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TRANSMISSION DYNAMICS AND CONTROL FOR A BRUCELLOSIS MODEL IN HINGGAN LEAGUE OF INNER MONGOLIA, CHINA

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Abstract. Brucellosis is one of the major infectious and contagious bacterial diseases in Hinggan League of Inner Mongolia, China. The number of newly infected human brucellosis data in this area has increased dramatically in the last

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10 years. In this study, in order to explore effective control and prevention measures we propose a deterministic model to investigate the transmission dynamics of brucellosis in Hinggan League. The model describes the spread of brucellosis among sheep and from sheep to humans. The model simulations agree with newly infected human brucellosis data from 2001 to 2011, and the trend of newly infected human brucellosis cases is given. We estimate that the control reproduction number $\mathcal{R}_c$ is about 1.9789 for the brucellosis transmission in Hinggan League and compare the effect of existing mixed cross infection between basic ewes and other sheep or not for newly infected human brucellosis cases. Our study demonstrates that combination of prohibiting mixed feeding between basic ewes and other sheep, vaccination, detection and elimination are useful strategies in controlling human brucellosis in Hinggan League.

1. **Introduction.** Brucellosis is one of the worlds major infectious and contagious bacterial disease of animals and humans caused by members of the genus Brucella. It will cause a variety of organ damage mainly to the reproductive system, and can lead to abortion and sterility. The disease primarily affects cattle and sheep, which sometimes infects horses, dogs, swine, and humans. In humans, Brucella abortus causes undulant fever, a disease characterized by intermittent fever, headaches, fatigue, joint and bone pain, psychotic disturbances, and other symptoms [23]. Brucellosis is prevalent for more than a century in many parts of the world, and it is well controlled in most developed countries. However, more than 500,000 new cases are reported each year around the world and the disease remains endemic in the Middle East, Asia, Africa, Latin America, the Mediterranean Basin and the Caribbean [4, 21].

Brucellosis results in infection by various species of Brucella, and transmits to other animals or human through direct contacts with infected animals or indirect transmission by brucella in the environment [13]. Sheep and goats and their products remain the main source of human infection [7]. In sheep, brucellosis is primarily a disease of the female. The infected sheep exhibit symptoms which may include abortion during the last third of pregnancy, retained afterbirth, and weak calves at birth. Infected sheep usually abort only once, subsequent calves may be born weak and some infected sheep will not exhibit any clinical symptoms of the disease. The survival time of brucella ranges from one to four months in the contaminated soil and water, and two months in milk and meat [13]. However, the brucella bacteria are easily killed by direct sunlight, high temperature and effective disinfectant [23].

In the early 20th century, reports confirm human brucellosis patients in China. Two human brucellosis cases were first reported in Chongqing province in 1905 and a human brucellosis case was reported in Fujian province in 1916 [36]. In the mid 1950s, the detection and prevention for humans and livestock were carried out in a planned way [39]. From 1996 to 2005, the human brucellosis incidence increased 15 times and 19,013 human brucellosis cases were reported in 2006 [22]. In 2009, livestock brucellosis was reported in 29 of 32 provinces, and 37 thousands new human brucellosis cases were reported, 18.6 times larger than that of human brucellosis in 2000 [31]. Most of the human brucellosis patients were infected by sheep-type brucella in China, accounting for 84.5% of the total cases [25]. Brucellosis has already been a natural focal disease in Inner Mongolia, since Inner Mongolia has the largest number of sheep in Mainland China, but sheep vaccination rate is only 31.6% [11]. In recent years, Brucellosis has already become one of the biggest public health threats in Hinggan League [20]. As early as in 1938, infected cattle were found at Hinggan League of Inner Mongolia Autonomous Region. From 2001 to 2009, 5,420 human brucellosis cases were reported in Hinggan League, in which

4,957 human brucellosis cases were infected by direct contact with infected sheep, 44 human brucellosis cases were infected by direct contact with infected cattle, and other 419 human brucellosis cases were infected by unknown cause [20]. From Figure 1, we know that human brucellosis cases increased year by year in Hinggan League. In 2010, newly infected human brucellosis cases of Hinggan League is about 20% of Inner Mongolia of China. So it is necessary to investigate the prevalence of brucellosis in Hinggan League.

FIGURE 1. Comparing human brucellosis cases in Hinggan League and Inner Mongolia of China.

In China, control measures for brucellosis within livestock include detection, vaccination and elimination of the infected animals. Combination of these measures are usually used to control the spreading of brucellosis [22]. Mathematical modeling has become an important tool in analyzing the epidemiological characteristics of infectious diseases and can provide useful control measures [10, 14, 34, 35]. Several mathematical models have been developed to study the transmission of brucellosis. Huang et al. [12] applied fuzzy mathematics and markov forecast to study a fuzzy dynamic mathematical model. Lu et al. [16] used brucellosis prevalence data from 1992 to 1998, then two models for forecasting the infection rate of brucellosis were established based on grey theories. Li et al. [15] applied the grey model and the weighted average model to forecast positive rate of brucellosis. However, all of their studies focus on the spread of brucellosis with statistical methods, and do not investigate the essential meaning of brucellosis transmission mechanism. Recently, more and more people use the dynamic model to study the spread of brucellosis. Zinsstag et al. [40] studied an susceptible, seropositive and immunized dynamic model to represent cattle-sheep-human brucellosis transmission characteristics of Mongolia. Alnseba et al. [1] proposed an susceptible, infected and the contaminated environment dynamical model for brucellosis epidemic in ovine with direct and indirect transmission. Hou et al. [11] proposed a dynamic model for the sheep-human transmission of brucellosis in Inner Mongolia, and they found that both

young and adult sheep vaccination and disinfection were appropriate strategies to control brucellosis in Inner Mongolia Autonomous Region of China.

From Niu et al. [20], we know that about 90% of human brucellosis cases are infected by direct contact with infected sheep in Hinggan League of Inner Mongolia. So in order to understand the transmission dynamics of brucellosis in Hinggan League of Inner Mongolia, we propose a deterministic $S_oE_oI_oV_oS_fE_fI_fV_fWS_hI_hY_h$ model to describe the spread of brucellosis among sheep and from sheep to humans. Comparing with Hou et al. [11], we divide flock of sheep into basic ewes and other sheep (which includes stock ram and fattening sheep). We first simulate the number of human brucellosis cases in Hinggan League of Inner Mongolia from 2001 to 2011, and then determine the control reproduction number $\mathcal{R}_0$, give some sensitivity and uncertainty analysis of the basic control reproduction number on control parameters and discuss some control strategies of brucellosis infection in Hinggan League of Inner Mongolia.

This paper is organized as follows. In Section 2, we present the dynamical model. And the mathematical analysis will be given in Section 3. Numerical simulations of human brucellosis cases in Hinggan League of Inner Mongolia from 2001 to 2011 and sensitivity analysis of the basic reproduction number $\mathcal{R}_0$ are carried out in Section 4. Section 5 gives a brief discussion about main results, shortcomings and future work.

2. **The dynamic model.**

In Hinggan League, the infected basic ewes remain the main source of human brucellosis infection, and in order to study different types of brucellosis transmission models among sheep and from sheep to humans, the flock of sheep can be divided into basic ewes and other sheep. In the real world, we know that basic ewes and other sheep are often mixed feeding together, therefore there exists mixed cross infection between other sheep and basic ewes. Hence, the transmission dynamic of brucellosis in Hinggan League of Inner Mongolia we considered is a multi-group model, which is a $S_oE_oI_oV_oS_fE_fI_fV_fWS_hI_hY_h$ structure with cross infection between sheep and human. In our model, $S_o(t), E_o(t), I_o(t), V_o(t)$ represent susceptible, recessive infected, quarantined seropositive infected, vaccinated other sheep at time t, respectively. $S_f(t), E_f(t), I_f(t), V_f(t)$ describe susceptible, recessive infected, quarantined seropositive infected, vaccinated basic ewes at time t, respectively. $W(t)$ denotes the quantity of sheep brucella in the environment at time t, we definite that the average number of bacteria that a host need to be infected brucellosis is called an infectious unit. $S_h(t), I_h(t), Y_h(t)$ represent susceptible individuals, acute infections, chronic infections at time t, respectively.

There are some assumptions for our model.

(i) Brucellosis in recessive infected period is hardly detected, so we assume sheep at recessive infected period can infect susceptible sheep and human. When recessive infected sheep are detected to be seropositive, which will enter into quarantined seropositive infected individuals compartment.

(ii) Susceptible sheep and human can be infected through ingesting brucella, which is called as the indirect transmission.

(iii) We assume sheep at recessive infected period and quarantined seropositive infected period discharge the same quantity of brucella into the environment per unite time.

There are also some other assumptions on the dynamical transmission of brucellosis in Hinggan League of Inner Mongolia among sheep and from sheep to humans,

which are demonstrated in the flowchart (see Figure 2). From Figure 2, the new infection occurred in the other sheep group, the basic ewes group and human group are given by $g(S_o) = (\beta_{oo}E_o+\beta_{of}E_f+\beta_oW)S_o$, $g(S_f) = (\beta_{ff}E_f+\beta_{fo}E_o+\beta_fW)S_f$ and $g(S_h) = (\beta_{ho}E_o + \beta_{hf}E_f + \beta_hW)S_h$, respectively.

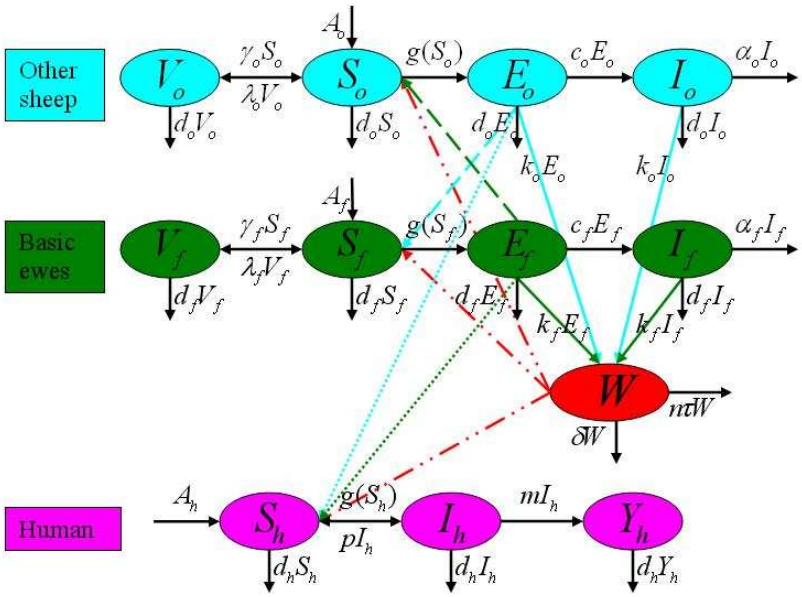

FIGURE 2. Transmission diagram on the dynamical transmission of brucellosis among sheep and from sheep to humans.

Based on previous assumptions, the following system of ordinary differential equations can be derived:

$$\begin{cases} \frac{dS_o}{dt} = A_o - (\beta_{oo}E_o + \beta_{of}E_f + \beta_oW)S_o + \lambda_oV_o - (\gamma_o + d_o)S_o, \\ \frac{dE_o}{dt} = (\beta_{oo}E_o + \beta_{of}E_f + \beta_oW)S_o - (c_o + d_o)E_o, \\ \frac{dI_o}{dt} = c_oE_o - (\alpha_o + d_o)I_o, \\ \frac{dV_o}{dt} = \gamma_oS_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} = A_f - (\beta_{ff}E_f + \beta_{fo}E_o + \beta_fW)S_f + \lambda_fV_f - (\gamma_f + d_f)S_f, \\ \frac{dE_f}{dt} = (\beta_{ff}E_f + \beta_{fo}E_o + \beta_fW)S_f - (c_f + d_f)E_f, \\ \frac{dI_f}{dt} = c_fE_f - (\alpha_f + d_f)I_f, \\ \frac{dV_f}{dt} = \gamma_fS_f - (\lambda_f + d_f)V_f, \\ \frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W, \\ \frac{dS_h}{dt} = A_h - (\beta_{ho}E_o + \beta_{hf}E_f + \beta_hW)S_h - d_hS_h + pI_h, \\ \frac{dI_h}{dt} = (\beta_{ho}E_o + \beta_{hf}E_f + \beta_hW)S_h - (m + d_h + p)I_h, \\ \frac{dY_h}{dt} = mI_h - d_hY_h. \end{cases} \tag{1}$$

The transmission of brucellosis for human and sheep population are shown in system (1). All parameters of system (1) are assumed to be nonnegative and described in Table 1.

TABLE 1. Descriptions of parameter in system (1).

Parameter	Comments
A_o	The recruitment rate of other sheep
A_f	The recruitment rate of basic ewes
A_h	The annual birth rate of human
d_o	The removed rate of Other sheep
d_f	The removed rate of Basic ewes
λ_o	The losing immunity rate of other sheep
λ_f	The losing immunity rate of basic ewes
γ_o	The efficient vaccination rate of other sheep
γ_f	The efficient vaccination rate of basic ewes
c_o	The seropositive detection rate of other sheep
c_f	The seropositive detection rate of basic ewes
α_o	The disease-related elimination rate of other sheep
α_f	The disease-related elimination rate of basic ewes
k_o	The brucella shedding rate by infected other sheep
k_f	The brucella shedding rate by infected basic ewes
δ	The decaying rate of brucella in the environment
n	Disinfection times
τ	The effective disinfection rate
d_h	The natural mortality rate of human
m	The transfer rate from acute infections to chronic infections
p	The transfer rate from acute infections to susceptible individuals
β_o	Brucella-to-other sheep transmission rate
β_f	Brucella-to-basic ewes transmission rate
β_h	Brucella-to-human transmission rate
β_{oo}	Other sheep-to-other sheep transmission rate
β_{of}	Basic ewes-to-other sheep transmission rate
β_{ff}	Basic ewes-to-basic ewes transmission rate
β_{fo}	Other sheep-to-basic ewes transmission rate
β_{ho}	Other sheep-to-human transmission rate
β_{hf}	Basic ewes-to-human transmission rate

3. **Dynamical behavior.** We know that system (1) can be divided into two independent systems

$$\begin{cases} \frac{dS_o}{dt} = A_o - (\beta_{oo}E_o + \beta_{of}E_f + \beta_o W)S_o + \lambda_o V_o - (\gamma_o + d_o)S_o, \\ \frac{dE_o}{dt} = (\beta_{oo}E_o + \beta_{of}E_f + \beta_o W)S_o - (c_o + d_o)E_o, \\ \frac{dI_o}{dt} = c_o E_o - (\alpha_o + d_o)I_o, \\ \frac{dV_o}{dt} = \gamma_o S_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} = A_f - (\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W)S_f + \lambda_f V_f - (\gamma_f + d_f)S_f, \\ \frac{dE_f}{dt} = (\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W)S_f - (c_f + d_f)E_f, \\ \frac{dI_f}{dt} = c_f E_f - (\alpha_f + d_f)I_f, \\ \frac{dV_f}{dt} = \gamma_f S_f - (\lambda_f + d_f)V_f, \\ \frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W. \end{cases} \tag{2}$$

$$\begin{cases} \frac{dS_h}{dt} = A_h - (\beta_{ho}E_o + \beta_{hf}E_f + \beta_h W)S_h - d_h S_h + pI_h, \\ \frac{dI_h}{dt} = (\beta_{ho}E_o + \beta_{hf}E_f + \beta_h W)S_h - (m + d_h + p)I_h, \\ \frac{dY_h}{dt} = mI_h - d_h Y_h. \end{cases} \tag{3}$$

So we only need to consider the dynamical behavior of system (2). There exists a disease-free equilibrium: $P_0 = (S_o^0, 0, 0, V_j^0, S_f^0, 0, 0, V_f^0, 0)$ for system (2). Where

$$S_o^0 = \frac{A_o(\lambda_o + d_o)}{d_o(d_o + \lambda_o + \gamma_o)}, V_o^0 = \frac{A_o\gamma_o}{d_o(d_o + \lambda_o + \gamma_o)}, S_f^0 = \frac{A_f(\lambda_f + d_f)}{d_f(d_f + \lambda_f + \gamma_f)},$$

$$V_f^0 = \frac{A_f\gamma_f}{d_f(d_f + \lambda_f + \gamma_f)}.$$

From system (2) we know that

$$\frac{d(S_o + E_o + I_o + V_o)}{dt} = A_o - d_o(S_o + E_o + I_o + V_o) - \alpha_o I_o \le A_o - d_o(S_o + E_o + I_o + V_o),$$

$$\frac{d(S_f + E_f + I_f + V_f)}{dt} = A_f - d_f(S_f + E_f + I_f + V_f) - \alpha_f I_f \le A_f - d_f(S_f + E_f + I_f + V_f),$$

then it follows that

$$\lim_{t\to\infty} \sup(S_o + E_o + I_o + R_o) \le \frac{A_o}{d_o}, \lim_{t\to\infty} \sup(S_f + E_f + I_f + R_f) \le \frac{A_f}{d_f},$$

$$\lim_{t\to\infty} \sup W \le (\frac{k_o A_o}{d_o} + \frac{k_f A_f}{d_f})\frac{1}{\delta + n\tau}.$$

So we can conclude that the set

$$\begin{aligned} X = \{&(S_o, E_o, I_o, V_o, S_f, E_f, I_f, V_f, W) | S_o, E_o, I_o, V_o, S_f, E_f, I_f, V_f, W \ge 0, \\ &0 \le S_o + E_o + I_o + V_o \le \frac{A_o}{d_o}, 0 \le S_f + E_f + I_f + R_f \le \frac{A_f}{d_f}, \\ &0 \le W \le (\frac{k_o A_o}{d_o} + \frac{k_f A_f}{d_f})\frac{1}{\delta + n\tau}.\} \end{aligned}$$

is positively invariant with respect to system (2).

We can calculate the control reproduction number of system (2) by the definition of $\mathcal{R}_c$ in [5, 8, 6], and the details are given in the Appendix A. For the disease-free equilibrium P_0, we have the following property.

Theorem 3.1. *If $\mathcal{R}_c < 1$, the disease-free equilibrium P_0 of system (2) is globally asymptotically stable in the region X.*

We put the proof of Theorem 3.1 in the Appendix B. Next we consider the existence of the positive solution of system (2). Define

$$X_0 = \{(S_o, E_o, I_o, V_o, S_f, E_f, I_f, V_f, W) \in X | E_o, I_o, E_f, I_f, W > 0\}, \partial X_0 = X | X_0.$$

To prove the main result about the existence of positive solution of system (2), we need the following Theorem.

Theorem 3.2. *When $\mathcal{R}_c > 1$, there exists a positive constant ε_1 such that when $\|E_o(0)\| < \varepsilon_1, \|I_o(0)\| < \varepsilon_1, \|E_f(0)\| < \varepsilon_1, \|I_f(0)\| < \varepsilon_1, \|W(0)\| < \varepsilon_1$ for $(S_o(0), E_o(0), I_o(0), V_o(0), S_f(0), E_f(0), I_f(0), V_f(0), W(0)) \in X_0$,*

$$\limsup_{t\to\infty} \max\{E_o(t), I_o(t), E_f(t), I_f(t), W(t)\} > \varepsilon_1.$$

We put the proof of Theorem 3.2 in the Appendix C. According to Theorem 3.2, we can get the following theorem about the existence of positive solution of system (2). Similarly, the reader can find the proof in the Appendix D.

Theorem 3.3. *If $\mathcal{R}_c > 1$, then system (2) admits at least one positive equilibrium, and there is a positive constant ε such that every solution $(S_o(t), E_o(t), I_o(t), V_o(t), S_f(t), E_f(t), I_f(t), V_f(t), W(t))$ of the system (2) with $((S_o(0), E_o(0), I_o(0), V_o(0), S_f(0), E_f(0), I_f(0), V_f(0), W(0)) \in X_0$ satisfies*

$$\min\{\liminf_{t\to\infty} E_o(t), \liminf_{t\to\infty} I_o(t), \liminf_{t\to\infty} E_f(t), \liminf_{t\to\infty} I_f(t), \liminf_{t\to\infty} W(t)\} \geq \varepsilon,$$

which implies that the system (2) is uniformly persistent.

So far, all our analysis focus on the mathematical model (2) and its dynamic behavior, such as the basic reproduction number, the existence of positive equilibrium. In the next section we will give data fitting about human brucellosis in Hinggan League of Inner Mongolia from 2001 to 2011 and sensitivity analysis on the basic production number.

4. Numerical simulations and sensitivity analysis.

Before carrying out the numerical simulations, we need to estimate the model parameters. The data concerning human brucellosis in Hinggan League of Inner Mongolia from 2001 to 2009 are obtained by [20] and the data from 2010 to 2011 are obtained mainly from epidemiological bulletins published by Hinggan League centers for disease control and prevention. However, the data involving sheep cannot be acquired easily, we have to rely on online news, our estimation or data fitting.

4.1. Estimation of epidemiological parameters and initial values.

The values of parameters are listed in Table 2. We explain the parameter values as follows:

[A] In Hinggan League of Inner Mongolia, basic ewes survival time is about 2-3 years and the number of basic ewes may reach about 4,200,000 in the future [9], so we estimate that basic ewes average removed rate is $d_f = \frac{1}{2.5} = 0.4$, and the recruitment rate of basic ewes is $A_f = 4,200,000 \times 0.4 = 1,680,000$. By [19], the removed rate of sheep is about 60%, so the parameter $d_o = 0.6$. In the same way we can give the new born number of other sheep is about $1,976,000$.

[B] In the fifth (2000) and sixth (2010) population census, the population of Hinggan League is 1,618,882 in 2000 and 1,613,250 in 2010 [28]. Because the human natural mortality rate of Inner Mongolia is $d_h = 0.00568$, we can estimate the annual birth rates of human is $A_h = 9,150$.

[C] From [11], the elimination rate of infected sheep is 0.15, so the seropositive detection rate of other sheep and basic ewes are $c_o = c_f = 0.15$. Quarantined seropositive infected sheep of Hinggan League can survival about 1 month, so the disease-related elimination rate of other sheep and basic ewes are $\alpha_o = \alpha_f = 12$.

For the initial values, $S_h(0) = 1,618,650$ and $I_h(0) = 56$ can be directly obtained, but $Y_h(0) = 157$ is assumed. In Inner Mongolia, there existed 35.51, 35.15 and 39.51 million sheep in 2000, 2001 and 2002, respectively. And in Hinggan League of Inner Mongolia there existed 5.6763 million sheep and 3.28 million basic ewes in 2002, so we can obtain there existed 5.102 million sheep and 2.96 million the basic ewes in 2000. Sheep vaccination rate of Inner Mongolia is only 31.6% [11], further we can estimate that the number of vaccinated basic ewes of Hinggan League in 2000 is 0.46 million ($2.96 \times 0.316 \times 0.6 \times 0.82$), that is $V_f(0) = 4.6 \times 10^5$. We assume the prevalence of brucellosis with basic ewes of Hinggan League in 2000 is about 1.0%,

so we obtain $E_f(0) = 19600, I_f(0) = 10000, S_f(0) = 2.46 \times 10^6$. In the same way we estimate that $S_o(0) = 1.78 \times 10^6, V_o(0) = 3.3 \times 10^5, E_o(0) = 14100, I_o(0) = 7300$. We assume that $W(0) = 1.0 \times 10^6$.

TABLE 2. The values of parameter in system (1).

Parameter	Mean value	Range	Unit	Source
A_o	1976000	None	$year^{-1}$	[A]
A_f	1680000	None	$year^{-1}$	[A]
A_h	9150	None	$year^{-1}$	[B]
d_o	0.6	(0-1)	$year^{-1}$	[A]
d_f	0.4	(0-1)	$year^{-1}$	[A]
λ_o	0.4	None	$year^{-1}$	[11]
λ_f	0.4	None	$year^{-1}$	[11]
γ_o	0.316×0.82	None	$year^{-1}$	[11]
γ_f	0.316×0.82	None	$year^{-1}$	[11]
c_o	0.15	95%,(0.1438,0.1562)	$year^{-1}$	[C]/LHS
c_f	0.15	95%,(0.1438,0.1562)	$year^{-1}$	[C]/LHS
α_o	12	None	$year^{-1}$	[C]
α_f	12	None	$year^{-1}$	[C]
k_o	15	None	$year^{-1}$	[11]
k_f	15	None	$year^{-1}$	[11]
δ	3.6	None	$year^{-1}$	[11]
n	0	None	$year^{-1}$	[11]
τ	0	None	$year^{-1}$	[11]
d_h	0.00568	None	$year^{-1}$	[19]
m	0.6	None	$year^{-1}$	[11]
p	0.4	None	$year^{-1}$	[11]
β_o	0.1×10^{-7}	$95\%,(0.0994, 0.1006) \times 10^{-7}$	$year^{-1}$	LHS
β_f	0.1×10^{-7}	$95\%,(0.0994, 0.1006) \times 10^{-7}$	$year^{-1}$	LHS
β_h	0.5271×10^{-10}	$95\%,(0.5135, 0.5407) \times 10^{-10}$	$year^{-1}$	Fitting
β_{oo}	0.18×10^{-6}	$95\%,(0.1794, 0.1806) \times 10^{-6}$	$year^{-1}$	LHS
β_{of}	0.135×10^{-6}	$95\%,(0.1344, 0.1356) \times 10^{-6}$	$year^{-1}$	LHS
β_{ff}	0.21×10^{-6}	$95\%,(0.2094, 0.2106) \times 10^{-6}$	$year^{-1}$	LHS
β_{fo}	0.135×10^{-6}	$95\%,(0.1344, 0.1356) \times 10^{-6}$	$year^{-1}$	LHS
β_{ho}	0.5896×10^{-9}	$95\%,(0.5338, 0.6454) \times 10^{-9}$	$year^{-1}$	Fitting
β_{hf}	1.3458×10^{-9}	$95\%,(1.3192, 1.3724) \times 10^{-9}$	$year^{-1}$	Fitting

4.2. **Numerical simulations.** Using system (1), we simulate newly infected human brucellosis data in Hinggan League of Inner Mongolia from 2001 to 2011 and predict the trend of newly infected human brucellosis infection. The numerical simulation of newly infected human brucellosis cases in Hinggan League from 2001 to 2011 is shown in Figure 3(a), which indicates that our model provides a good match to the reported data. In our paper, $Z(t)$ represents newly infected human brucellosis annually, and $Z(t) = X(t) - X(t-1)$, where,

$$\frac{dX(t)}{dt} = (\beta_{ho}E_o(t) + \beta_{hf}E_f(t) + \beta_h W(t))S_h(t).$$

Moreover, with the current control measures, our model indicates the tendency of newly infected human brucellosis cases epidemics with time, which is presented in Figure 3(b). It can be predicted that newly infected human brucellosis cases $Z(t)$ would continue increasing in the next seven or eight years, then the number of newly infected human brucellosis cases will decrease slowly. The tendency of newly infected human brucellosis data $Z(t)$ in Hinggan League of Inner Mongolia for long time is shown in Figure 3(c). Therefore, if no further effective prevention and control measures are taken, the disease will not vanish. With the parameter values in Table 2, we obtain the control reproduction number of Hinggan League brucellosis is about 1.9789, which indicates that human brucellosis will persist in Hinggan League of Inner Mongolia under the current control and prevention measures. From Hou et al. [11], we know that the control reproduction number of Inner Mongolia brucellosis is about 1.8. As to Hinggan League brucellosis is more serious than other city of Inner Mongolia, so the control reproduction number of Hinggan League brucellosis is a little bigger. Hence, the control reproduction number of Inner Mongolia $\mathcal{R}_c \approx 1.9789$ is reasonable.

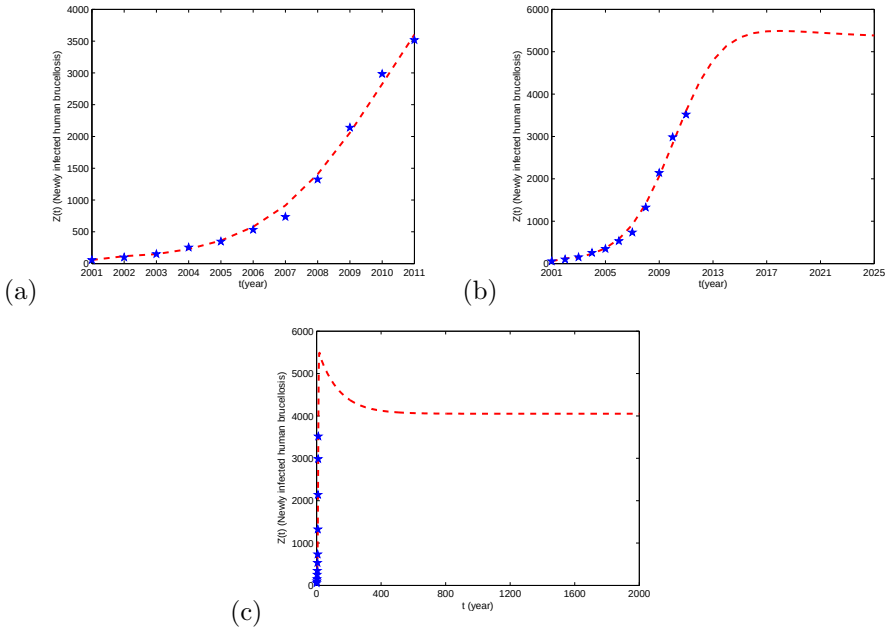

FIGURE 3. (a) The reported human brucellosis data in Hinggan League from 2001 to 2011 and the simulation of newly infected human brucellosis cases $Z(t)$ from the model. (b) The tendency of newly infected human brucellosis data $Z(t)$ in Hinggan League of Inner Mongolia with next 14 years. (c) The tendency of newly infected human brucellosis data $Z(t)$ in Hinggan League of Inner Mongolia for long time. The dashed curve is simulated by using our model, five-pointed star is the human brucellosis reported data. The values of parameters are given in Table 2, the unit is a year.

4.3. **Sensitivity and uncertainty analysis of $\mathcal{R}_c$.** The estimations of $\beta_{oo}, \beta_{of}, \beta_o, \beta_{ff}, \beta_{fo}, \beta_f$ were uncertain, so Latin hypercube sampling was used to sample these 6 parameters that appear in the derived expression for $\mathcal{R}_c$. In China, we

also know that control measures for brucellosis within livestock include detection, vaccination and elimination of the infected animals [22]. But from previous analysis of [C], we can see that the disease-related elimination rates of other sheep and basic ewes are $\alpha_o = \alpha_f = 12$. Hence, we only need to give the sensitivity analysis of $\mathcal{R}_c$ for the control parameters γ_o, γ_f, c_o and c_f. For the sensitivity analysis, Latin hypercube sampling was also used to sample these 4 control parameters that appear in the derived expression for $\mathcal{R}_c$. Uncertainty and sensitivity analysis based on Latin hypercube sampling has been previously applied to disease transmission models [3, 2, 24]. These 10 parameters included in the sensitivity analysis (Table 2) were sampled 1000 times within their respective ranges using a Latin hypercube sampling algorithm to optimize the sampling of parameter space. For the probability density function (PDF) of these parameters, a normal distribution was selected for these 10 parameters [26].

For each of the 1000 samples, we determined the aggregate $\mathcal{R}_c$. The distribution in values is shown for $\mathcal{R}_c$ in Figure 4. From the method of Latin hypercube sampling about these parameters, we know that the mean value of $\mathcal{R}_c$ is found to be 1.9895, the minimum value of $\mathcal{R}_c$ is found to be 1.2648 and the maximum value of $\mathcal{R}_c$ is found to be 5.039.

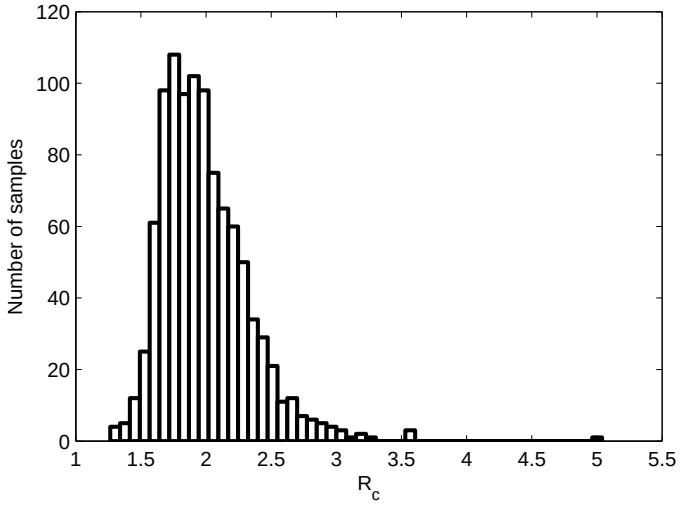

FIGURE 4. Histogram of the estimated aggregate $\mathcal{R}_c$ value from Latin hypercube sampling.

Partial rank correlation coefficients (PRCCs) for the aggregate $\mathcal{R}_c$ estimates and each of the 10 parameters are listed in Table 3 in descending order. The ordering of these PRCCs directly corresponds to the level of statistical influence the associated input parameter has on the variability of the aggregate $\mathcal{R}_c$. The PRCCs associated with the four control parameters $(c_f, c_o, \gamma_f, \gamma_o)$ that are the most influential in determining the magnitude of the aggregate $\mathcal{R}_c$, ($|\text{PRCC}| > 0.5$), are statistically significant ($p < 0.05$). The most influential control parameter on the aggregate $\mathcal{R}_c$ was found to be the seropositive detection rate of basic ewes c_f ($|\text{PRCC}|=0.9544$), and then in descending order were the efficient vaccination rate of basic ewes γ_f ($|\text{PRCC}|=0.8531$), the seropositive detection rate of other sheep c_o ($|\text{PRCC}|=0.7173$), the efficient vaccination rate of other sheep γ_o ($|\text{PRCC}|=0.5245$).

Due to the estimation uncertainty of six parameters $\beta_{oo}, \beta_{of}, \beta_o, \beta_{ff}, \beta_{fo}, \beta_f$. The PRCCs associated with these six parameters that are not influential in determining the magnitude of the aggregate $\mathcal{R}_c$, ($|\text{PRCC}| < 0.5$), are also statistically significant ($p < 0.05$).

TABLE 3. Partial rank correlation coefficients (PRCCs) for the aggregate $\mathcal{R}_c$ and each input parameter variable.

Input	$\mathcal{R}_c$		Input	$\mathcal{R}_c$	
parameter	PRCC	p value	Parameter	PRCC	p value
c_f	-0.9544	0.00	β_f	0.2720	0.00
γ_f	-0.8531	0.00	β_{of}	0.2586	0.00
c_o	-0.7173	0.00	β_{fo}	0.2231	0.00
γ_o	-0.5245	0.00	β_o	0.1452	0.00
β_{ff}	0.4398	0.00	β_{oo}	0.1130	0.00

4.4. **Newly infected human brucellosis cases $Z(t)$ in terms of control parameters.** In order to find better control strategies for brucellosis infection, we perform some analysis of newly infected human brucellosis cases $Z(t)$ in terms of different control measures. From previous sensitivity and uncertainty analysis of $\mathcal{R}_c$ we can see that the efficient vaccination rate of basic ewes (γ_f) is more influential than the efficient vaccination rate of other sheep (γ_o). In the real world, the detection methods of basic ewes and other sheep are the same, so in the following analysis we suggest the seropositive detection rates basic ewes and other sheep are also the same. We give the figure to show that the number of newly infected human brucellosis cases $Z(t)$ decreases with the increase of seropositive detection rates of basic ewes and other sheep (c_f, c_o). Therefore, the variations of newly infected human brucellosis cases $Z(t)$ with time for different control parameters and values of $\mathcal{R}_c$ in Figure 5, where corresponding to the control parameters are $\gamma_f = \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.15, \mathcal{R}_c \approx 1.9789$, $\gamma_f = \gamma_o = 1 \times 0.82, c_o = c_f = 0.15, \mathcal{R}_c \approx 1.3167$, $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.3, \mathcal{R}_c \approx 1.2217$, $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.5, \mathcal{R}_c \approx 0.9757$, respectively. Other parameters are same as in Table 2, in which 0.82 is the immunization protection rate of vaccine, the value is from Hou et al. [11]. From Figure 5 we can see that the number of newly infected human brucellosis cases $Z(t)$ increases with the increase of $\mathcal{R}_c$. Figure 5 indicates the final scale of newly infected human brucellosis cases $Z(t)$ will decrease when the efficient vaccination rates of basic ewes and other sheep γ_f, γ_o are increasing, and it can not eradicate the human brucellosis even if all sheep were vaccinated. When the efficient vaccination rates of basic ewes and other sheep are $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82$ and seropositive detection rates of basic ewes and other sheep recessive infected sheep are $c_o = c_f = 0.5$, the basic reproduction number is about $\mathcal{R}_c \approx 0.9757$, which means the human brucellosis of Hinggan League will vanish (see the solid line of Figure 5). The above analysis demonstrates that Hinggan League brucellosis can be controlled when the efficient vaccination rates of basic ewes and other sheep are $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82$ and seropositive detection rates of basic ewes and other sheep are $c_o = c_f = 0.5$. In

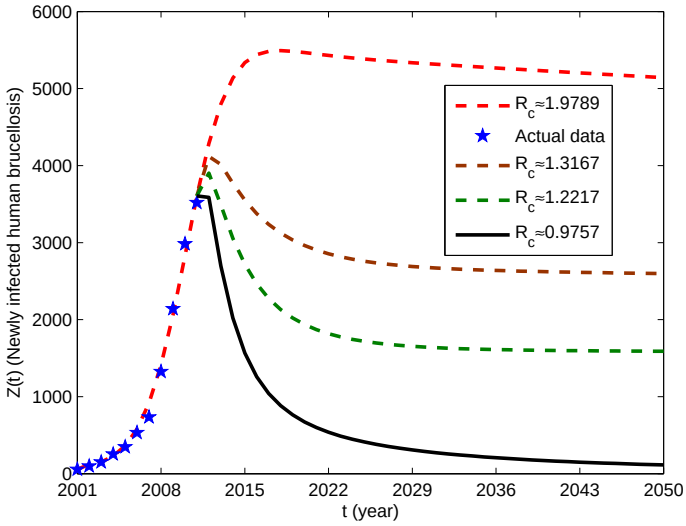

FIGURE 5. The variations of newly infected human brucellosis cases $Z(t)$ of system (1) for different control parameters and values of $\mathcal{R}_c$ since 2011. The unit is a year.

fact, it is difficult to reach this detection level, so we need to use other control measures for Hinggan League brucellosis. In the next section, we will give the influence of prohibiting mixed feeding between basic ewes and other sheep.

4.5. **The influence of prohibiting mixed feeding between basic ewes and other sheep.** In Hinggan League, basic ewes and other sheep are often mixed feeding together. Therefore, not only does there exist single species internal infection from basic ewes and other sheep, but also exists the mixed cross infection for brucellosis between basic ewes and other sheep. In order to cut the mixed cross infection for brucellosis between basic ewes and other sheep, the mixed feeding of flock must be prohibited. A comfortable strategy is quarantining basic ewes from the entire flock. In this circumstances, system (1) will become the following system

$$\begin{cases} \frac{dS_o}{dt} = A_o - (\beta_{oo}E_o + \beta_o W)S_o + \lambda_o V_o - (\gamma_o + d_o)S_o, \\ \frac{dE_o}{dt} = (\beta_{oo}E_o + \beta_o W)S_o - (c_o + d_o)E_o, \\ \frac{dI_o}{dt} = c_o E_o - (\alpha_o + d_o)I_o, \\ \frac{dV_o}{dt} = \gamma_o S_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} = A_f - (\beta_{ff}E_f + \beta_f W)S_f + \lambda_f V_f - (\gamma_f + d_f)S_f, \\ \frac{dE_f}{dt} = (\beta_{ff}E_f + \beta_f W)S_f - (c_f + d_f)E_f, \\ \frac{dI_f}{dt} = c_f E_f - (\alpha_f + d_f)I_f, \\ \frac{dV_f}{dt} = \gamma_f S_f - (\lambda_f + d_f)V_f, \\ \frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W, \\ \frac{dS_h}{dt} = A_h - (\beta_{ho}E_o + \beta_{hf}E_f + \beta_h W)S_h - d_h S_h + pI_h, \\ \frac{dI_h}{dt} = (\beta_{ho}E_o + \beta_{hf}E_f + \beta_h W)S_h - (m + d_h + p)I_h, \\ \frac{dY_h}{dt} = mI_h - d_h Y_h. \end{cases} \tag{4}$$

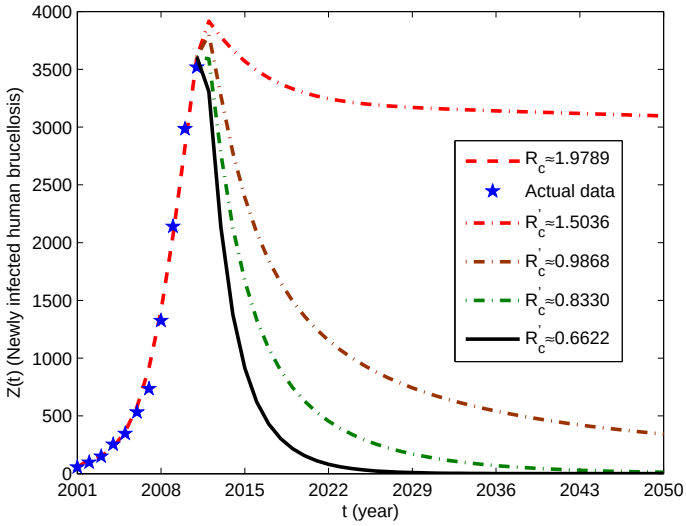

FIGURE 6. The variations of newly infected human brucellosis cases $Z(t)$ of system (4) for different control parameters and values of $\mathcal{R}_c^{'}$. The unit is a year.

We can calculate the expression of the control reproduction number $\mathcal{R}_c^{'}$ for system (4), and the details are also given in the Appendix A. We are interested in comparing the influence of existing mixed cross infection between basic ewes and other sheep or not for newly infected human brucellosis cases $Z(t)$. So we show the variations of newly infected human brucellosis cases $Z(t)$ of system (4) with time for different control parameters and values of $\mathcal{R}_c^{'}$ in Figure 6, where corresponding to the control parameters are $\gamma_f = \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.15, \beta_{of} = \beta_{fo} = 0, \mathcal{R}_c^{'} \approx 1.5036$, $\gamma_f = \gamma_o = 1 \times 0.82, c_o = c_f = 0.15, \beta_{of} = \beta_{fo} = 0, \mathcal{R}_c^{'} \approx 0.9868$, $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.3, \beta_{of} = \beta_{fo} = 0, \mathcal{R}_c^{'} \approx 0.8330$, $\gamma_f = 1 \times 0.82, \gamma_o = 0.316 \times 0.82, c_o = c_f = 0.5, \beta_{of} = \beta_{fo} = 0, \mathcal{R}_c^{'} \approx 0.6622$, respectively. Other parameters are same as in Table 2. In order to give the trend of newly infected human brucellosis cases $Z(t)$ for different control parameters in the future, we show the variations of newly infected human brucellosis cases $Z(t)$ of system (4) with time for different values of $\mathcal{R}_c^{'}$ in Figure 6. Comparing Figure 5 with Figure 6, we know that the reproduction number of system (4) is lower than system (1) in the same situation. From Figure 6 we can also see when the efficient vaccination rates and the seropositive detection rates of basic ewes and other sheep reach $\gamma_f = \gamma_o = 1 \times 0.82, c_o = c_f = 0.15$, the control reproduction number of system (4) is about $\mathcal{R}_c^{'} = 0.9868$, and the final scale of newly infected human brucellosis cases $Z(t)$ will tend to zero in the future. But in Figure 5, when the efficient vaccination rates and the seropositive detection rates of basic ewes and other sheep reach $\gamma_f = \gamma_o = 1 \times 0.82, c_o = c_f = 0.15$, the control reproduction number of system (1) is about $\mathcal{R}_c = 1.3167$, which means Hinggan League brucellosis will become endemic. Hence, the mixed cross infection between basic ewes and other sheep plays an important role in the persistence of brucellosis, and it is a very

important factor for brucellosis transmission. From Figure 6 we also observe that the extinction time of newly infected human brucellosis cases $Z(t)$ will come in advance if the seropositive detection rates of basic ewes and other sheep are increasing. So we can conclude that combination of prohibiting mixed feeding between basic ewes and other sheep, vaccination and detection is more effective than only combination of vaccination and detection to control brucellosis of Hinggan League.

5. **Conclusion and discussion.** Brucellosis is one of the biggest public health threats in China. Facing up to the epidemic situation in China, both the central and local governments have been seeking forceful methods to reduce brucellosis transmission. Various prevention and control measures have been proposed by many researchers, which include detection of infected animals, immunization of susceptible animals and elimination of infected animals [22]. Some researches suggest that combining these measures can be more effective in controlling the brucellosis. In this paper, we proposed a deterministic $S_oE_oI_oV_oS_fE_fI_fV_fWS_hI_hY_h$ model to describe the spread of brucellosis among sheep and from sheep to humans in Hinggan League of Inner Mongolia. The model simulations (see Figure 3(a)) agreed with the human brucellosis data reported by [20] and Hinggan League centers for disease control, and we gave an estimate of the control reproduction number $\mathcal{R}_c$, about 1.9789, which implies that brucellosis of Hinggan League cannot be controlled with the current strategies. By analyzing the tendency of newly infected human brucellosis cases $Z(t)$ with next 14 years (see Figure 3(b)), we can conclude that newly infected human brucellosis cases would increase in the next seven or eight years, and then decrease slowly. By some sensitivity and uncertainty analysis of the control reproduction number $\mathcal{R}_c$ on control parameters, we find that vaccination and detection are very important factors for brucellosis.Comparing Figure 5 with Figure 6, we know that if all sheep are vaccinated, the reproduction number of system (1) and (4) are 1.3167 and 0.9868, which means Hinggan League brucellosis will become endemic and disappeared, respectively. We can conclude that the mixed cross infection between basic ewes and other sheep is also important factor for brucellosis, so cutting the mixed cross infection is more important for brucellosis control. It only needs to prohibit mixed feeding between basic ewes and other sheep. In Hinggan League, vaccination, detection and elimination are the most commonly used control measures for brucellosis. However we do not have data on the cost of these control measures. It is well known that prohibiting mixed feeding between basic ewes and other sheep takes a small price to pay. In this sense, we suggest that brucellosis can be controlled by combination of prohibiting mixed feeding between basic ewes and other sheep, vaccination, detection and elimination.

All of our analysis demonstrate that the characteristics of brucellosis epidemics in Hinggan League of Inner Mongolia include the extremely low sheep vaccination rate and the seropositive detection rate of recessive infected sheep, poor understanding of the transmission dynamics of brucellosis and mixed cross infection between basic ewes and other sheep. Hou et al. [11] concluded that when both young and adult sheep vaccination rate reach 86% every year, human brucellosis in Inner Mongolia can be eradicated. Our study also shows that increasing sheep vaccination coverage rate is necessary and crucial in control brucellosis transmission in Hinggan League of Inner Mongolia. We suggest the first step is to cut the mixed cross infection between basic ewes and other sheep; and then increase the seropositive detection rate of recessive infected sheep; enhance the awareness of prevention brucellosis for herdsman; cull quarantined seropositive infected sheep in time.

It should be noted that there are some limitations in our model. Firstly, we only give the existence of the endemic equilibrium of system (2) when $\mathcal{R}_c > 1$ and we are interested in the uniqueness and global stability of the endemic equilibrium. Secondly, in the numerical simulations, we do not have the real data of c_f, c_o and the cost of every control strategy, so the best optimal control strategy can not be given. Thirdly, there exists a strong age structure, sex structure and occupational stigma for human brucellosis [17], in this paper, which was not taken into account. As a result, we need to address these features in the future.

Acknowledgments. We would like to the reviewers for their comments and suggestions that helped to improve this paper greatly.

Appendix A: Calculation of the control reproduction number of system (2) and (4). According to the definition of $\mathcal{R}_c$ in [5, 8, 6], we order the infected variables first by disease state, and only need the vector $x = (E_o, I_o, E_f, I_f, W)$. Considering the following auxiliary system:

$$\begin{cases} \frac{dE_o}{dt} = (\beta_{oo}E_o + \beta_{of}E_f + \beta_o W)S_o - (c_o + d_o)E_o, \\ \frac{dI_o}{dt} = c_o E_o - (\alpha_o + d_o)I_o, \\ \frac{dE_f}{dt} = (\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W)S_f - (c_f + d_f)E_f, \\ \frac{dI_f}{dt} = c_f E_f - (\alpha_f + d_f)I_f, \\ \frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W. \end{cases} \tag{5}$$

We follow the recipe from van den Driessche and Watmough [8] and Mukandavire et al. [18] to obtain

$$F = \begin{pmatrix} \beta_{oo}S_o^0 & 0 & \beta_{of}S_o^0 & 0 & \beta_o S_o^0 \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{fo}S_f^0 & 0 & \beta_{ff}S_f^0 & 0 & \beta_f S_f^0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} d_o + c_o & 0 & 0 & 0 & 0 \\ -c_o & d_o + \alpha_o & 0 & 0 & 0 \\ 0 & 0 & d_f + c_f & 0 & 0 \\ 0 & 0 & -c_2 & d_f + \alpha_f & 0 \\ -k_o & -k_o & -k_f & -k_f & \delta + n\tau \end{pmatrix}.$$

The inverse of V equals

$$V^{-1} = \begin{pmatrix} \frac{1}{d_o+c_o} & 0 & 0 & 0 & 0 \\ \frac{c_o}{(d_o+c_o)(d_o+\alpha_o)} & \frac{1}{d_o+\alpha_o} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{d_f+c_f} & 0 & 0 \\ 0 & 0 & \frac{c_f}{(d_f+c_f)(d_f+\alpha_f)} & \frac{1}{d_f+\alpha_f} & 0 \\ \frac{k_o(d_o+c_o+\alpha_o)}{(\delta+n\tau)(d_o+c_o)(d_o+\alpha_o)} & \frac{k_o}{(\delta+n\tau)(d_o+\alpha_o)} & \frac{k_f(d_f+c_f+\alpha_f)}{(\delta+n\tau)(d_f+c_f)(d_f+\alpha_f)} & \frac{k_f}{(\delta+n\tau)(d_f+\alpha_f)} & \frac{1}{\delta+n\tau} \end{pmatrix},$$

Hence, the next generation matrix is

$$FV^{-1} = \begin{pmatrix} A_{11} & A_{12} & A_{13} & A_{14} & A_{15} \\ 0 & 0 & 0 & 0 & 0 \\ A_{31} & A_{32} & A_{33} & A_{34} & A_{35} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

So the control reproduction number is

$$\mathcal{R}_c = \rho(FV^{-1}) = \frac{A_{11} + A_{33} + \sqrt{(A_{11} - A_{33})^2 + 4A_{13}A_{31}}}{2},$$

where

$$A_{11} = \frac{S_o^0}{d_o + c_o}(\beta_{oo} + \frac{\beta_o k_o(d_o + \alpha_o + c_o)}{(\delta + n\tau)(d_o + \alpha_o)}), A_{13} = \frac{S_o^0}{d_f + c_f}(\beta_{of} + \frac{\beta_o k_f(d_f + \alpha_f + c_f)}{(\delta + n\tau)(d_f + \alpha_f)}),$$

$$A_{31} = \frac{S_f^0}{d_o + c_o}(\beta_{fo} + \frac{\beta_f k_o(d_o + \alpha_o + c_o)}{(\delta + n\tau)(d_o + \alpha_o)}), A_{33} = \frac{S_f^0}{d_f + c_f}(\beta_{ff} + \frac{\beta_f k_f(d_f + \alpha_f + c_f)}{(\delta + n\tau)(d_f + \alpha_f)}),$$

$$S_o^0 = \frac{A_o(d_o + \lambda_o)}{d_o(d_o + \lambda_o + \gamma_o)}, S_f^0 = \frac{A_f(\lambda_f + d_f)}{d_f(\lambda_f + d_f + \gamma_f)}.$$

Due to the last three equations are independent of the first nine equations, so system (1) and system (2) have the same control reproduction number. Thus, the control reproduction number of (1) is also $\mathcal{R}_c$.

In the same way, we can calculate the control reproduction number of system (4) is

$$\mathcal{R}_c^{'} = \rho(FV^{-1}) = \frac{A_{11} + A_{33} + \sqrt{(A_{11} - A_{33})^2 + 4A_{13}^{'}A_{31}^{'}}}{2},$$

where

$$A_{11} = \frac{S_o^0}{d_o + c_o}(\beta_{oo} + \frac{\beta_o k_o(d_o + \alpha_o + c_o)}{(\delta + n\tau)(d_o + \alpha_o)}), A_{13}^{'} = \frac{\beta_o k_f(d_f + \alpha_f + c_f)S_o^0}{(d_f + c_f)(\delta + n\tau)(d_f + \alpha_f)},$$

$$A_{31}^{'} = \frac{\beta_f k_o(d_o + \alpha_o + c_o)S_f^0}{(d_o + c_o)(\delta + n\tau)(d_o + \alpha_o)}, A_{33} = \frac{S_f^0}{d_f + c_f}(\beta_{ff} + \frac{\beta_f k_f(d_f + \alpha_f + c_f)}{(\delta + n\tau)(d_f + \alpha_f)}),$$

$$S_o^0 = \frac{A_o(d_o + \lambda_o)}{d_o(d_o + \lambda_o + \gamma_o)}, S_f^0 = \frac{A_f(\lambda_f + d_f)}{d_f(\lambda_f + d_f + \gamma_f)}.$$

Appendix B: Global stability of the disease-free equilibrium of system (2).

Proof. Let $M = F - V$, and define $s(M) = \max\{Re\lambda : \lambda \text{ is an eigenvalue of } M\}$, so $s(M)$ is a simple eigenvalue of M with a positive eigenvector [27]. By the **Theorem 2** of van den Driessche and Watmough [8], there hold two equivalences:

$$\mathcal{R}_0 > 1 \Leftrightarrow s(M) > 0, \mathcal{R}_0 < 1 \Leftrightarrow s(M) < 0.$$

To prove the locally stability of disease-free equilibrium, we check the assumptions (A1)-(A5) in van den Driessche and Watmough [8]. Condition (A1)-(A4) are easily verified, while (A5) is satisfied if all eigenvalues of the 12×12 matrix

$$J\,|_{P_0} = \begin{pmatrix} M & 0 \\ J_3 & J_4 \end{pmatrix},$$

have negative real parts, where $J_3 = -F$

$$J_4 = \begin{pmatrix} -(d_o + \gamma_o) & \lambda_0 & 0 & 0 \\ \gamma_o & -(d_o + \lambda_o) & 0 & 0 \\ 0 & 0 & -(d_f + \gamma_f) & \lambda_f \\ 0 & 0 & \gamma_f & -(d_f + \lambda_f) \end{pmatrix}.$$

Calculated the eigenvalues of J_4,

$$s(J_4) = \max\{-d_o, -d_f, -(d_o + \lambda_o + \gamma_o), -(d_f + \lambda_f + \gamma_f)\} < 0.$$

If $\mathcal{R}_c < 1$, then $s(M) < 0$ and $s(J\mid_{P_0}) < 0$, the disease-free equilibrium P_0 of (2) is locally asymptotically stable.

From the fourth equation of system (2), we have

$$\begin{aligned}
\frac{dV_o}{dt} &= \gamma_o S_o - (\lambda_o + d_o)V_o \\
&= \gamma_o(N_o - (E_o + I_o + V_o)) - (\lambda_o + d_o)V_o \\
&\leq \gamma_o \frac{A_o}{d_o} - (\lambda_o + \gamma_o + d_o)V_o. \\
\frac{dV_f}{dt} &= \gamma_f S_f - (\lambda_f + d_f)V_f \\
&= \gamma_f(N_f - (E_f + I_f + V_f)) - (\lambda_f + d_f)V_f \\
&\leq \gamma_f \frac{A_f}{d_f} - (\lambda_f + \gamma_f + d_f)V_f.
\end{aligned}$$

So we can have that for a small enough positive number ϵ_1, there exists $t_o > 0$ and $t_f > 0$ such that for all $t > \max\{t_o, t_f\}$,

$$V_o \leq \frac{A_o\gamma_o}{d_o(\lambda_o + \gamma_o + d_o)} + \epsilon_1 = V_o^0 + \epsilon_1, V_f \leq \frac{A_f\gamma_f}{d_f(\lambda_f + d_f + \gamma_f)} + \epsilon_1 = V_f^0 + \epsilon_1.$$

Also from the equations of system (2), we have

$$\begin{aligned}
\frac{dS_o}{dt} &= A_o + \lambda_o V_o - (\gamma_o + d_o)S_o - (\beta_{oo}E_o + \beta_{of}E_f + \beta_o W)S_o \\
&\leq A_o + \lambda_o(V_o^0 + \epsilon_1) - (d_o + \gamma_o)S_o. \\
\frac{dS_f}{dt} &= A_f + \lambda_f V_f - (d_f + \gamma_f)S_f - (\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W)S_f \\
&\leq A_f + \lambda_f(V_f^0 + \epsilon_1) - (d_f + \gamma_f)S_f.
\end{aligned}$$

Then

$$\limsup_{t\to\infty} S_o = \frac{A_o + \lambda_o(V_o^0 + \epsilon_1)}{d_o + \gamma_o} = S_o^0 + \epsilon_2, (\epsilon_2 = \frac{\lambda_o\epsilon_1}{d_o + \gamma_o}).$$

$$\limsup_{t\to\infty} S_f = \frac{A_f + \lambda_f(V_f^0 + \epsilon_1)}{d_f + \gamma_f} = S_f^0 + \epsilon_3, (\epsilon_3 = \frac{\lambda_f\epsilon_1}{d_f + \gamma_f}).$$

We only need to consider system (2) and the feasible region X. From above conclusion, we know that $S_o \leq S_o^0 + \epsilon_2, S_f \leq S_f^0 + \epsilon_3$ with all $t > \max\{t_o, t_f\}$. Thus, when $t > \max\{t_o, t_f\}$, we derive

$$\begin{cases}
\frac{dE_o}{dt} \leq (S_o^0 + \epsilon_2)(\beta_{oo}E_o + \beta_{of}E_f + \beta_o W) - (d_o + c_o)E_o, \\
\frac{dI_o}{dt} = c_o E_o - (d_o + \alpha_o)I_o, \\
\frac{dE_f}{dt} \leq (S_f^0 + \epsilon_3)(\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W) - (d_f + c_f)E_f, \\
\frac{dI_f}{dt} = c_f E_f - (d_f + \alpha_f)I_f, \\
\frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W.
\end{cases}$$

Considering the following auxiliary system

$$\begin{cases} \frac{dE_o^{'}}{dt} = (S_o^0 + \epsilon_2)(\beta_{oo}E_o^{'} + \beta_{of}E_f^{'} + \beta_o W^{'}) - (d_o + c_o)E_o^{'}, \\ \frac{dI_o^{'}}{dt} = c_o E_o^{'} - (d_o + \alpha_o)I_o^{'}, \\ \frac{dE_f^{'}}{dt} = (S_f^0 + \epsilon_3)(\beta_{ff}E_f^{'} + \beta_{fo}E_o^{'} + \beta_f W^{'}) - (d_f + c_f)E_f^{'}, \\ \frac{dI_f^{'}}{dt} = c_f E_f^{'} - (d_f + \alpha_f)I_f^{'}, \\ \frac{dW^{'}}{dt} = k_o(E_o^{'} + I_o^{'}) + k_f(E_f^{'} + I_f^{'}) - (\delta + n\tau)W^{'}. \end{cases} \tag{6}$$

Let M_0 be the matrix defined by

$$M_0 = \begin{pmatrix} \beta_{oo} & 0 & \beta_{of} & 0 & \beta_o \\ 0 & 0 & 0 & 0 & 0 \\ \beta_{of} & 0 & \beta_{ff} & 0 & \beta_f \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and set $M_1 = M + \epsilon_4 M_0$. It follows from **Theorem 2** in van den Driessche and Watmough [8] that $\mathcal{R}_c < 1$ if and only if $s(M) < 0$. Thus, there exists an $\epsilon_4 > 0$ small enough such that $s(M_1) < 0$. Using the Perron-Frobenius theorem, all eigenvalues of the matrix M_1 have negative real parts when $s(M_1) < 0$. Therefore it has

$$(E_o^{'}(t), I_o^{'}(t), E_f^{'}(t), I_f^{'}(t), W^{'}(t)) \to (0, 0, 0, 0, 0), t \to \infty,$$

which implies that the zero solution of system (6) is globally asymptotically stable. Using the comparison principle of Smith and Waltman [27], we know that

$$(E_o(t), I_o(t), E_f(t), I_f(t), W(t)) \to (0, 0, 0, 0, 0), t \to \infty.$$

By the theory of asymptotic autonomous system of Thieme [29], it is also known that

$$(S_j(t), S_o(t), V_o(t), S_f(t), V_f(t)) \to (S_j^0, S_o^0, V_o^0, S_f^0, V_f^0), t \to \infty.$$

So P_0 is globally attractive when $\mathcal{R}_c < 1$. It follows that the disease-free equilibrium P_0 of (2.2) is globally asymptotically stable when $\mathcal{R}_c < 1$. The proof is end. □

Appendix C: The proof of Theorem 3.2.

Proof. Considering the following system

$$\begin{cases} \frac{dS_o}{dt} = A_o + \lambda_o V_o - (\gamma_o + d_o)S_o, \\ \frac{dV_o}{dt} = \gamma_o S_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} = A_f + \lambda_f V_f - (\gamma_f + d_f)S_f, \\ \frac{dV_f}{dt} = \gamma_f S_f - (\lambda_f + d_f)V_f. \end{cases} \tag{7}$$

Using **Corollary 3.2** in Zhao [38], it then follows that system (7) has a unique positive equilibrium $(S_o^0, V_o^0, S_f^0, V_f^0)$ and which is globally asymptotically stable.

As to $\mathcal{R}_c > 1 \Leftrightarrow s(M) > 0$, choose $\varepsilon > 0$ small enough such that $s(M_2) > 0$, where $M_2 = M - \varepsilon M_0$. Let us consider a perturbed system

$$\begin{cases} \frac{dS_o}{dt} = A_o - (d_o + \gamma_o)S_o + \lambda_o V_o - \varepsilon_1 S_o(\beta_{oo} + \beta_{of} + \beta_o), \\ \frac{dV_o}{dt} = \gamma_o S_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} = A_f - (d_f + \gamma_f)S_f + \lambda_f V_f - \varepsilon_1 S_f(\beta_{ff} + \beta_{fo} + \beta_f), \\ \frac{dV_f}{dt} = \gamma_f S_f - (\lambda_f + d_f)V_f. \end{cases} \tag{8}$$

Because the positive equilibrium $(S_o^0, V_o^0, S_f^0, V_f^0)$ of system (7) is globally asymptotically stable, we can restrict $\varepsilon_1 > 0$ small enough such that (8) admits a unique positive equilibrium $(S_o^0(\varepsilon_1), V_o^0(\varepsilon_1), S_f^0(\varepsilon_1), V_f^0(\varepsilon_1))$ which is globally asymptotically stable. $S_o^0(\varepsilon_1)$ and $S_f^0(\varepsilon_1)$ are continuous in ε_1, so we can further restrict ε_1 small enough such that $S_o^0(\varepsilon_1) > S_o^0 - \varepsilon$ and $S_f^0(\varepsilon_1) > S_f^0 - \varepsilon$.

For the sake of contradiction of **Theorem 2.2**, that there is a $T > 0$ such that $E_o(t) < \varepsilon_1, I_o(t) < \varepsilon_1, E_f(t) < \varepsilon_1, I_f(t) < \varepsilon_1, W(t) < \varepsilon_1$, for all $t \geq T$. Then for $t \geq T$, we have

$$\begin{cases} \frac{dS_o}{dt} \geq A_o - (d_o + \gamma_o)S_o + \lambda_o V_o - \varepsilon_1 S_o(\beta_{oo} + \beta_{of} + \beta_o), \\ \frac{dV_o}{dt} = \gamma_o S_o - (\lambda_o + d_o)V_o, \\ \frac{dS_f}{dt} \geq A_f - (d_f + \gamma_f)S_f + \lambda_f V_f - \varepsilon_1 S_f(\beta_{ff} + \beta_{fo} + \beta_f), \\ \frac{dV_f}{dt} = \gamma_f S_f - (\lambda_f + d_f)V_f. \end{cases}$$

Since the equilibrium $(S_o^0(\varepsilon_1), V_o^0(\varepsilon_1), S_f^0(\varepsilon_1), V_f^0(\varepsilon_1))$ of (8) is globally asymptotically stable and $S_o^0(\varepsilon_1) > S_o^0 - \varepsilon$, $S_f^0(\varepsilon_1) > S_f^0 - \varepsilon$. There exists a $T_1 > T > 0$ such that $S_o(t) > S_o^0 - \varepsilon$, $S_f(t) > S_f^0 - \varepsilon$ for $t > T_1$. As a consequence, for $t > T_1$, there holds

$$\begin{cases} \frac{dE_o}{dt} \geq (S_o^0 - \varepsilon)(\beta_{oo}E_o + \beta_{of}E_f + \beta_o W) - (d_o + c_o)E_o, \\ \frac{dI_o}{dt} = c_o E_o - (d_o + \alpha_o)I_o, \\ \frac{dE_f}{dt} \geq (S_f^0 - \varepsilon)(\beta_{ff}E_f + \beta_{fo}E_o + \beta_f W) - (d_f + c_f)E_f, \\ \frac{dI_f}{dt} = c_f E_f - (d_f + \alpha_f)I_f, \\ \frac{dW}{dt} = k_o(E_o + I_o) + k_f(E_f + I_f) - (\delta + n\tau)W. \end{cases}$$

Considering the following system

$$\begin{cases} \frac{dE_o^{'}}{dt} = (S_o^0 - \varepsilon)(\beta_{oo}E_o^{'} + \beta_{of}E_f^{'} + \beta_o W^{'}) - (d_o + c_o)E_o^{'}, \\ \frac{dI_o^{'}}{dt} = c_o E_o^{'} - (d_o + \alpha_o)I_o^{'}, \\ \frac{dE_f^{'}}{dt} = (S_f^0 - \varepsilon)(\beta_{ff}E_f^{'} + \beta_{fo}E_o^{'} + \beta_f W^{'}) - (d_f + c_f)E_f^{'}, \\ \frac{dI_f^{'}}{dt} = c_f E_f^{'} - (d_f + \alpha_f)I_f^{'}, \\ \frac{dW^{'}}{dt} = k_o(E_o^{'} + I_o^{'}) + k_f(E_f^{'} + I_f^{'}) - (\delta + n\tau)W^{'}. \end{cases}$$

Since the matrix M_2 have positive eigenvalue $s(M_2)$ with a positive eigenvector. It is easy to see that $(E_o^{'}(t), I_o^{'}(t), E_f^{'}(t), I_f^{'}(t), W^{'}(t)) \to (\infty, \infty, \infty, \infty, \infty)$ as $t \to \infty$. Using the comparison principle of Smith and Waltman [27], we also know that $(E_o(t), I_o(t), E_f(t), I_f(t), W(t)) \to (\infty, \infty, \infty, \infty, \infty)$ as $t \to \infty$, which leads to a

contradiction. Therefore we claim that

$$\limsup_{t\to\infty} \max\{E_o(t), I_o(t), E_f(t), I_f(t), W(t)\} > \varepsilon_1.$$

This completes the proof. □

Appendix D: The proof of Theorem 3.3.

Proof. Now we prove that system (2) is uniformly persistent with respect to $(X_0, \partial X_0)$. By the form of (2), it is easy to see that both X and X_0 are positively invariant and ∂X_0 is relatively closed in X. Furthermore system (2) is point dissipative. Let

$$M_\partial = \{(S_o(0), E_o(0), I_o(0), V_o(0), S_f(0), E_f(0), I_f(0), V_f(0), W(0))|(S_o(t), E_o(t),$$

$$I_o(t), V_o(t), S_f(t), E_f(t), I_f(t), V_f(t), W(t)) \in \partial X_0, \forall t \geq 0\}.$$

We now show that

$$M_\partial = \{(S_o(t), 0, 0, V_o(t), S_f(t), 0, 0, V_f(t), 0)|S_o(t), V_o(t), S_f(t), V_f(t) \geq 0.\} \quad (9)$$

Noting that $\{(S_o(t), 0, 0, V_o(t), S_f(t), 0, 0, V_f(t), 0)|S_o(t), V_o(t), S_f(t), V_f(t) \geq 0\} \subseteq M_\partial$, we only need to prove

$$M_\partial \subseteq \{(S_o(t), 0, 0, V_o(t), S_f(t), 0, 0, V_f(t), 0)|S_o(t), V_o(t), S_f(t), V_f(t) \geq 0\}.$$

Assume $(S_o(0), E_o(0), I_o(0), V_o(0), S_f(0), E_f(0), I_f(0), V_f(0), W(0)) \in M_\partial$. It suffices to show that $E_o(t) = 0, I_o(t) = 0, E_f(t) = 0, I_f(t) = 0, W(t) = 0, \forall t \geq 0$. Suppose not. Then there exist $t_0 \geq 0$ such that $E_o(t_0) = 0, I_o(t_0) = 0, E_f(t_0) > 0, I_f(t_0) > 0, W(t_0) = 0$, from the equation

$$\frac{dI_f(t_0)}{dt} = c_f E_f(t_0) - (d_f + \alpha_f) I_f(t_0) \geq c_f E_f(t_0).$$

It follows that there is an $\eta > 0$ such that $I_f(t) > 0$, for $t_0 < t < t_0 + \eta$. This means that $(S_o(t), E_o(t), I_o(t), V_o(t), S_f(t), E_f(t), I_f(t), V_f(t), W(t))$ does not belong ∂X_0 for $t_0 < t < t_0 + \eta$, which contradicts the assumption that $(S_o(0), E_o(0), I_o(0), V_o(0), S_f(0), E_f(0), I_f(0), V_f(0), W(0)) \in M_\partial$. This proves system (9),

$$M_\partial = \{(S_o(t), 0, 0, V_o(t), S_f(t), 0, 0, V_f(t), 0)|S_o(t), V_o(t), S_f(t), V_f(t) \geq 0.\}.$$

As to P_0 is globally asymptotically stable for system (2). It is clear that there is only a equilibrium P_0 in M_∂, by afore-mentioned claim, it then follows that P_0 is isolated invariant set in X, $W^s(P_0) \cap X_0 = \emptyset$. Clearly, every orbit in M_∂ converges to P_0, P_0 is acyclic in M_∂. Using **Theorem 4.6** in Thieme [30], we conclude that system (2) is uniformly persistent with respect to $(X_0, \partial X_0)$.

By the result of [37, 33, 32], system (2) has an equilibrium $(S_o^*, E_o^*, I_o^*, V_o^*, S_f^*, E_f^*, I_f^*, V_f^*, W^*) \in X_0$. We further claim that $S_o^*, V_o^*, S_f^*, V_f^* > 0$. Suppose that $S_o^* = V_o^* = S_f^* = V_f^* = 0$, from the equation of (2), we can get $E_o^* = I_o^* = E_f^* = I_f^* = W^* = 0$. It is a contradiction. Then $(S_o^*, E_o^*, I_o^*, V_o^*, S_f^*, E_f^*, I_f^*, V_f^*, W^*)$ is a componentwise positive equilibrium of system (2). This completes the proof. □

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