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -b & 0 & 0 & 0 \\ 0 & -\frac{\beta_2 N_2}{\mu} & 0 & 0 & -\mu & -\frac{\beta_3 N_2}{\mu} & 0 \\ 0 & \frac{\beta_2 N_2}{\mu} & 0 & 0 & 0 & \frac{\beta_3 N_2}{\mu} - A & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -\mu \end{bmatrix}$$

The corresponding eigenvalues of  $J(E_1)$  are

$$\lambda_1 = \lambda_2 = -b, \lambda_3 = \lambda_4 = -\mu, \lambda_5 = -(a+b+\epsilon),$$

$$\lambda_6 = \frac{\beta_1 M N_1}{bB} - B = B(R_{02} - 1),$$

$$\lambda_7 = \frac{\beta_3 N_2}{\mu} - A = A(R_{01} - 1).$$

Note that  $\lambda_6$  is negative if  $R_{02} < 1$  and  $\lambda_7$  is negative if  $R_{01} < 1$ .

Therefore, the disease-free equilibrium ( $E_1$ ) of the model system (1) is locally asymptotically stable due to all negative real part eigenvalues if  $R_{01} < 1$  and  $R_{02} < 1$ .  $\square$

### 3.4 The Stability of Pig Disease Free Equilibrium ( $E_2$ )

**Theorem 2.** The pig disease free equilibrium ( $E_2$ ) is locally asymptotically stable if  $R_{01} > 1$  and  $R_{02} < 1$ .

**Proof.** The pig disease-free equilibrium

$$E_2 = \left( \frac{N_1}{b}, 0, 0, 0, \frac{A}{\beta_3}, \frac{\beta_3 N_2 - \mu A}{\beta_3 A}, \frac{\gamma \beta_3 N_2 - \mu A}{\mu \beta_3 A} \right)$$

exists and is positive if  $R_{01} > 1$ .

The model system (1) has the Jacobian matrix at the point  $E_2$  as:

$$J(E_2) = \begin{bmatrix} -b & -\frac{\beta_1 M N_1}{b} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_1 M N_1}{b} - B & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta & -(a+b+\epsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -b & 0 & 0 & 0 \\ 0 & -\frac{\beta_2 N_2}{\mu} & 0 & 0 & -\mu R_{01} & -A & 0 \\ 0 & \frac{\beta_2 N_2}{\mu} & 0 & 0 & \mu(R_{01} - 1) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -\mu \end{bmatrix}$$



The corresponding eigenvalues of  $J(E_2)$  are

$$\lambda_1 = \lambda_2 = -b, \lambda_3 = -\mu, \lambda_4 = -(a + b + \epsilon),$$

$$\lambda_5 = \frac{\beta_1 M N_1}{b} - B = B(R_{02} - 1),$$

$$\lambda_6 = \frac{-\mu R_{01} + \sqrt{\mu^2 R_{01}^2 - 4\mu A(R_{01} - 1)}}{2},$$

$$\lambda_7 = \frac{-\mu R_{01} - \sqrt{\mu^2 R_{01}^2 - 4\mu A(R_{01} - 1)}}{2}.$$

Note that  $\lambda_5$  is negative if  $R_{02} < 1$  while  $\lambda_6$  and  $\lambda_7$  have negative real part if  $R_{01} > 1$ .

Therefore, the pig disease free equilibrium ( $E_2$ ) of the system (1) is locally asymptotically stable if  $R_{01} > 1$  and  $R_{02} < 1$ .  $\square$

### 3.5 The Stability of Endemic Equilibrium ( $E^*$ )

**Theorem 3.** The endemic equilibrium ( $E^*$ ) is locally asymptotically stable if  $R_{01} < 1$  and  $R_{02} > 1$ .

**Proof.** The endemic equilibrium

$$E^* = (S_p^*, I_p^*, Q_p^*, R_p^*, S_h^*, I_h^*, R_h^*),$$

where

$$S_p^* = \frac{N_1}{bR_{02}}$$

$$I_p^* = \frac{b(R_{02} - 1)}{\beta_1 M}$$

$$Q_p^* = \frac{\delta I_p^*}{(a + b + \epsilon)}$$

$$R_p^* = \frac{\epsilon \delta I_p^*}{b(a + b + \epsilon)}$$

$$I_h^* = \frac{1}{2\beta_3 A} \left( X + \sqrt{X^2 + 4A\beta_2\beta_3 N_2 I_p^*} \right)$$

$$S_h^* = \frac{N_2}{\beta_2 I_p^* + \beta_3 I_h^* + \mu}$$

$$R_h^* = \frac{\gamma I_h^*}{\mu}$$

exists and is positive if  $R_{02} > 1$ .

The model system (1) has the Jacobian matrix at the point  $E^*$  as:

$$J(E_2) = \begin{bmatrix} -b - \Psi_1 & -\Psi_2 & 0 & 0 & 0 & 0 & 0 \\ \Psi_1 & \Psi_2 - B & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta & -(a + b + \epsilon) & 0 & 0 & 0 & 0 \\ 0 & 0 & \epsilon & -b & 0 & 0 & 0 \\ 0 & -\beta_2 S_h^* & 0 & 0 & -\Psi_3 - \mu & -\beta_3 S_h^* & 0 \\ 0 & \beta_2 S_h^* & 0 & 0 & \Psi_3 & \beta_3 S_h^* - A & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -\mu \end{bmatrix}$$

where

$$\Psi_1 = \frac{\beta_1 M N_1 (R_{02} - 1)}{B R_{02}}, \Psi_2 = \frac{M \beta_1 N_1}{b R_{02}}, \Psi_3 = \frac{-\beta_2 N_1 (R_{02} - 1)}{B R_{02}} + \beta_3 I_h^*.$$

The corresponding eigenvalues of  $J(E^*)$  are

$$\lambda_1 = -b, \lambda_2 = -\mu, \lambda_3 = -(a + b + \epsilon),$$

$$\lambda_4 = \frac{-[\beta_1 M (I_p^* - S_p^*) + B + b] + Y_1}{2},$$

$$\lambda_5 = \frac{-[\beta_1 M (I_p^* - S_p^*) + B + b] - Y_1}{2},$$

$$\lambda_6 = \frac{-[\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu] + Y_2}{2},$$

$$\lambda_7 = \frac{-[\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu] - Y_2}{2},$$

where

$$Y_1 = \sqrt{[\beta_1 M (I_p^* - S_p^*) + B + b]^2 - 4[\beta_1 M (B I_p^* - b S_p^*) + B b]},$$

and

$$Y_2 = \sqrt{[\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu]^2 - 4[\beta_2 A I_p^* + \beta_3 A I_h^* + \mu(A - \beta_3 S_h^*)]}.$$

Next, we consider

$$\begin{aligned} \beta_1 M (I_p^* - S_p^*) + B + b &= \beta_1 M \left( \frac{b(R_{02} - 1)}{\beta_1 M} - \frac{N_1}{b R_{02}} \right) + B + b \\ &= b(R_{02} - 1) - \frac{\beta_1 M N_1}{b R_{02}} + B + b \end{aligned}$$

$$= bR_{02}$$

$$> 0,$$

and

$$\begin{aligned}\beta_1 M(BI_p^* - bS_p^*) + Bb &= \beta_1 M \left( \frac{Bb(R_{02} - 1)}{\beta_1 M} - \frac{bN_1}{bR_{02}} \right) + Bb \\ &= bB(R_{02} - 1) - \frac{b\beta_1 MN_1}{bR_{02}} + Bb \\ &= Bb(R_{02} - 1).\end{aligned}$$

We obtain that  $\lambda_4$  and  $\lambda_5$  has negative real part if  $R_{02} > 1$ .

Then, let us consider

$$\begin{aligned}\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu \\ &= \beta_2 I_p^* + \beta_3 \left( I_h^* - \frac{N_2}{\beta_2 I_p^* + \beta_3 I_h^* + \mu} \right) + A + \mu \\ &= \frac{\beta_3^2 I_h^{*2} + [2\beta_2 \beta_3 I_p^* + 2\mu \beta_3 + \beta_3 A] I_h^* + \beta_2^2 I_p^{*2} + (A + 2\mu) \beta_2 I_p^* + \mu(A + \mu) - \beta_3 N_2}{\beta_2 I_p^* + \beta_3 I_h^* + \mu} \\ &= \frac{[\beta_3^2 + \beta_3 A] I_h^{*2} + [2\beta_2 \beta_3 I_p^* + \beta_3 (2\mu + A - N_2) + A(\beta_2 I_p^* + \mu)] I_h^*}{\beta_2 I_p^* + \beta_3 I_h^* + \mu} \\ &\quad + \frac{\beta_2 I_p^* (\beta_2 I_p^* + A + 2\mu - N_2) + \mu(A + \mu) - \beta_3 N_2}{\beta_2 I_p^* + \beta_3 I_h^* + \mu}.\end{aligned}$$

Since  $I_h^* = \frac{1}{2\beta_3 A} (X + \sqrt{X^2 + 4A\beta_2 \beta_3 N_2 I_p^*})$ , it follows that

$$\beta_3 A I_h^{*2} - [\beta_3 N_2 - A(\beta_2 I_p^* + \mu)] I_h^* - \beta_2 N_2 I_p^* = 0.$$

Then, we get

$$\begin{aligned}\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu \\ &= \frac{\beta_3^2 I_h^{*2} + [2\beta_2 \beta_3 I_p^* + \beta_3 (2\mu + A)] I_h^* + \beta_2 I_p^* (\beta_2 I_p^* + A + 2\mu) + \mu^2 + \mu A (1 - R_{01})}{\beta_2 I_p^* + \beta_3 I_h^* + \mu}.\end{aligned}$$

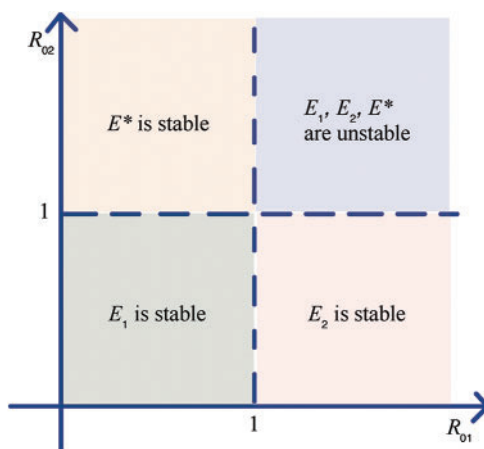
If  $R_{01} < 1$ ,  $I_p^*$  and  $I_h^*$  are positive i.e.,  $R_{02} > 1$ , then we obtain

$$\beta_2 I_p^* + \beta_3 (I_h^* - S_h^*) + A + \mu > 0.$$

Thus, the eigenvalues  $\lambda_5$  and  $\lambda_6$  have a negative real part if  $R_{01} < 1$  and  $R_{02} > 1$ .

Therefore, the endemic equilibrium ( $E^*$ ) of the model system (1) is locally asymptotically stable if  $R_{01} < 1$  and  $R_{02} > 1$ .  $\square$

We can draw the bifurcation diagram to describe the basic reproduction numbers and stability as shown in Fig. 2.



**Figure 2:** The bifurcation region

#### 4 Numerical Examples and Discussion

The numerical results of the system (1) are computed by using MATLAB with the given initial values:

$$S_p(0) = 4057, I_p(0) = 1000, Q_p(0) = 0, R_p(0) = 0, S_h(0) = 50000, I_h(0) = 1000, R_h(0) = 0.$$

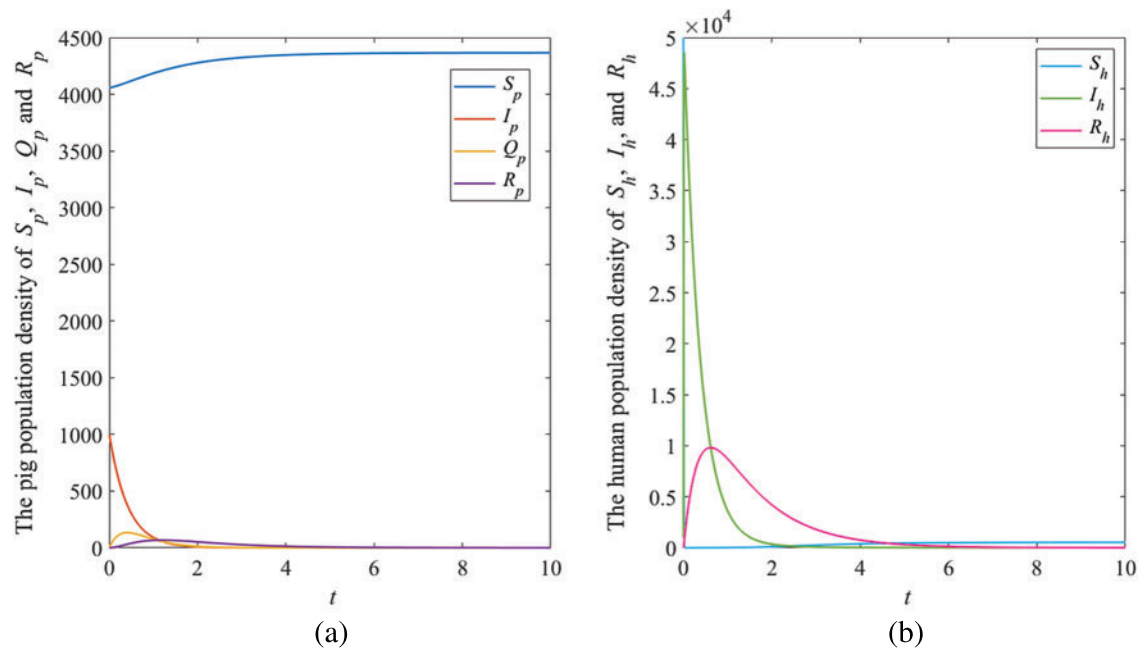
The numerical results of the system (1) with the parameter values as shown in Tab. 1. Note that the parameters and initial values are obtained from data in [35] and [42].

**Table 1:** Parameter values of the system (1)

Parameter	Value	Source
$N_1$	3275	Taken from [35]
$b$	0.75	Taken from [35]
$\beta_1$	0.000365	Taken from [35]
$N_2$	500	Calculated
$\beta_3$	0.00465	Assumed

The solution trajectories tend to the disease-free equilibrium ( $E_1$ ) which satisfy Theorem 1 with the remaining parameter values  $\mu = 0.9, \alpha = 0.9, \gamma = 0.9, M = 0.9, a = 0.9, \beta_2 = 0.1$ , and  $\delta = 0.9$  as

shown in Fig. 3. The calculated reproduction numbers of this case are  $R_{01} = 0.9568 < 1$  and  $R_{02} = 0.5625 < 1$ .

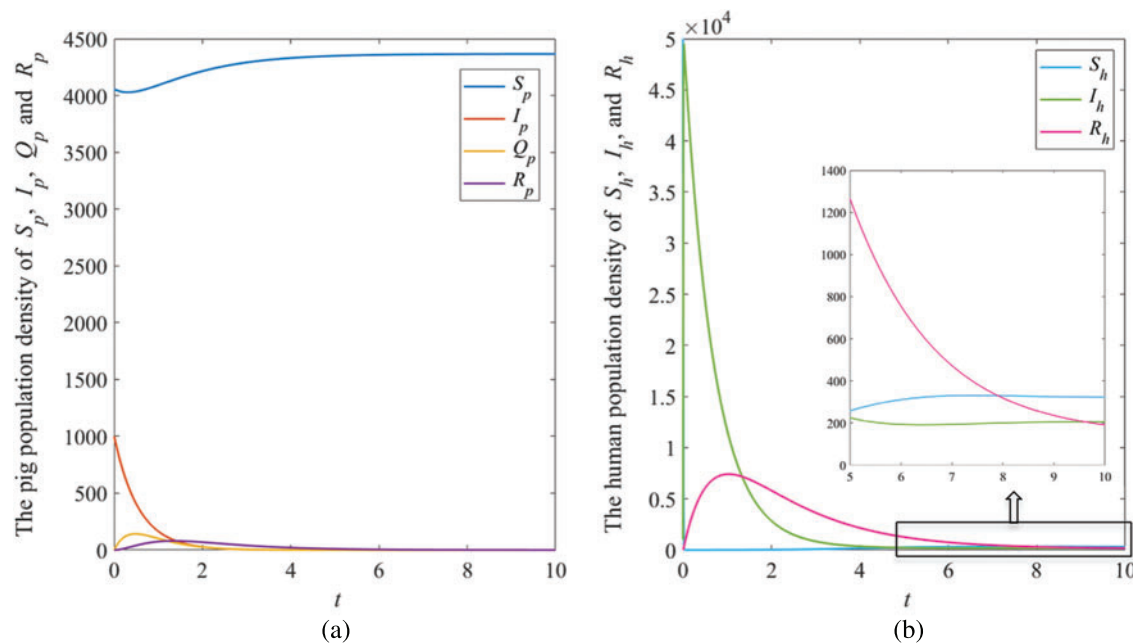


**Figure 3:** The simulation results of the system (1), (a) The plots of asymptotic population density of pig susceptible population ( $S_p$ ), pig infectious population ( $I_p$ ), pig isolated population ( $Q_p$ ), and pig recovery population ( $R_p$ ) with respect to time  $t$  tends to the equilibrium ( $E_1$ ), and (b) The plots of asymptotic population density of human susceptible population ( $S_h$ ), human infectious population ( $I_h$ ), and human recovery population ( $R_h$ ) with respect to time  $t$  tends to the equilibrium ( $E_1$ )

The results showed that the number of pig infectious cases dramatically decreased in the first quarter. After that, the decreasing of pig infectious cases slowly decreased. Then, it tended to the equilibrium value. Fig. 3a, the number of pig susceptible population increased after infectious cases were recovered. In addition, the isolated population increased when the number of infectious cases increased to control the spread of the disease. Then, it decreased after the number of infectious cases decreased. In human case (Fig. 3b), the number of infectious populations increased in the first quarter, then the number increased and approached to the equilibrium number. Therefore, the parameters of this case provided that the disease died out.

The solution trajectories tend to the disease-free equilibrium ( $E_2$ ) which satisfy Theorem 2 with the remaining parameter values  $\mu = 0.6, \alpha = 0.5, \gamma = 0.4, M = 0.3, a = 0.7, \beta_2 = 0.1$ , and  $\delta = 0.8$  as shown in Fig. 4. The calculated reproduction numbers of this case are  $R_{01} = 2.5833 > 1$  and  $R_{02} = 0.2125 < 1$ .

The results showed that the number of pig infectious cases dramatically decreased in the first quarter. After that, the decreasing of pig infectious cases slowly decreased. Then, it tended to the equilibrium value. The number of pig susceptible population increased after infectious cases decreased. In human case (Fig. 4b), the number of infectious populations dramatically increased in the first quarter, then it decreased and approached to the equilibrium number. Therefore, the result of this case indicated that the disease in pig died out while it still appeared in human.



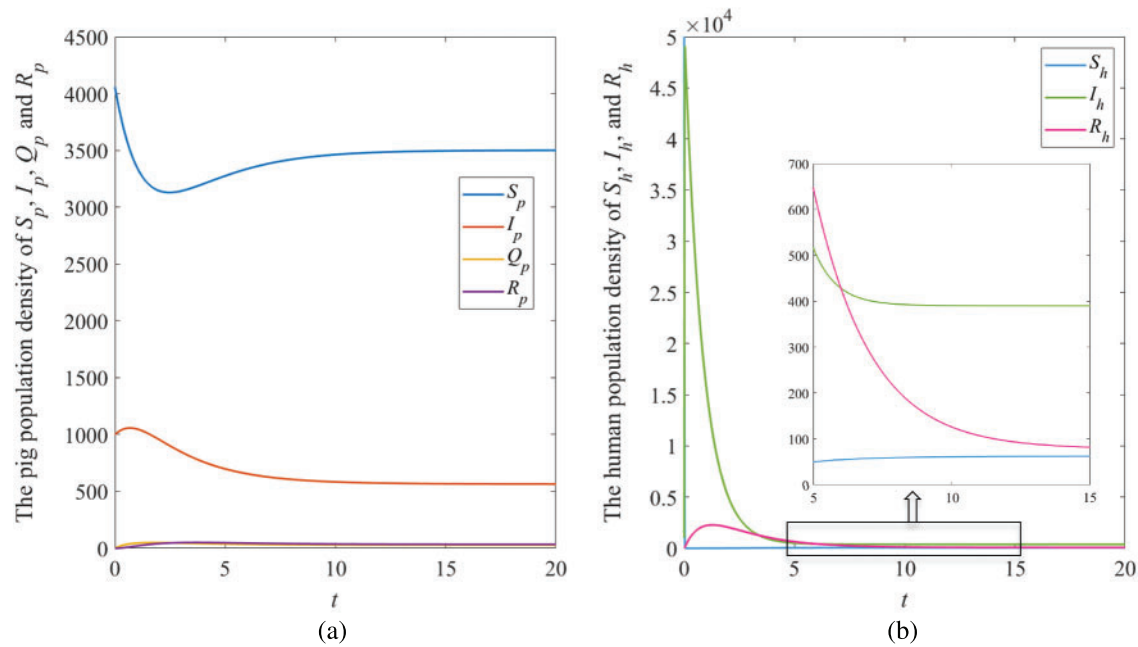
**Figure 4:** The simulation results of the system (1), (a) The plots of asymptotic population density of pig susceptible population ( $S_p$ ), pig infectious population ( $I_p$ ), pig isolated population ( $Q_p$ ), and pig recovery population ( $R_p$ ) with respect to time  $t$  tends to the equilibrium ( $E_2$ ), and (b) The plots of asymptotic population density of human susceptible population ( $S_h$ ), human infectious population ( $I_h$ ), and human recovery population ( $R_h$ ) with respect to time  $t$  tends to the equilibrium ( $E_2$ )

The numerical results of the system (1) with the remaining parameter values  $\mu = 0.5$ ,  $\alpha = 0.6$ ,  $\gamma = 0.1$ ,  $M = 0.9$ ,  $a = 0.3$ ,  $\beta_2 = 0.01$ , and  $\delta = 0.1$  with  $R_{01} = 0.9568 < 1$  and  $R_{02} = 1.2473 > 1$ . The solution trajectories tend to the endemic equilibrium ( $E^*$ ) which satisfy Theorem 3 as shown in Fig. 5. The number of pig susceptible population decreased in the first five quarters then it increased and approached to the equilibrium. The number of pig infectious population increase in the first quarter then it tends to equilibrium after five quarters. In human case, the number of human infectious population increased sharply in the first two quarters. After that, it decreased and tend to equilibrium value. The number of recovery population increased in the first quarter then it decreased and approached to equilibrium value. The result of this case showed that the disease still appeared in both human and pigs.

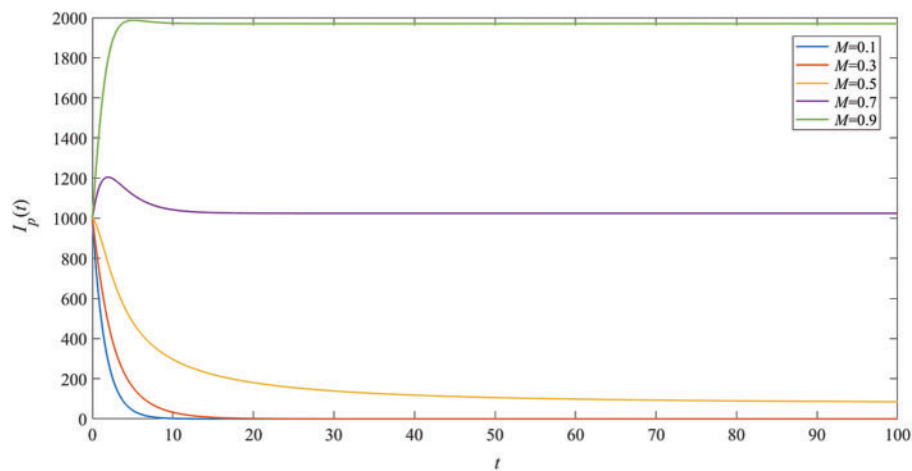
To consider the effect of moisture, the numerical results of the system (1) with the remaining parameter values  $\mu = 0.5$ ,  $\alpha = 0.6$ ,  $\gamma = 0.1$ ,  $a = 0.01$ ,  $\beta_2 = 0.01$ , and  $\delta = 0.01$ . The solutions trajectories are computed with various values of relative humidity  $M$  as 0.1, 0.3, 0.5, 0.7, and 0.9 is shown in Fig. 6.

In Fig. 6, the result indicates that when the relative humidity ( $M$ ) increases the pig infectious population density also increases. Particularly, the trajectories of pig infectious population decrease and approach to constant values when  $M = 0.1$ , 0.3, and 0.5. On the other hand, the number of pig infectious population increase and go to equilibrium value when  $M = 0.7$  and 0.9. Moreover, when  $M = 0.1$  and 0.3 the trajectories approach zero. This means that the disease will die out when there is

less moisture in the air. Therefore, the result confirms that there is an effect of the moisture in the air to the infection of *Streptococcus suis* as mentioned in the literature.



**Figure 5:** The simulation results of the system (1), (a) The plots of asymptotic population density of pig susceptible population ( $S_p$ ), pig infectious population ( $I_p$ ), pig isolated population ( $Q_p$ ), and pig recovery population ( $R_p$ ) with respect to time  $t$  tends to the equilibrium ( $E^*$ ), and (b) The plots of asymptotic population density of human susceptible population ( $S_h$ ), human infectious population ( $I_h$ ), and human recovery population ( $R_h$ ) with respect to time  $t$  tends to the equilibrium ( $E^*$ )



**Figure 6:** The simulation results of the pig infectious population ( $I_p$ ) for various values of relative humidity  $M$  with respect to time  $t$

## 5 Conclusions

We have proposed the mathematical model for predicting the disease transmission of *Streptococcus suis* between pigs and humans, which is developed from the SIQR model that considered only in the pig population. The pigs-human epidemiology model of *Streptococcus suis* transmission which is SIQR-SIR model is investigated. The model combined with susceptible ( $S_p$ ), infection ( $I_p$ ), isolation ( $Q_p$ ), and recovery ( $R_p$ ) for pig population and susceptible ( $S_h$ ), infectious ( $I_h$ ), and recovery ( $R_h$ ) for human population. Moreover, we studied the effect of moisture in the air on disease transmission. The equilibria of the model were analyzed. Then, the next-generation matrix was used to find the basic reproduction numbers  $R_{01}$ , and  $R_{02}$ . We obtained the conditions of the equilibrium points  $E_1$ ,  $E_2$ , and  $E^*$  which are locally asymptotically stable in Theorem 1, Theorem 2, and Theorem 3, respectively. The results show that if  $R_{01} < 1$  and  $R_{02} < 1$ , the equilibrium points  $E_1$  is locally asymptotically stable while  $E_2$  is locally asymptotically stable if  $R_{01} > 1$  and  $R_{02} < 1$ . Finally, the endemic equilibrium ( $E^*$ ) is locally asymptotically stable if  $R_{01} < 1$  and  $R_{02} > 1$ . The bifurcation diagram is also presented. The numerical examples confirmed the results of Theorems. The results indicated that moisture affects disease transmission. When the air has higher moisture the transmission of the pathogen is also high. Moreover, the model can be improved by considering infection of newborn pigs from infectious adult female pigs.

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