Research Article

Dynamical Analysis of the *SEIB* Model for Brucellosis Transmission to the Dairy Cows with Immunological Threshold

Wei Zhang,^{1,2} Juan Zhang,^{1,2} Yong-Ping Wu ,³ and Li Li ^{2,4}

¹Complex Systems Research Center, Shanxi University, Taiyuan, Shanxi 030006, China

²Shanxi Key Laboratory of Mathematical Techniques and Big Data Analysis on Disease Control and Prevention, Shanxi University, Shanxi 030006, China

³College of Physics Science and Technology, Yangzhou University, Yangzhou, Jiangsu Province 225002, China

⁴School of Computer and Information Technology, Shanxi University, Shanxi, Taiyuan 030006, China

Correspondence should be addressed to Yong-Ping Wu; ypwu@yzu.edu.cn and Li Li; lili831113@sxu.edu.cn

Received 23 February 2019; Revised 22 April 2019; Accepted 28 April 2019; Published 23 May 2019

Academic Editor: Toshikazu Kuniya

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As we all know, bacteria is different from virus which with certain types can be killed by the immune cells in the body. The brucellosis, a bacterial disease, can invade the body by indirect transmission from environment, which has not been researched by combining with immune cells. Considering the effects of immune cells, we put a minimum infection dose of brucellosis invading into the dairy cows as an immunological threshold and get a switch model. In this paper, we accomplish a thorough dynamics analysis of a *SEIB* switch model. On the one hand, we can get a disease-free and bacteria-free steady state and up to three endemic steady states which may be thoroughly analyzed in different cases of a minimum infection dose in a switch model. On the other hand, we calculate the basic reproduction number R_0 and know that the disease-free and bacteria-free steady state is a global stability when $R_0 < 1$, and the one of the endemic steady state is a conditionally global stability when $R_0 > 1$. We find that different amounts of R_0 may lead to different steady states of brucellosis, and considering the effects of immunology is more serious in mathematics and biology.

1. Introduction

Brucellosis is the zoonotic sex contagion which is named as Mediterranean relax heat, wave heat, or wave form heat [1–3]. It is characterized by extensive host, strong infectivity, and difficulty in radical treatment after infection [4, 5]. Besides, it has a serious harm to economy, society, and public health. Therefore, brucellosis is listed as one of the communicable diseases that must be notified in the World Organization for Animal Health and is classified as second kind of animal diseases in China. Meanwhile, it is as the first zoonotic disease to be controlled in the National Medium and Long Term Plan for Animal Disease Prevention and Control 2011–2020 formulated by the ministry of agriculture. Brucellosis is acute or chronic infectious disease caused by *Brucella* which is a group of small bulbous gram-negative bacteria [6, 7]. In 1985, the World Health Organization (WHO) divided *Brucella* into

six species and nineteen biological types [7]. They are more popular with *Br. melitensis*, *Br. brovis*, and *Br. suis* in China, among which the most popular *Brucella* is *Br. melitensis* and the second is *Br. brovis*. The most noticeable symptom of brucellosis is miscarriage in cows and orchitis in bulls. In addition, it can cause arthritis in the knee and wrist frequently [2, 3]. The route of spread is through direct contact with diseased animals and indirect infection with bacteria in the environment [8, 9]. And it can be divided into three ways. The first transmitted way is contacting with skin, such as direct contact with droppings, vaginal secretions, and vaginal delivery content of sick animals. It also can be indirectly exposed to the environment and objects contaminated by sick animals. The second infected way is via the digestive tract, like eating food, water, or milk contaminated with pathogens. And the last way of transmission is through the respiratory tract.

TABLE 1: The description of parameters in original model.

Parameter	Description	Dimension
A	The annual introduction number of dairy cows	$year^{-1}$
q	The annual birth rate of dairy cows	$year^{-1}$
d	The annual natural elimination rate	$year^{-1}$
δ	Clinical outcome rate	$year^{-1}$
c	The annual elimination rate for the positive cows	$year^{-1}$
m	The annual quantity of Brucella	$year^{-1}$
w	The annual natural mortality of Brucella	$year^{-1}$
β_1	Cow-to-cow transmission rate	none
β_2	Brucella-to-cow transmission rate	none
h	The sterilizing rate in a disinfection	none
e	The number of disinfections every year	once

When bacteria enter the body, mainly in the liver, spleen, bone marrow, lymphatic tissue, and other cells, those will grow, multiply, and produce endotoxin which can cause damaged tissues. In the meantime, it often releases bacteria and toxins into the blood causing systemic bacteremia and allergic reactions. But the Brucella does not take effect immediately when it enters the body. The bacteria with a small number will be killed by immune cells and only amounts of bacteria over a certain threshold called a minimum infection dose (MID) in environment can do harm to body [10, 11]. And different bacteria have variant thresholds which should be tested by experiment. Some bacteria threshold can be seen in [12]. Also, there were many authors researching the brucellosis, who had been unconscious of the impact on immunization [13–19]. By referring to [20, 21], we will put the pathogeny of brucellosis and MID into this paper. The authors [12, 20, 21] incorporated a MID, c , into the incidence term $\alpha(B)$, in a cholera transmission model, which was a piecewise continuous function which was zero under the MID and was a *Holling – II* response curve over that.

$$\alpha(B) = \begin{cases} 0 & \text{if } B < c, \\ \frac{a(B-c)}{(B-c)+H} & \text{if } B \geq c. \end{cases} \quad (1)$$

where the parameter c represents MID, H is half-saturation pathogen density, and the description of parameter a is maximum rate of infection. One of typical mathematical models given in [22] is as follows:

$$\begin{aligned} \dot{S} &= A + q(S + E + I) - \beta_1 SI - \beta_2 SV - dS, \\ \dot{E} &= \beta_1 SI + \beta_2 SV - dE - \delta E, \\ \dot{I} &= \delta E - dI - cI, \\ \dot{B} &= m(E + I) - (w + eh)B. \end{aligned} \quad (2)$$

where all parameters are positive and the description of parameters can be found in Table 1.

In [22], the authors gave a detailed demonstration of the basic reproduction number R_0 ($R_0 = (A(w+eh)\beta_1\delta + Am(d+c+\delta)\beta_2)/(d+c)(d+\delta)(w+eh)(d-q)$), which can estimate

the occurrence of the epidemic, and made brief dynamical analysis on the global stability.

Basing on model (2) and considering the immunology of Brucella in the environment, we get a switch system with MID (M). If the number of Brucella in environment (B) is less than or equal to the MID, then we can get a globally asymptotically stable disease-free and bacteria-free steady state while the basic reproduction number is less than or equal to 1, an unstable disease-free and bacteria-free equilibrium and a conditionally globally stable endemic steady state when the basic reproduction number is more than 1. Else if $B > M$, it is going to be a little bit complicated. Combining with the relationship between parameter M and M_g (which is the formula received from the system), we can attain the three different switch systems. One of them can attain four equilibria and can produce a backward bifurcation [23, 24] and might be two steady states at most under certain parameter conditions.

2. Dynamical Analysis of the Model

2.1. The Dynamical Model. We classify the dairy cows into three compartments: the susceptible compartment S , the exposed compartment E , and the infectious compartment I in Figure 1. And the Brucella in environment is denoted by B .

One of the key differences of (2) is the incidence term $\alpha(B)S$ of indirect transmission of Brucella to susceptible cow [12], where $\alpha(B)$ is the pathogen density dependent component. Unlike [12, 20] using *Holling – II* response curve when the bacteria density is over the MID, a simple linear form is used in this paper. There are chiefly the following reasons. Firstly, the half-saturation pathogen density (H) is difficult to be determined in actual application. Secondly, the Brucella which is discharged to the nature by dairy cows and other livestock is hard to reach saturation. Lastly, that can get more practical conclusions with immunological threshold than [22]. The expression of $\alpha(B)$ in this paper is defined as

$$\alpha(B) = \begin{cases} 0 & \text{if } 0 \leq B \leq M, \\ a(B-M) & \text{if } B > M. \end{cases} \quad (3)$$

FIGURE 1: A flow diagram of brucellosis transmission.

TABLE 2: The description of new parameters in model (4).

Parameter	Description	Dimension
M	MID	cell liter ⁻¹
a	Maximum rate of infection	none
β	Indirect transmission rate	none

If the number of bacteria is more than MID, then the Brucella of environment can bring the indirect influence to the dairy cows. At this time, a is representative of maximum rate of indirect infection, and M is denoted MID of Brucella, $B - M$ is representative of bacteria amount which can enter the body and take effect. If the number is less than or equal to MID, then the bacteria of environment have no effect on the susceptible and dairy cows were infected just by the direct transmission from infected to susceptible cow.

So the model is a system of ordinary differential equation as follows:

$$\begin{aligned}
 \dot{S} &= A + q(S + E + I) - \beta SI - dS - \alpha(B)S, \\
 \dot{E} &= \beta SI + \alpha(B)S - dE - \delta E, \\
 \dot{I} &= \delta E - dI - cI, \\
 \dot{B} &= m(E + I) - (w + eh)B,
 \end{aligned} \tag{4}$$

where the description of other parameters which are not mentioned in Table 1 can be found in Table 2.

Remark 1. In this paper, we also use the condition $d > q$ like [22].

2.2. Forward Invariance. The first equation of (4) gives us that $\dot{S}(t) \geq 0$, when $S = 0$. In the same way, we can get $\dot{E}(t) \geq 0$ and $\dot{I}(t) \geq 0$. Hence, we know that $S(t) \geq 0$, $E(t) \geq 0$, and $I(t) \geq 0$ for $t > 0$. As $\dot{S} + \dot{E} + \dot{I} \leq A - (d - q)(S + E + I)$, we can get $0 \leq S + E + I \leq A/(d - q)$ by using the character of $\dot{S} + \dot{E} + \dot{I}$. Cause $\dot{S} + \dot{E} + \dot{I} = A - (d - q)(S + E + I)$ is a linear function [12]. In the same way, if $B = 0$, then $\dot{B}(t) = 0$. Thus, the inequality $B(t) \geq 0$ for $t > 0$ is true. If $B(0) \in [0, m(E + I)/(w + eh))$, then $B(t) \in [0, m(E + I)/(w + eh))$ for any $t > 0$. To sum up, we get the following theorem.

Theorem 2 (feasible region). *The set*

$$\Omega = \left\{ (S, E, I, B) : 0 \leq S + E + I \leq \frac{A}{d - q}, 0 \leq B < \frac{mA}{(w + eh)(d - q)}, S \geq 0, E \geq 0, I \geq 0 \right\} \tag{5}$$

defines a forward invariant region of system (2).

2.3. Equilibria of System (4)

2.3.1. Equilibria When Bacteria Are Less Than or Equal to MID. When $0 \leq B \leq M$, then $\alpha(B) = 0$ and the model is

$$\begin{aligned}
 \dot{S} &= A + q(S + E + I) - \beta SI - dS, \\
 \dot{E} &= \beta SI - dE - \delta E, \\
 \dot{I} &= \delta E - dI - cI, \\
 \dot{B} &= m(E + I) - (w + eh)B.
 \end{aligned} \tag{6}$$

We solve the equation and can get $E_0 = (S_0, 0, 0, 0)$, a disease-free and bacteria-free equilibrium where $S_0 = A/(d - q)$, and a endemic equilibrium $E_1 = (S^1, E^1, I^1, B^1)$, where

$$\begin{aligned}
 S^1 &= \frac{(d + \delta)(d + c)}{\beta\delta}, \\
 E^1 &= \frac{(d + c)[\beta\delta A - (d - q)(d + \delta)(d + c)]}{\beta\delta[(d + \delta)(d + c) - q(d + c + \delta)]}, \\
 I^1 &= \frac{\beta\delta A - (d - q)(d + \delta)(d + c)}{\beta[(d + \delta)(d + c) - q(d + c + \delta)]}, \\
 B^1 &= \frac{m(d + c + \delta)[\beta\delta A - (d - q)(d + \delta)(d + c)]}{(w + eh)\beta\delta[(d + \delta)(d + c) - q(d + c + \delta)]}.
 \end{aligned} \tag{7}$$

Remark 3. $S^1 > 0$, $E^1 > 0$, $I^1 > 0$ and $B^1 > 0$.

According to the spectral radius theory [25, 26], we can directly obtain the basic reproduction number based on [22] when $\beta_2 = 0$. That is

$$R_0 = \rho(FV^{-1}) = \frac{\beta\delta A}{(d-q)(d+c)(d+\delta)}. \quad (8)$$

Then we can attain the existence of equilibria: if $R_0 \leq 1$, then there is only one equilibrium E_0 ; if $R_0 > 1$, then there are disease-free and bacteria-free equilibrium E_0 and endemic equilibrium E_1 .

2.3.2. Equilibria When Bacteria Are More Than MID. When $B > M$, the equation is

$$\begin{aligned} \dot{S} &= A + q(S + E + I) - \beta SI - dS - a(B - M)S, \\ \dot{E} &= \beta SI + a(B - M)S - dE - \delta E, \\ \dot{I} &= \delta E - dI - cI, \\ \dot{B} &= m(E + I) - (w + eh)B. \end{aligned} \quad (9)$$

Obviously, there is no disease-free and bacteria-free equilibria because of $M \geq 0$. Thus, system (9) implies that

$$\begin{aligned} E &= \frac{(d+c)(w+eh)B}{m(d+c+\delta)}, \\ I &= \frac{\delta(w+eh)B}{m(d+c+\delta)}, \\ S &= \frac{(d+\delta)E}{\beta I + \alpha(B)} = \frac{q(E+I) + A}{\beta I + \alpha(B) + d - q}. \end{aligned} \quad (10)$$

The equation of S about B can be written as $F(B) = 0$. Define $F(B) = B^2(w+eh)[(d+c)(d+\delta) - q(d+c+\delta)][\beta\delta(w+eh) + am(d+c+\delta)] + Bm(d+c+\delta)\{-aM(w+eh)[(d+c)(d+\delta) - q(d+c+\delta)] - (w+eh)[\beta\delta A - (d-q)(d+\delta)(d+c)] - am(d+c+\delta)A\} + aMAm^2(d+c+\delta)^2$. From the expression, we can know that $F(B)$ is a quadratic function, in which the first

and third coefficients are positive. Therefore, the existence of solution for $F(B) = 0$ depends on the second coefficient and Δ . If the second coefficient is nonnegative, no matter Δ is, then $F(B) = 0$ has no positive solution. If the second coefficient is negative, the positive solution depends on the sign of Δ : when $\Delta < 0$, there is no solution; when $\Delta = 0$, there is one positive solution; and when $\Delta > 0$, there are two positive solutions.

Now we simplify them and can get the new second coefficient b_1 :

$$b_1 := m(d+c+\delta) \cdot [(-\beta\delta(w+eh) - ma(d+c+\delta))A + b_{11}], \quad (11)$$

where

$$b_{11} := -aM(w+eh)[(d+c)(d+\delta) - q(d+c+\delta)] + (w+eh)(d-q)(d+\delta)(d+c), \quad (12)$$

and the new Δ about A :

$$\begin{aligned} \Delta(A) &:= A^2m^2(d+c+\delta)^2[-\beta\delta(w+eh) - am(d+c+\delta)]^2 + A\{-2m^2(d+c+\delta)^2(w+eh) \\ &\quad \cdot [\beta\delta(w+eh) + am(d+c+\delta)] \\ &\quad \cdot [aM[(d+c)(d+\delta) - q(d+c+\delta)] \\ &\quad + (d-q)(d+\delta)(d+c)]\} + m^2(d+c+\delta)^2 \\ &\quad \cdot \{-aM(w+eh)[(d+c)(d+\delta) - q(d+c+\delta)] \\ &\quad + (w+eh)(d-q)(d+\delta)(d+c)\}^2. \end{aligned} \quad (13)$$

We can know b_1 is a liner function about A and $\Delta(A)$ is still a quadratic function which the first and third coefficients are positive and the second coefficient is less than zero. Then we calculate the $\Delta(A)$ and can know that the sign of $\Delta(A)$ is always positive. So we can solve $\Delta(A) = 0$ and get A_1, A_2 , where

$$A_1 = (w+eh)$$

$$A_2 = \frac{aM[(d+c)(d+\delta) - q(d+c+\delta)] + (d-q)(d+c)(d+\delta) - 2\sqrt{aM[(d+c)(d+\delta) - q(d+c+\delta)](d-q)(d+c)(d+\delta)}}{\beta\delta(w+eh) + ma(d+c+\delta)}, \quad (14)$$

$$A_2 = (w+eh)$$

$$A_2 = \frac{aM[(d+c)(d+\delta) - q(d+c+\delta)] + (d-q)(d+c)(d+\delta) + 2\sqrt{aM[(d+c)(d+\delta) - q(d+c+\delta)](d-q)(d+c)(d+\delta)}}{\beta\delta(w+eh) + ma(d+c+\delta)}.$$

By the analysis with the feature of function, we can get the conclusion as follows.

Proposition 4. *The existence of positive solutions for $F(B) = 0$:*

(i) When $b_{11} \geq 0$: if $0 \leq A < A_2$, then $F(B) = 0$ does not get any positive solution; if $A = A_2$, then $F(B) = 0$ has one positive solution B_1^+ ; if $A > A_2$, then $F(B) = 0$ has two positive solutions B_1^+, B_2^+ ($B_1^+ < B_2^+$).

TABLE 3

$F(B) = 0$ has one positive solution B^+	$F(B) = 0$ has two positive solutions B_1^+, B_2^+
$M \geq 0, A = A_2$	$M \geq 0, A > A_2$
$M > \widetilde{M}, A = A_1$	$M > \widetilde{M}, 0 \leq A < A_1$

(ii) When $b_{11} < 0$: if $A_1 < A < A_2$, then $F(B) = 0$ does not get any positive solution; if $A = A_1$ or $A = A_2$, then $F(B) = 0$ has one positive solution $B^+ (= B_1^+ = B_2^+)$; if $0 \leq A < A_1$ or $A > A_2$, then $F(B) = 0$ has two positive solutions B_1^+, B_2^+ .

Note the $\widetilde{M} = (d-q)(d+c)(d+\delta)/a((d+c)(d+\delta)-q(d+c+\delta))$ (by the equation of $b_{11} = 0$) and we can put Proposition 4 to the chart as shown in Table 3.

It just discusses the existence of positive solutions, but we need consider the value of M , where only internal equilibria exist while positive solutions are more than M . Hence, we will get conclusions as follows.

Theorem 5. The existence of internal equilibria about $B > M$:

(1) If $M > 0$:

- (i) when $A > A_f$, there is an internal equilibrium E_2^+ ;
- (ii) when $A = A_f > A_g$, there is an internal equilibrium E_2^+ ;
- (iii) when $\max\{A_g, A_2\} < A < A_f$, there are two internal equilibria E_1^+, E_2^+ ;
- (iv) when $A = A_2 > A_g$, there is an internal equilibrium E^+ ;

(2) If $M = 0$ and $A > A_2 = A_g$, then there is an internal equilibrium E_2^+ .

where

$$A_f = \frac{\beta\delta(w+eh)((d+c)(d+\delta)-q(d+c+\delta))M+m(d+c+\delta)(d-q)(d+c)(d+\delta)}{\beta\delta m(d+c+\delta)},$$

$$A_g = \frac{2\beta\delta(w+eh)^2+am(w+eh)(d+c+\delta)((d+c)(d+\delta)-q(d+c+\delta))M+(w+eh)m(d+c+\delta)(d-q)(d+c)(d+\delta)}{m(d+c+\delta)[\beta\delta(w+eh)+ma(d+c+\delta)]}. \quad (15)$$

Proof. We have known the positive value of B ; now we need combine with the inequality $B > M$. The proof uses character of function $F(B)$.

Firstly, we should know the value of $F(M)$. When $B = M$, we can get $F(M) = \beta\delta(w+eh)^2((d+c)(d+\delta)-q(d+c+\delta))M^2-m(d+c+\delta)(w+eh)(\beta\delta A-(d-q)(d+c)(d+\delta))M$, and the relationship between $F(M)$ and 0 is translated into the relationship between A and A_f :

- (1) When $M > 0$: if $A > A_f$, then $F(M) < 0$, if $A = A_f$, then $F(M) = 0$, and if $A < A_f$, then $F(M) > 0$.
- (2) When $M = 0$, we can get $F(M) = 0$.

Secondly, we need to know position of M and symmetry axis of $F(M)$. We put them into the formula $g(M) := [2\beta\delta(w+eh)^2+am(w+eh)(d+c+\delta)((d+c)(d+\delta)-q(d+c+\delta))M-m(d+c+\delta)[(w+eh)(\beta\delta A-(d-q)(d+c)(d+\delta))+ma(d+c+\delta)A]$. In the same way, we translate them into the relationship between A and A_g . It means that when $M \geq 0$: if $A > A_g$, then $g(M) < 0$ (M is on the left of axis of symmetry); if $A = A_g$, then $g(M) = 0$ (M is the axis of symmetry); and if $A < A_g$, then $g(M) > 0$ (M is on the right of axis of symmetry).

Thirdly, we calculate the relationship of A_f, A_g, A_1, A_2 . Then we can get the relationships among them as follows:

- (1) The relationships of $A_f > A_1$ and $A_g > A_1$ are always true.
- (2) When $0 < M \leq M_g$, there is $A_g \leq A_2 \leq A_f$. When $M > M_g$, there is $A_2 \leq A_f < A_g$. When $M = 0$, there is $A_g = A_2 \leq A_f$.

Finally, we use the above-mentioned relational expressions and combine with Proposition 4; then, we can obtain the final chart as shown in Table 4.

Because positive solutions of $F(B)$ is equal to existences of internal equilibria of system. Thus, the existence of internal equilibria about $B > M$ is proved. $\square$

Remark 6. $M_g := m^2(d+c+\delta)^2a(d-q)(d+c)(d+\delta)/\beta^2\delta^2(w+eh)^2((d+c)(d+\delta)-q(d+c+\delta))$ is the criterion when $A_2 = A_g$.

After knowing internal equilibria when $B > M$, we want to get the relationship between internal equilibria and R_0 , so we need to change the form above all. We had known the $R_0 = \beta\delta A/(d-q)(d+c)(d+\delta)$. Thus, we change A_2 to $P_2 := \beta\delta A_2/(d-q)(d+c)(d+\delta)$, A_g to $P_g := \beta\delta A_g/(d-q)(d+c)(d+\delta)$, and A_f to $P_f := \beta\delta A_f/(d-q)(d+c)(d+\delta)$. From the expression of these, we get $R_0 = 1 < P_f$ when $M > 0$ and $R_0 = 1 = P_f$ when $M = 0$.

TABLE 4

$F(B)$	$F(M)$	$g(M)$	Conclusions	Solutions
$M \geq 0, A = A_2$	–	$M \geq 0, A > A_g$	$M > 0, A = A_2 > A_g$	B^+
	$M > 0, A > A_f$	–	$M > 0, A > A_f$	B_2^+
$M \geq 0, A > A_2$	$M = 0$	$M \geq 0, A > A_g$	$M = 0, A > A_2 = A_g$	B_2^+
	$M > 0, A = A_f$	$M \geq 0, A > A_g$	$M > 0, A = A_f > A_g$	B_2^+
	$M > 0, A < A_f$	$M \geq 0, A > A_g$	$M > 0, \max\{A_2, A_g\} < A < A_f$	B_1^+, B_2^+

FIGURE 2: Density of the infected dairy cows as a function of the basic reproduction number with $0 < M < M_g$. The common values of (a), (b), and (c) used for the parameters are as follows: $w = 3, d = 2, c = 0.8, \delta = 4, q = 1, \beta = 0.2$, and $a = 2$. The different values are (a) $m = 0.1, M = 0.1$; (b) $m = 0.0932, M = 0.0468$; and (c) $m = 0.05, M = 0.0135$. The forward bifurcation occurs at $R_0 = 1$ in all the pictures and the backward bifurcation occurs at $R_0 = 1.2101$ in picture (a), at $R_0 = 1.1056$ in picture (b) and at $R_0 = 1.0567$ in picture (c).

Theorem 7 (existence of equilibria in Figures 2 and 3). *The equilibrium $E_0 = (S_0, 0, 0, 0)$ always exists in Ω . Here are the existences of internal equilibria:*

(1) If $0 < M < M_g$:

- (i) If $P_2 > 1$: when $1 < R_0 < P_2$, there only exists E_1 ; when $R_0 = P_2$, there exist E_1, E_1^+ ; when $P_2 < R_0 < P_f$, there exist E_1, E_1^+, E_2^+ ; when $R_0 = P_f$, there exist E_1, E_2^+ ; when $R_0 > P_f$, there exists E_2^+ .
- (ii) If $P_2 = 1$: when $R_0 = P_2$, there exists E_1^+ ; when $1 < R_0 < P_f$, there exist E_1, E_1^+, E_2^+ ; when $R_0 = P_f$, there exist E_1, E_2^+ ; when $R_0 > P_f$, there exists E_2^+ .
- (iii) If $P_2 < 1$: when $R_0 = P_2$, there exists E_1^+ ; when $P_2 < R_0 \leq 1$, there exist E_1^+, E_2^+ ; when $1 < R_0 < P_f$, there exist E_1, E_1^+, E_2^+ ; when $R_0 = P_f$, there exist E_1, E_2^+ ; when $R_0 > P_f$, there exists E_2^+ .

- (2) If $M \geq M_g$: when $1 < R_0 \leq P_f$, there exists E_1 ; when $R_0 > P_f$, there exists E_2^+ .
- (3) If $M = 0$ (in this case, $P_f = 1$): when $R_0 > P_2$, there exists E_2^+ .

Theorem 7 describes the existence of equilibria under the different parameters condition (Table 5), where disease-free and bacteria-free equilibrium E_0 always exists. Besides, we give the stability of the equilibria, which will be proved in next part. We just give the stability of E_0 and E_1 ; others are

too difficult to give the strict mathematical proofs because of lacking exact expression. Therefore, the local stability of E_1^+ and E_2^+ will be demonstrated numerically in Section 4.

3. Stability of Equilibria

3.1. Local Stability of E_0 and E_1 . We calculate the Jacobian to analyze the local stability of each of the equilibria. When $0 \leq B \leq M$, the Jacobian is

$$J(S, E, I, B) = \begin{pmatrix} q - d - \beta I & q & q - \beta S & 0 \\ \beta I & -(d + \delta) & \beta S & 0 \\ 0 & \delta & -(d + c) & 0 \\ 0 & m & m & -(w + eh) \end{pmatrix}. \quad (16)$$

Now considering E_0 , we use λ for eigenvalues. We can compute

$$\det(\lambda I - J_{E_0}) = [\lambda - (q - d)](\lambda + w + eh) \cdot [(\lambda + d + \delta)(\lambda + d + c) - \beta \delta S_0]. \quad (17)$$

Note $h(\lambda) = (\lambda + d + \delta)(\lambda + d + c) - \beta \delta S_0$. We can find that $\lambda_1 = -(d - q) < 0$, $\lambda_2 = -(w + eh) < 0$. Now we just consider the eigenvalues of $h(\lambda)$. The Routh – Hurwitz coefficients of the $h(\lambda)$ are

$$\begin{aligned} \Delta_1 &= d + c + d + \delta > 0, \\ \Delta_2 &= (d + c + d + \delta)[(d + \delta)(d + c) - \beta \delta S_0]. \end{aligned} \quad (18)$$

TABLE 5: Existence of endemic equilibria of model (4).

Condition 1	Condition 2	Condition 3	Endemic equilibria
$0 < M < M_g$	$P_2 > 1$	$1 < R_0 < P_2$	E_1
		$R_0 = P_2$	E_1, E^+
		$P_2 < R_0 < P_f$	E_1, E_1^+, E_2^+
		$R_0 = P_f$	E_1, E_2^+
	$P_2 = 1$	$R_0 > P_f$	E_2^+
		$R_0 = P_2$	E^+
		$1 < R_0 < P_f$	E_1, E_1^+, E_2^+
		$R_0 = P_f$	E_1, E_2^+
	$P_2 < 1$	$R_0 > P_f$	E_2^+
		$R_0 = P_2$	E^+
$M \geq M_g$	—	$P_2 < R_0 \leq 1$	E_1^+, E_2^+
		$1 < R_0 < P_f$	E_1, E_1^+, E_2^+
		$R_0 = P_f$	E_1, E_2^+
		$R_0 > P_f$	E_2^+
$M = 0$	—	$1 < R_0 \leq P_f$	E_1
		$R_0 > P_f$	E_2^+
		$R_0 > P_2$	E_2^+

FIGURE 3: The relationship between the basic reproduction number and infected dairy cows while $M \geq M_g$ and $M = 0$, where (2) and (3) of Theorem 9 holds. The common values of (a) and (b) used for the parameters are as follows: $w = 3, d = 2, c = 0.8, \delta = 4, q = 1, \beta = 0.2, a = 2$, and $m = 0.1$. The different values of (a) and (b) are (a) $M = 0.5$, (b) $M = 0$. There is a forward bifurcation at $R_0 = 1$ in (a). The bifurcation at $R_0 = 0.6383$ in (b) is still forward.

If $R_0 < 1$, then $\Delta_2 > 0$; if $R_0 > 1$, then $\Delta_2 < 0$. Thus the eigenvalues of $h(\lambda)$ are negative when $R_0 < 1$. So the disease-free and bacteria-free equilibria E_0 are locally asymptotically stable while $R_0 < 1$. If $R_0 > 1$, then E_0 is a saddle-point equilibrium.

Now considering E_1 . The characteristic polynomial of Jacobian of E_1 is

$$\det(\lambda I - J_{E_1}) = a_0 \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0, \quad (19)$$

where

$$\begin{aligned} a_0 &= 1, \\ a_1 &= \frac{\beta \delta A - q(d - q)(d + c + \delta)}{(d + \delta)(d + c) - q(d + c + \delta)} \\ &\quad + (d + c + d + \delta), \\ a_2 &= \frac{\beta \delta A - [q(d - q)(d + c + \delta)](d + \delta + d + c)}{(d + \delta)(d + c) - q(d + c + \delta)}, \\ a_3 &= \beta \delta A - (d - q)(d + \delta)(d + c). \end{aligned} \quad (20)$$

The *Routh – Hurwitz* coefficients of the $\det(\lambda I - J_{E_0})$ are

$$\begin{aligned}\Delta_1 &= a_1, \\ \Delta_2 &= a_1 a_2 - a_3, \\ \Delta_3 &= a_3 (a_1 a_2 - a_3).\end{aligned}\tag{21}$$

where $a_1 > (\beta\delta A - q(d-q)(d+c+\delta))/((d+\delta)(d+c) - q(d+c+\delta)) > (\beta\delta A - (d-q)(d+\delta)(d+c))/((d+\delta)(d+c) - q(d+c+\delta)) > 0$ and $a_3 > 0$, when $R_0 > 1$.

As for the condition $a_1 a_2 - a_3$, we have the following expression:

$$a_1 a_2 - a_3 = h_1 + h_2 + h_3, \tag{22}$$

where

$$\begin{aligned}h_1 &= \frac{[\beta\delta A - q(d-q)(d+c+\delta)] \{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta) - [q\beta\delta A - q(d-q)(d+c)(d+\delta)]\}}{[(d+c)(d+\delta) - q(d+c+\delta)]^2}, \\ h_2 &= \frac{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta)^2 - (d+c+d+\delta)[q\beta\delta A - q(d-q)(d+c)(d+\delta)]}{(d+c)(d+\delta) - q(d+c+\delta)}, \\ h_3 &= -[\beta\delta A - (d-q)(d+\delta)(d+c)].\end{aligned}\tag{23}$$

Now, we magnify them:

$$\begin{aligned}h_1 &> \frac{[(d-q)(d+c)(d+\delta) - q(d-q)(d+c+\delta)] \{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta) - [q\beta\delta A - q(d-q)(d+c)(d+\delta)]\}}{[(d+\delta)(d+c) - q(d+c+\delta)]^2} \\ &= \frac{(d-q) \{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta) - q[\beta\delta A - (d-q)(d+c)(d+\delta)]\}}{(d+\delta)(d+c) - q(d+c+\delta)}, \\ h_1 + h_2 &> \frac{\{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta)(d+\delta+d+c+d-q) - [q\beta\delta A - q(d-q)(d+c)(d+\delta)](d+\delta+d+c+d-q)\}}{(d+\delta)(d+c) - q(d+c+\delta)}, \\ h_1 + h_2 + h_3 &> \frac{[\beta\delta A - q(d-q)(d+c+\delta)](d+c+d+\delta)(d+\delta+d+c+d-q) - [\beta\delta A - (d-q)(d+c)(d+\delta)][2dq - q^2 + (d+c)(d+\delta)]}{(d+\delta)(d+c) - q(d+c+\delta)} \\ &> \frac{[\beta\delta A - (d-q)(d+c)(d+\delta)](d+c+d+\delta)(d+c+d+\delta+d-q) - [\beta\delta A - (d-q)(d+c)(d+\delta)][2dq - q^2 + (d+c)(d+\delta)]}{(d+\delta)(d+c) - q(d+c+\delta)} \\ &> \frac{[\beta\delta A - (d-q)(d+c)(d+\delta)] [(d+c)^2 + (d+\delta+d-q)(d+\delta+d+c) + q^2 - 2d^2]}{(d+\delta)(d+c) - q(d+c+\delta)}.\end{aligned}\tag{24}$$

We know that $d > q$, so $(d+c)(d+\delta) > q(d+c+\delta)$ and then $a_1 a_2 - a_3 > 0$. Hence, the *Routh–Hurwitz* conditions are satisfied when $R_0 > 1$ and E_1 is locally asymptotically stable.

Theorem 8 (local stability). *System (4) has equilibria as follows:*

When $0 \leq B \leq M$ and $R_0 < 1$, there is only one equilibrium E_0 which is locally asymptotically stable. When $0 \leq B \leq M$ and $R_0 > 1$, E_0 is unstable and a unique endemic equilibrium E_1 exists which is locally asymptotically stable.

We study the stability of equilibria in order to explain more epidemiological implications. Taking Figure 2(a) as an example, we can get the following biological significance. If E_0 is stable ($R_0 < 1$), the brucellosis will fade as time goes by. If E_1 is stable ($1 < R_0 < P_2$), disease will maintain the level of I_1 all the time. With the scope of R_0 ($P_2 < R_0 < P_f$), disease will continue at low-level state (I_1) when the current status is in the attraction domain of E_1 and will be going to stay at high-level state (I_2^+) while the current status exactly situate in the attraction domain of E_2^+ , which will be demonstrated

numerically in Section 4. Similarly, disease will be going to stay at the level of I_2^+ only if $R_0 > P_f$ is true.

3.2. Global Stability of E_0 and E_1 . In this section, we will demonstrate the global stability of the endemic equilibrium of system (4) by using the *Lyapunov function* [27, 28].

Theorem 9. *If $0 \leq B \leq M$ and $R_0 < 1$, then E_0 is globally asymptotically stable in Ω .*

Proof. As we can see, B is not acting on the other subclasses. So we can define the *Lyapunov function*

$$V(S, E, I, B) = S - S_0 - S_0 \ln \frac{S}{S_0} + E + \frac{d + \delta}{\delta} I. \quad (25)$$

Then the derivative of V along system (6) is

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{S_0}{S}\right) [A - (d - q)S + q(E + I) - \beta SI] \\ &\quad + [\beta SI - (d + \delta)E] + \frac{d + \delta}{\delta} [\delta E - (d + c)I] \\ &= \left(1 - \frac{S_0}{S}\right) [A - (d - q)S + q(E + I)] + \beta S_0 I \\ &\quad - (d + \delta)E + \frac{d + \delta}{\delta} [\delta E - (d + c)I] \\ &= \left(1 - \frac{S_0}{S}\right) [A - (d - q)S + q(E + I)] \\ &\quad + I \frac{\beta \delta A - (d - q)(d + c)(d + \delta)}{\delta(d - q)}. \end{aligned} \quad (26)$$

According to the definition of the invariant region, we can know that $S \leq S_0$. So $dV/dt < 0$ is true and $dV/dt = 0$ holds only if $S = S_0$, when $R_0 < 1$. $dV/dt = 0$ holds only if $S = S_0$ when $R_0 = 1$. Thus the disease-free and bacteria-free equilibrium E_0 is globally asymptotically stable by *LaSalle's Invariance Principle* [29]. $\square$

Theorem 10. *If $0 \leq B \leq M$, $R_0 > 1$, and $q = 0$, then E_1 is a conditionally globally asymptotically stable in Ω .*

Proof. In this proof, we use the same way to demonstrate the stability when $q = 0$. Similarly, B is not acting on the other subclasses, so we define the *Lyapunov function* as follows:

$$\begin{aligned} V(S, E, I, B) &= S - S^1 - S^1 \ln \frac{S}{S^1} + E - E^1 - E^1 \ln \frac{E}{E^1} \\ &\quad + \frac{d + \delta}{\delta} \left(I - I^1 - I^1 \ln \frac{I}{I^1} \right). \end{aligned} \quad (27)$$

Then the derivative of V along system (6) is

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{S^1}{S}\right) [A - dS - \beta SI] \\ &\quad + \left(1 - \frac{E^1}{E}\right) [\beta SI - (d + \delta)E] \\ &\quad + \frac{d + \delta}{\delta} \left(1 - \frac{I^1}{I}\right) [\delta E - (d + c)I]. \end{aligned} \quad (28)$$

Let $s = S/S^1$, $e = E/E^1$, and $i = I/I^1$. Then we can get

$$\begin{aligned} A - dS^1 - \beta S^1 I^1 &= 0, \\ \beta S^1 I^1 - (d + \delta)E^1 &= 0, \\ \delta E^1 - (d + c)I^1 &= 0. \end{aligned} \quad (29)$$

Combining with them, we can obtain that

$$\begin{aligned} \frac{dV}{dt} &= S^1 \left(2d - ds - \frac{d}{s}\right) + \beta S^1 I^1 \left(1 - \frac{1}{s} + i - \frac{si}{e}\right) \\ &\quad + E^1 \left[(d + \delta) - \frac{(d + \delta)e}{i}\right] \\ &\quad + I^1 \left[-\frac{(d + \delta)(d + c)i}{\delta} + \frac{(d + c)(d + \delta)}{\delta}\right] \\ &= dS^1 \left(2 - s - \frac{1}{s}\right) + \beta S^1 I^1 \left(3 - \frac{1}{s} - \frac{si}{e} - \frac{e}{i}\right). \end{aligned} \quad (30)$$

Combining with inequality of arithmetic and geometric means, we can know $2 - s - 1/s \leq 0$ and $(3 - 1/s - si/e - e/i) \leq 0$. Thus,

$$\frac{dV}{dt} \leq 0. \quad (31)$$

According to the definition of the invariant region, only if $s = e = i = 1$ means that $S = S^1$, $E = E^1$ and $I = I^1$, then $dV/dt = 0$. Similarly with Theorem 9, we can get the endemic equilibrium E_1 which is globally asymptotically stable by *LaSalle's Invariance Principle* in Ω when $q = 0$. $\square$

4. Numerical Results

In an epidemic model, the basic reproduction number R_0 is calculated and verified to be a threshold for the dynamics of the disease. The ultimate objective is to control the disease by making the basic reproduction number R_0 . In the previous analysis of this paper, we analyze the different equilibria in variant ranges of R_0 . Now, we should know the final infectious size of dairy cows depending on the R_0 .

In Figure 4(a), only if $R_0 < \min\{1, P_2\}$ (Figures 2(a), 2(b), 2(c), 3(a), and 3(b)), we can know that the number of infected dairy cows will obtain the same disease-free and bacteria-free equilibrium E_0 no matter how much the initial values are and the relationship about M , M_g , and 0 is. In biology, this means only if the basic reproduction number demands the formula,

FIGURE 4: Time series of the infected dairy cows. The common values of (a) and (b) used for the parameters are as follows: $w = 3$, $d = 2$, $c = 0.8$, $\delta = 4$, $q = 1$, $\beta = 0.2$, $a = 2$, $m = 0.2$. The different values of (a) and (b) are (a) $A = 10$ and (b) $A = 50$. The solid line or S1 means that I is entering system (6); the dotted line or S2 indicates that I is going into system (9).

FIGURE 5: The common values of (a) and (b) used for the parameters are as follows: $w = 3$, $d = 2$, $c = 0.8$, $\delta = 4$, $q = 1$, $\beta = 0.2$, $a = 2$, $m = 0.1$, $M = 0.1$, and $A = 25$. Different colors are representative of different initial values, of which the dot line means that it enters system (6) and the full line means that it goes into system (9) as in Figure 4.

the final infectious size of dairy cows is tending to zero. It is to say, the disease will vanish without any measures after a certain time.

Figure 4(b) demonstrates that when $R_0 > \max\{P_2, P_f\}$, the infected dairy cows will obtain the endemic equilibrium E_2^+ . In the meantime, different values of M have different equilibria: when $M = 0.5$, $I_2^+ = 15.3531$; when $M = 0.1$, $I_2^+ = 15.9432$; and when $M = 0$, $I_2^+ = 16.0625$. This means that only if the basic reproduction number satisfies

the above formula, brucellosis will never disappear and it would become epidemic disease. In addition, the more size of MID will lead to the less final infected dairy cows. It might be easy to understand that when the MID is heightened, the bacteria invading the body are diminished by immunology theory [10].

Figures 5 and 7 show that when $\min\{1, P_2\} < R_0 < P_f$, the final size of infectious cows will be in bistable state. Figure 5 comes from Figure 2(a) when $P_2 < R_0 < P_f$ and Figure 7

FIGURE 6: The tendency of S , E , and I .

(a) The tendency of bacteria

(b) The tendency of infected dairy cows

FIGURE 7: The common values of (a) and (b) used for the parameters are as follows: $w = 3$, $d = 2$, $c = 0.8$, $\delta = 4$, $q = 1$, $\beta = 0.2$, $a = 2$, $m = 0.05$, $M = 0.014$, and $A = 22$. FIGURE 8: The tendency of S , E , and I .

comes from Figure 2(c) when $1 < R_0 < P_f$, in which Figures 5(a) and 7(a) are time series plots of the number of Brucella and Figures 5(b) and 7(b) are time series plots of the number of infected dairy cows. Figures 6 and 8 show tendency of S , E , I . From them we can see that when R_0 meets the relational expression, different initial values will eventually lead to two equilibria; one is E_1 and the other is E_2^+ ($E_1 < E_2^+$). In

biology, brucellosis will become endemic in dairy cows with two different stable states.

5. Conclusion and Discussion

Brucellosis is becoming more and more popular in recent years, since it has formed serious endemic disease and it cannot be thoroughly removed in northeast and northwest China. In this paper, we take account of immune influence of Brucella just like researching of cholera [12]. We build the new brucellosis model based on [22]. Compared with other studies, we are more concerned with the effects of immunology on Brucella. We just change the transmission rate from constant β_2 to the piecewise continuous function between the research [12] and our paper. But the mathematical results we obtain are more complicated and more biological. The original work obtained the basic reproduction number and got two equilibria and then simply analyzed the stability state. In this paper, we take immunological threshold into consideration; then we get the whole mathematical analysis of equilibria in each case and do the locally and globally stable analysis about some equilibria. The MID of Brucella has not been tested by experiment, so we get the serious discussion

with $M > 0$ and $M = 0$: when $0 < M < M_g$ (the formation of M_g is given in this paper), we get the forward bifurcation occurring at $R_0 = 1$ and backward bifurcation appearing at $R_0 = P_2$; when $M \geq M_g$, it just has forward bifurcation at $R_0 = 1$; when $M = 0$, the model is exactly same as in [22] and produces the forward bifurcation at $R_0 = P_2$. By mathematical analysis of them, we can get the global stability E_0 when $R_0 < 1$ and a global stability E_1 when $R_0 > 1$. The character of $E_{1,2}^+$ is given by numerical simulations. From that, we know it will be in bistable state when the basic reproduction number demands $\max\{1, P_2\} < R_0 < P_f$. We make the basic reproduction number as the measuring standard in order to give suggestions of the brucellosis prevention [30, 31]. While the basic reproduction number is less than 1, brucellosis is becoming a disease-free and bacteria-free stable state. After a certain time, brucellosis will be removed. And while the basic reproduction number is more than 1 by recording to different relationship about M , M_g and 0, the brucellosis will be endemic disease which will hold a stable state with variant values of infected dairy cows. Using the analysis of this paper, we can reduce the number of infected dairy cows to prevent the spreading of epidemic disease, which can even obtain zero by adjusting the size of the basic reproduction number.

Refs. [20, 21] using *Holling – II* functional response both show that MID is vital for the spread of cholera. Reducing the pathogen density to under the MID in an aquatic reservoir would be more effective measure to control cholera. Similarly, in order to control and prevent the spreading of brucellosis, we can place an emphasis on the incident rate because of the existence of MID. If we can adjust the initial value of *Brucella* to be below the MID and constrain growth and reproduction conditions of *Brucella* in environment, then we can obtain the incidence rate of indirect transmission of *Brucella* to susceptible cow to be zero all the time and the ultimate scale of brucellosis will decrease and even disappear.

In further planning, the incidence term $\alpha(B)$ can be used in the formation by a *Holling – II* response which is more reasonable in biology than the linear form in this paper. In addition, we have not yet captured and processed the real data about brucellosis of dairy cows to get more effective conclusions. That is what we are going to do next. Furthermore, the real spreading of brucellosis is via multiple zones rather than one zone and the multizone patchy model can describe the transmission in multiple regions. Hence, with considering the network models [32, 33] or reaction-diffusion equations [34–38], conclusions can accord well with practice even more.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

The project is funded by the National Natural Science Foundation of China under Grants (11671241, 11601292, 41875097,

and 11501338), Program for the Outstanding Innovative Teams (OIT) of Higher Learning Institutions of Shanxi, Natural Science Foundation of Shanxi Province Grant no. 201801D221003, and High-Level Talent Project of Jiangsu Province (Six Talent Peaks and Grant no. JNHB-071).

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