A COMPARISON OF DETERMINISTIC AND STOCHASTIC PREDATOR-PREY MODELS WITH DISEASE IN THE PREDATOR

HONGXIAO HU*
College of Science, University of Shanghai for Science and Technology
Shanghai 200093, China

LIGUANG XU
Department of Applied Mathematics, Zhejiang University of Technology
Hangzhou 310023, China

KAI WANG
Department of Medical Engineering and Technology, Xinjiang Medical University
Urumqi 830011, China

(Communicated by Thomas Wanner)

ABSTRACT. In this paper, we study the dynamics of deterministic and stochastic models for a predator-prey, where the predator species is subject to an SIS form of parasitic infection. The deterministic model is a system of ordinary differential equations for a predator-prey model with disease in the predator only. The existence and local stability of the boundary equilibria and the uniform persistence for the ODE model are investigated. Based on these results, some threshold values for successful invasion of disease or prey species are obtained. A new stochastic model is derived in the form of continuous-time Markov chains. Branching process theory is applied to the continuous-time Markov chain models to estimate the probabilities for disease outbreak or prey species invasion. The deterministic and stochastic threshold theories are compared and some relationships between the deterministic and stochastic thresholds are derived. Finally, some numerical simulations are introduced to illustrate the main results and to highlight some of the differences between the deterministic and stochastic models.

1. Introduction. Eco-epidemiology is an important branch in mathematical biology, which both considers the ecology and the epidemiology issues simultaneously. In [5], Anderson and May first founded an eco-epidemiology model. Following their work, many researchers have proposed and investigated different models for studying the spread of diseases among interacting species (see [8, 9, 10, 19, 21, 22, 23, 26, 39, 40, 42] and the references therein).

Particularly, a transmissible disease in the predator population also is a very common phenomenon in ecological species. The biologically relevant examples for
this phenomenon in a number of ecosystems are found in Table 2 of [16]. For examples, rabies and Sarcoptes spp. in foxes (Vulpis vulpis) and coyotes (Canis latrans), where the prey is then given by rabbits; the Phocine Distemper Virus in both the common seal (Phoca vitulina) and the striped dolphin (Stenella coeruleoalba), Otostrongylus circumlitis, and Uncinaria lucasi, affecting ringed seals (Phoca hispida) and northern fur seals (Callorhinus ursinus) respectively, in this case the obvious prey are fishes; finally, the Avian Pox, Newcastle Disease, Influenza, Pasteurella multocida, Apergillus fumigatus and Leukocytozoon simondi are infectious agents all affecting a wide range of avian species, in this case the prey are mainly insects (see [40]).

In this paper, we assume the disease does not cross the species barrier and consider an eco-epidemiological predator-prey model with disease in the predator only. In our knowledge, many researchers are interested in the dynamics of the predator-prey system with disease in the predator only (see [9, 24, 25, 36, 40, 43] and the references therein). Venturino [40] formulated some eco-epidemiological models with disease in the predators only based on the classical Lotka-Volterra type predator-prey model and SIS epidemic model. In these models, the authors assumed that the infected individuals do not reproduce and have weak ability to catch prey. By studying the long-term behavior of these models they show whether and how the presence of the disease in the predator species affects the behavior of the ecological system. But also they point out whether the introduction of a sound prey can affect the dynamics of the disease in the predator population. Following the work [40], Haque and Venturino [25] studied a ratio-dependent eco-epidemiological predator-prey model with non-recoverable disease in the predator species. Equilibria of the system are analysed and the disease effects on the model are discussed. Suppose the predator has an alternative source of food, Haque [24] studied a predator-prey model with logistic growth in the prey population that includes an SIS parasitic infection in the predator population. They pointed out that the infection in the predator species can be taken as biological control. Unlike previous models, the model considered here includes the recovery rate and the disease-related mortality rate. In addition, we assume that the predators have a logistic growth rate and the infected predators have less efficiency to capture prey and less “return” compared to the healthy predators.

On the other hand, Whittle [41] first applied branching process theory to the one species SIS continuous-time Markov chain (CTMC) process in 1955. They derived a good approximation to the probability of an outbreak when the initial number of infectives is small in the population density. In Whittle’s approximation, when the basic reproduction number satisfies $R_0 > 1$, the probability of an outbreak and disease extinction are $1 - 1/R_0$ and $1/R_0$, respectively, where $i$ is the initial number of infectious. Hence, the stochastic thresholds are closely related to the deterministic thresholds but also depend on the initial number of infected individuals. Following Whittle’s work, the branching process theory is applied frequently to estimate the probability of extinction to populations or epidemics CTMC models (see [2, 3, 4, 11, 12, 18, 28, 29, 30, 35, 44]). Recently, some scholars applied the branching process to research the competing interacting species with disease (see [2, 12, 29, 30]). They formulated new stochastic models based on CTMCs and stochastic differential equations (SDEs) for the deterministic models. Applying the
branching process theory to the CTMC models, they obtained approximate probabilities for pathogen coexistence, pathogen extinction or species invasion. Comparing the dynamics of the stochastic epidemic model to the deterministic epidemic model by numerical simulations, they shown that the two models differ considerably in predicting the pathogen coexistence, pathogen extinction or species invasion. But, to our knowledge, little attention has been paid to the interaction between predator-prey and disease in the stochastic setting.

Inspired by above works, a new deterministic predator-prey species model with disease in the predator is founded to include an SIS parasitic infection (see Section 2). The existence and stability of the boundary equilibrium points along with the uniform persistence are studied for the ODE model. Based on the ODE model we formulate a new CTMC model (see Section 3). Applying the branching process theory to the CTMC model, we estimate the probabilities for disease outbreak or successful invasion of prey species when the basic reproduction number is greater than the threshold value. This property is totally different to the ODE model in predicting the disease outbreak or prey species invasion. Numerical examples are introduced to illustrate three distinct cases (see Section 4), with sample paths from the CTMC model compared to solutions of the ODE model. Furthermore, using the Kolmogorov-Smirnov test and the Shapiro-Wilk normality test, we find that the CTMC sample paths have a quasi-stationary distribution, which follows the normal distribution. Finally, we close the paper with conclusions and remarks in Section 5.

2. ODE model.

2.1. Model assumptions and formulation. We have two populations: the prey and the predator species, whose population densities at time $t$ are denoted by $x_1(t)$ and $x_2(t)$, respectively. We make the following assumptions to formulate the mathematical model:

$(H_1)$ The disease spreads within the predator population only. In the presence of disease, the predator population $x_2$ is divided into two classes: susceptible predator $S$ and infected predator $I$. That is $x_2 = S + I$.

$(H_2)$ The disease-free predator-prey model satisfies the classical Lotka-Volterra type:

$$
\begin{aligned}
\dot{x}_1 &= x_1(r_1 - a_1x_1 - \eta x_2), \\
\dot{x}_2 &= x_2(r_2 + \epsilon \eta x_1 - a_2x_2),
\end{aligned}
$$

where $r_i$ and $a_i$ are the intrinsic per capita growth rate and the intraspecific competition rate of $x_i$ for $i = 1, 2$, respectively, $\eta$ is the predation rate, $\epsilon$ is the conversion factor.

$(H_3)$ The infected predators have less efficiency to catch the prey and have less ‘return’ by catching a prey compared to the healthy predators. Hence, we assume $p\eta$ and $q\epsilon$ are the predation rate and the conversion rate of an infected predator, respectively, where $0 \leq p, q \leq 1$.

$(H_4)$ The increased densities through intraspecific crowding have a negative impact on reproduction and survival. In particular, for $i = 1, 2$, the birth rate of species $x_i$ can be reduced to $x_i(b_i - c_i a_i x_i) > 0$, whereas the death rate of species $x_i$ can be increased to $x_i(d_i + (1 - c_i) a_i x_i)$, where $b_i$ and $d_i$ are the birth rate and the death rate of the species $x_i$, respectively, $r_i = b_i - d_i$. The parameters $0 \leq c_i \leq 1$ subdivide the intraspecific competition effects into those affecting births ($c_i$) and those affecting deaths ($1 - c_i$).
(H5) The disease cannot be transmitted vertically. The infected predator population has a recovery rate $\gamma$ and a mortality rate $\mu$. The transmission rate among the susceptible predator population and infected predator population follows the simple mass action with transmission rate $\beta$.

Based on the aforementioned assumptions, we obtain the following SIS type model:

$$
\begin{align*}
    \dot{x}_1 &= x_1 \left( r_1 - a_1 x_1 - \eta(S + q I) \right), \\
    \dot{S} &= \varepsilon \eta x_1 S + x_2 (b_2 - c_2 a_2 x_2) - S(d_2 + (1 - c_2) a_2 x_2) - \beta S I + \gamma I, \\
    \dot{I} &= pq \varepsilon \eta x_1 I + \beta S I - I(d_2 + (1 - c_2) a_2 x_2) - \gamma I - \mu I,
\end{align*}
$$

where all of the coefficients are positive except $r_2 \in \mathbb{R}$ and $0 \leq p, q, c_i \leq 1, i = 1, 2$. By the second and third equations of (2), we have

$$
\dot{x}_2 = \varepsilon \eta x_1 (S + q I) + x_2 (r_2 - a_2 x_2) - \mu I. 
$$

In the following, we will give some useful notations, which will be used in this paper.

$$
\begin{align*}
    \mathbb{R}^n &= \{(y_1, y_2, \cdots, y_n) : y_i \in \mathbb{R}, i = 1, 2, \cdots, n\}, \\
    \mathbb{R}^n_+ &= \{(y_1, y_2, \cdots, y_n) : y_i \geq 0, i = 1, 2, \cdots, n\}, \\
    u_0 &= \frac{r_1}{a_1}, \quad v_0 = \frac{r_2}{a_2}, \\
    \Sigma &= \{(x_1, S, I) \in \mathbb{R}^3 : 0 \leq x_1 \leq u_0, 0 \leq S + I \leq v_0 + \frac{\varepsilon \eta u_0}{a_2}\}, \\
    \tilde{x}_1 &= \frac{u_0 - \eta v_0/a_1}{1 + \varepsilon \eta^2/a_1 a_2}, \quad \tilde{x}_2 = \frac{v_0 + \varepsilon \eta u_0/a_2}{1 + \varepsilon \eta^2/a_1 a_2}, \\
    \tilde{R} &= \frac{pq \varepsilon \eta u_0}{d_2 + \gamma + \mu}, \quad \tilde{R}_1 = \frac{\beta \tilde{x}_2}{d_2 + \gamma + \mu + (1 - c_2) a_2 \tilde{x}_2 - pq \varepsilon \eta \tilde{x}_1}.
\end{align*}
$$

Remark 1. $-\varepsilon \eta u_0/a_2 < v_0 < r_1/\eta$ is the necessary and sufficient condition for $\tilde{x}_1, \tilde{x}_2 > 0$. $R_0$ always is positive when $v_0 > 0$. When $\tilde{x}_1, \tilde{x}_2 > 0$, the sign of $R_1$ is determined by the denominator of $R_1$. But by the biological meaning, $R_0$ and $R_1$ are the basic reproduction numbers at the disease-free equilibrium points $(0, v_0, 0)$ and $(\tilde{x}_1, \tilde{x}_2, 0)$, respectively (see Section 2.3). Therefore $R_0$ and $R_1$ always are positive on the practical situation.

The disease-free predator-prey system (1) has been studied by many researchers. Combining with the results in [14, 15] (see the Theorem 3.7.1 in [14] and main theorem in [15]), it is easy to show that

**Lemma 2.1.** $\mathbb{R}_+^2$ is a positive invariant set of system (1). For solution paths of system (1) in $\mathbb{R}_+^2$:

(a) If $v_0 \leq -\varepsilon \eta u_0/a_2$, paths with $x_1(0) = 0$ and $x_2(0) > 0$ go to $N_0(0, 0)$, and paths with $x_1(0) > 0$ and $x_2(0) \geq 0$ go to $N_1(u_0, 0)$.

(b) If $-\varepsilon \eta u_0/a_2 < v_0 \leq 0$, paths with $x_1(0) > 0$ and $x_2(0) = 0$ go to $N_1(u_0, 0)$, paths with $x_1(0) = 0$ and $x_2(0) > 0$ go to $N_0(0, 0)$, paths with $x_1(0) > 0$ and $x_2(0) > 0$ go to $N_3(\tilde{x}_1, \tilde{x}_2)$.

(c) If $0 < v_0 < r_1/\eta$, paths with $x_1(0) > 0$ and $x_2(0) = 0$ go to $N_1(u_0, 0)$, paths with $x_1(0) = 0$ and $x_2(0) > 0$ go to $N_2(0, v_0)$, paths with $x_1(0) > 0$ and $x_2(0) > 0$ go to $N_3(\tilde{x}_1, \tilde{x}_2)$.

(d) If $v_0 \geq r_1/\eta$, paths with $x_1(0) > 0$ and $x_2(0) = 0$ go to $N_1(u_0, 0)$, and paths with $x_1(0) \geq 0$ and $x_2(0) > 0$ go to $N_2(0, v_0)$. 


When $x_1 = 0$ in the model (2), we obtain the prey-absent system
\[
\begin{cases}
\dot{S} = x_2(b_2 - c_2a_2x_2) - S(d_2 + (1 - c_2)a_2x_2) - \beta SI + \gamma I, \\
\dot{I} = \beta SI - I(d_2 + (1 - c_2)a_2x_2) - \gamma I - \mu I,
\end{cases}
\tag{4}
\]
and have following lemma.

**Lemma 2.2.** (See Appendix A for the proof) $\mathbb{R}_+^2$ is a positive invariant set of system (4). The trivial equilibrium $(0,0)$ in model (4) is globally asymptotically stable when $r_2 \leq 0$. When $r_2 > 0$, we have following two cases:

(a) If $R_0 \leq 1$, the disease-free equilibrium point $(v_0,0)$ of model (4) is globally asymptotically stable in $\mathbb{R}_+^2 \setminus \{(0,0)\}$;

(b) If $R_0 > 1$, model (4) has a unique positive equilibrium $(\bar{S}, \bar{I})$ with $\bar{x}_2 = \bar{S} + \bar{I} \in (0, v_0)$, which is globally asymptotically stable in the interior of $\mathbb{R}_+^2$, and $(0,0)$ and $(v_0, 0)$ are unstable.

**Lemma 2.3.** Let $(x_1(t), S(t), I(t))$ be the solution of system (2) with initial value $(x_1(0), S(0), I(0)) \in \mathbb{R}_+^3$. Then $\lim_{t \to \infty} (x_1(t), S(t), I(t)) \in \Sigma$, i.e. system (2) is point dissipative (i.e. the existence of a bounded globally attracting set, see [20, 38]).

**Proof.** By the first equation of system (2), we have $\dot{x}_1 \leq x_1(r_1 - a_1x_1)$. It follows from comparison theorem that $\limsup_{t \to \infty} x_1(t) \leq u_0$. Hence, by (3), for any small $\theta > 0$ if $t$ is large enough, $x_2$ satisfies
\[
\dot{x}_2 \leq x_2(\varepsilon \eta u_0 + \theta) + r_2 - a_2x_2.
\tag{5}
\]
When $v_0 \leq -\varepsilon \eta u_0 / a_2$, from (5) we get $\dot{x}_2 \leq x_2(\varepsilon \theta - a_2x_2)$. It follows from the arbitrary choice of $\theta$ that $\lim_{t \to \infty} x_2(t) = 0$. When $v_0 > -\varepsilon \eta u_0 / a_2$, by the comparison theorem and (5), yields
\[
\limsup_{t \to \infty} x_2(t) \leq \frac{\varepsilon \eta u_0 + r_2}{a_2} = v_0 + \frac{\varepsilon \eta u_0}{a_2}.
\]
This completes the proof. \qed

Consider the systems:
\[
\begin{align*}
\dot{u} &= H(t, u), \\
\dot{z} &= h(z),
\end{align*}
\tag{6}
\tag{7}
\]
where $H(t, u)$ and $h(u)$ are continuous and locally Lipschitz in $u$ in $\mathbb{R}^n$ and solutions exist for all positive time.

**Definition 2.4.** Equation (6) is called asymptotically autonomous with limit equation (7) if $\lim_{t \to \infty} H(t, u) = h(u)$ uniformly for $u \in \mathbb{R}^n$.

**Lemma 2.5.** (See [37]) Let $e$ be a locally asymptotically stable equilibrium of (7) and $\omega$ be the $\omega$-limit set of a forward bounded solution $u(t)$ of (6). If $\omega$ contains a point $z_0$ such that the solution of (7), with $z(0) = z_0$ converges to $e$ as $t \to \infty$, then $\omega = \{e\}$, i.e. $\lim_{t \to \infty} u(t) = e$.

2.2. Model analysis. System (2) has five boundary equilibrium points in the form $(x_1, S, I)$ as following,
\[
E_0 = (0,0,0), \quad E_1 = (v_0,0,0), \quad E_2 = (0,v_0,0), \quad E_3 = (\bar{x}_1, \bar{x}_2, 0), \quad E_4 = (0, \bar{S}, \bar{I}),
\]
where $\bar{I} = f(\bar{x}_2)$, $\bar{S} - \bar{I}$ and $\bar{x}_2$ is the positive solution of equation $f(x) = g(x)$ in $(0, v_0)$ with $v_0 > 0$, where
\[
f(x) := \frac{x(r_2 - a_2x)}{\mu} \quad \text{and} \quad g(x) := \frac{\beta - (1 - c_2)a_2x - d_2 + \gamma + \mu}{\beta}.
\]
The trivial equilibrium point $E_0$ and axial equilibrium point $E_1$ exist for all parametric values. The axial equilibrium point $E_2$ exists if $r_2 > 0$. The disease-free equilibrium point $E_3$ exists when $-\varepsilon \eta u_0/a_2 < v_0 < r_1/n$. Since $f(x)$ has two zero points 0 and $v_0$, $g(0) = -(d_2 + \gamma + \mu)/\beta < 0$ and $g(v_0) = v_0(1 - 1/R_0)$ (see Fig. 1), the feasibility conditions of the prey-absent equilibrium point $E_4$ are $r_2 > 0$ and $R_0 > 1$.

In order to discuss the locally stability of each boundary equilibria, the Jacobian matrix $J(E) = (J_{ij})_{3\times3}$ of system (2) at any arbitrary point $E(x_1, S, I)$ is calculated and it has the following form:

$$J(E) = \begin{pmatrix} r_1 - 2a_1x_1 - \eta(S + qI) & -\eta x_1 & -q\eta x_1 \\ \varepsilon\eta S & J_{22} & J_{23} \\ pq\varepsilon\eta I & (\beta - (1-c_2)a_2)I & J_{33} \end{pmatrix}, \quad (8)$$

where

$$J_{22} = \varepsilon \eta x_1 + r_2 - 2c_2a_2x_2 - (1-c_2)a_2x_2 - (1-c_2)a_2S - \beta I,$$

$$J_{23} = b_2 - 2c_2a_2x_2 - ((1-c_2)a_2 + \beta)S + \gamma,$$

$$J_{33} = pq\varepsilon\eta x_1 + \beta S - (d_2 + (1-c_2)a_2x_2) - \gamma - \mu - (1-c_2)a_2I.$$

The eigenvalues of Jacobian matrix $J(E_0)$ in $x_1$, $S$ and $I$ directions are $r_1$, $r_2$ and $-(d_2 + \gamma + \mu)$, respectively. The eigenvalues of $J(E_1)$ in the directions $x_1$, $S$ and $I$ are $-r_1$, $r_2 + \varepsilon \eta u_0$ and $-pq\varepsilon\eta u_0(1-1/R)$, respectively. And the eigenvalues of $J(E_2)$ in the $x_1$, $S$ and $I$ directions are $r_1 - \eta v_0$, $-r_2$ and $\beta v_0(1-1/R_0)$, respectively.

At the equilibrium point $E_3$, we calculate the Jacobian matrix

$$J(E_3) = \begin{pmatrix} -a_1\tilde{x}_1 & -\eta\tilde{x}_1 & -q\eta\tilde{x}_1 \\ \varepsilon\eta\tilde{x}_2 & -a_2\tilde{x}_2 & * \\ 0 & 0 & \beta\tilde{x}_2(1 - \frac{1}{R_1}) \end{pmatrix}.$$ 

Let the upper left block of $J(E_3)$ be

$$A = \begin{pmatrix} -a_1\tilde{x}_1 & -\eta\tilde{x}_1 \\ \varepsilon\eta\tilde{x}_2 & -a_2\tilde{x}_2 \end{pmatrix}.$$ 

Consequently, we have

$$\text{Trace}(A) = -(a_1\tilde{x}_1 + a_2\tilde{x}_2) < 0 \quad \text{and} \quad \text{Det}(A) = (a_1a_2 + \varepsilon\eta^2)\tilde{x}_1\tilde{x}_2 > 0.$$ 

The characteristic equation of $A$ is

$$\lambda^2 - \text{Trace}(A)\lambda + \text{Det}(A) = 0.$$
Denote by $\lambda_1$ and $\lambda_2$ the two eigenvalues of $A$. If $\lambda_1, \lambda_2 \in \mathbb{R}$, then $\lambda_1 + \lambda_2 = \text{Trace}(A) < 0$ and $\lambda_1\lambda_2 = \text{Det}(A) > 0$. That is the two eigenvalues of $A$ are negative or $\text{Re}(\lambda_1) = \text{Re}(\lambda_2) = \text{Trace}(A) < 0$. Therefore, the eigenvalues in the $x_1$ and $S$ directions have negative real parts. The third eigenvalue of $J(E_4)$ is $\beta \bar{x}_2 (1 - 1/R_1)$.

Calculating the Jacobian matrix at the equilibrium point $E_4$,

$$
J(E_4) = \begin{pmatrix}
  r_1 - \eta(\bar{S} + q\bar{I}) & 0 & 0 \\
  \varepsilon\eta\bar{S} & J_{22}(E_4) & J_{23}(E_4) \\
  p\varepsilon\eta\bar{I} & (\beta - (1-c_2)a_2)\bar{I} & -(1-c_2)a_2\bar{I}
\end{pmatrix}.
$$

By the second equation of system (2), it has

$$
\bar{S}(r_2 - a_2\bar{x}_2) = \bar{I}(I\bar{S} - \gamma - b_2 + c_2a_2\bar{x}_2).
$$

Substituting (9) into $J_{22}(E_4)$ and $J_{23}(E_4)$ yields,

$$
J_{22}(E_4) = -\frac{1}{\bar{S}}(-c_2a_2I^2 + (\gamma + b_2)\bar{I}) - a_2\bar{S}
$$

and

$$
J_{23}(E_4) = -a_2(v_0 - \bar{x}_2)\frac{\bar{S}}{\bar{T}} - c_2a_2\bar{x}_2 - (1-c_2)a_2\bar{S} < 0.
$$

Since $(\gamma + b_2)/c_2a_2 > v_0$, the quadratic polynomial

$$
-c_2a_2I^2 + (\gamma + b_2)I > 0, \text{ for all } I \in (0,v_0).
$$

Consequently, $J_{22}(E_4) < 0$. By $R_0 > 1$, it has $\beta > (1-c_2)a_2$. Consequently, the eigenvalues in the $S$ and $I$ directions are negative. The eigenvalue in the $x_1$ direction is $r_1 - \eta(\bar{S} + q\bar{I})$. According to the above analysis, we have the following theorem.

**Theorem 2.6.** The existence and local stability of each boundary equilibrium point is as following:

(a) When $v_0 \leq -\varepsilon\eta\mu_0/a_2$, $E_0$ and $E_1$ exist, $E_0$ is unstable and $E_1$ is locally stable.

(b) When $-\varepsilon\eta\mu_0/a_2 < v_0 \leq 0$, there are three boundary equilibrium points, which are $E_0$, $E_1$, $E_2$, $E_0$ and $E_1$ are unstable, $E_3$ is locally stable if $R_1 < 1$ while it is unstable if $R_1 > 1$.

(c) When $0 < v_0 < r_1/\eta$, there are four boundary equilibrium points, which are $E_0$, $E_1$, $E_2$ and $E_3$. $E_4$ exists if $R_0 > 1$. $E_0$, $E_1$, $E_2$ and $E_4$ are always unstable. $E_3$ is locally stable if $R_1 < 1$ while it is unstable if $R_1 > 1$.

(d) When $v_0 \geq r_1/\eta$, there are three boundary equilibrium points, which are $E_0$, $E_1$ and $E_2$. $E_3$ and $E_1$ are unstable. $E_3$ exists if $R_0 < 1$. $E_0$, $E_1$, $E_2$ and $E_4$ are always unstable. $E_3$ is locally stable if $R_1 < 1$ while it is unstable if $R_1 > 1$. The prey-absent equilibrium point $E_4$ exists if $R_0 > 1$. $E_4$ is locally stable if $r_1/\eta < \bar{S} + q\bar{I}$ while it is unstable if $r_1/\eta > \bar{S} + q\bar{I}$.

**Proof.** (a) By $v_0 \leq -\varepsilon\eta\mu_0/a_2$, it has $d_2 - \varepsilon\eta\mu_0 \geq b_2 > 0$. Hence, $R < 1$ and $E_1$ is unstable. (c) When $0 < v_0 < r_1/\eta$ and $R_0 > 1$, we obtain $\bar{S} + q\bar{I} < \bar{x}_2 < v_0 < r_1/\eta$. Therefore, $E_4$ is unstable. The other cases have been analyzed by the above. This completes the proof.

It is difficult to study the existence of positive equilibrium point of system (2). Here we prove some results on the uniform persistence of the system (2) by using the Theorem 4.6 in [38].
Definition 2.7. (see [38]) A component \( x_1(t) \) of a positive solution of the model (2) is defined to be uniformly persistent if there is a positive constant \( \delta \) such that \( \liminf_{t \to \infty} x_1(t) > \delta \) for any initial value.

Definition 2.8. The model (2) is defined to be uniformly persistent if there is a positive constant \( \delta \) such that

\[
\liminf_{t \to \infty} x_1(t) > \delta, \quad \liminf_{t \to \infty} S(t) > \delta \quad \text{and} \quad \liminf_{t \to \infty} I(t) > \delta,
\]

for any initial value.

Define

\[
X_0 = \{(x_1, S, I) \in \mathbb{R}^3 : x_1 > 0, S \geq 0, I > 0\}, \quad \partial X_0 = \mathbb{R}^3 \setminus X_0, \quad \Omega_0 = \bigcup_{z \in Y_0} \omega(z),
\]

\[
Y_0 = \{(x_1(0), S(0), I(0)) \in \partial X_0 : (x_1(t), S(t), I(t)) \in \partial X_0, \forall t \geq 0\},
\]

where \( \omega(z) \) is the omega limit set of solution \((x_1(t), S(t), I(t))\) with initial value \(z\).

By the Lemmas 2.1 and 2.2, Theorem 2.6, it is easy to see that

\[
\Omega_0 = \begin{cases} \{E_0, E_1, E_2, E_3\}, & \text{if } -\varepsilon \eta u_0/a_2 < v_0 \leq \eta_0, \\ \{E_0, E_1, E_2, E_3\}, & \text{if } 0 < v_0 < r_1/\eta \text{ and } R_0 \leq 1, \\ \{E_0, E_1, E_2, E_3, E_4\}, & \text{if } 0 < v_0 < r_1/\eta \text{ and } R_0 > 1, \\ \{E_0, E_1, E_2, E_4\}, & \text{if } v_0 \geq r_1/\eta > S + q_1 \text{ and } R_0 > 1. \end{cases}
\]

Furthermore, \( \Omega_0 \) is an acyclic isolated covering of itself for above any case. We have the following results.

Theorem 2.9. Suppose \(-\varepsilon \eta u_0/a_2 < v_0 < r_1/\eta \text{ and } R_0 > 1\) (cases (b) and (c) in Theorem 2.6). Then system (2) is uniformly persistent in \(X_0\), i.e. there is a \( \delta > 0 \) such that

\[
\liminf_{t \to \infty} x_1(t) > \delta, \quad \liminf_{t \to \infty} S(t) > \delta \quad \text{and} \quad \liminf_{t \to \infty} I(t) > \delta,
\]

for any solution \((x_1(t), S(t), I(t))\) of system (2) with initial value in \(X_0\).

Proof. Let \((x_1(t), S(t), I(t))\) be any solution of system (2) with initial value in \(X_0\). We first to show that \(x_1\) and \(I\) are uniformly persistent, that is \(\partial X_0\) is a strong repeller for \(X_0\) (see [38]). It is easy to see that \(\mathbb{R}^3\) and \(X_0\) are all positively invariant. Hence, \(\partial X_0\) is positively invariant and relatively closed in \(\mathbb{R}^3\). Since (2) is point dissipative (see Lemma 2.3) and \(\Omega_0\) is an acyclic isolated covering of itself. By the Theorem 4.6 in [38], \(\partial X_0\) is a strong repeller for \(X_0\) if it is a weak repeller. Therefore, in the following, we only need to prove \(x_1\) and \(I\) are weakly persistent.

We claim that \(\limsup_{t \to \infty} x_1(t) = 0\). Otherwise, suppose that \(\limsup_{t \to \infty} x_1(t) = \eta_0 > 0\). Then for any \(\xi > 0\), there is a \(T_1 > 0\) such that \(x_1(t) \leq \xi/\varepsilon \eta\) for all \(t \geq T_1\). By (3), we have \(x_2 \leq x_2(r_2 + \xi - a_2 x_2)\), for all \(t \geq T_1\). It follows from the comparison theorem that \(\limsup_{t \to \infty} x_2(t) \leq \xi/a_2\) if \(v_0 \leq 0\), and \(\limsup_{t \to \infty} x_2(t) \leq v_0 + \xi/a_2\) if \(v_0 > 0\). Since \(\xi\) is arbitrary, we have \(\limsup_{t \to \infty} x_2(t) = 0\) when \(v_0 \leq 0\), and \(\limsup_{t \to \infty} x_2(t) \leq v_0\) when \(v_0 > 0\). Hence, when \(-\varepsilon \eta u_0/a_2 < v_0 < 0\), for any \(0 < \zeta < r_1\) there is a \(T_2 > 0\) such that \(S(t) + q I(t) \leq x_2(t) \leq \zeta/\eta\) for all \(t \geq T_2\). By (2), it has \(x_1(t) \geq x_1(r_1 - \zeta - a_1 x_1)\), for all \(t \geq T_2\). By the comparison theorem and \(\zeta\) chosen arbitrary, we have \(\liminf_{t \to \infty} x_1(t) \geq u_0 > 0\), which is a contradiction. When \(0 < v_0 < r_1/\eta\), for any \(0 < \kappa < r_1 - \eta v_0\) there is a \(T_3 > 0\) such that \(S(t) + q I(t) \leq x_2(t) \leq v_0 + \kappa/\eta\) for all \(t \geq T_3\). By (2), it has \(x_1(t) \geq x_1(r_1 - \eta v_0 - \kappa - a_1 x_1)\), for all \(t \geq T_3\). Consequently, by \(\kappa\) chosen arbitrary,
we have $\liminf_{t \to \infty} x_1(t) \geq (r_1 - \eta v_0)/a_1 > 0$, which is a contradiction. Therefore, the $\limsup_{t \to \infty} x_1(t) > 0$.

Now we claim that $\limsup_{t \to \infty} I(t) > 0$. If the claim does not arise, we have $\limsup_{t \to \infty} I(t) = 0$. Then system (1) is the limiting equation of the first two equations of system (2). By (b) and (c) of Lemma 2.1, we obtain all solution paths of system (1) are attracted by (equations of system (2). By (b) and (c) of Lemma 2.1, we obtain all solution paths of system (1) are attracted by (equations of system (2).

By Lemma 2.5, the $\omega$-limit set of $(x_1(t), S(t), I(t))$ is $E_0(\tilde{x}_1, \tilde{x}_2, 0)$. That is $\lim_{t \to \infty} x_1(t) = \tilde{x}_1$ and $\lim_{t \to \infty} S(t) = \tilde{x}_2$. It follows from $R_1 > 1$ that $\beta > (1 - c_2)a_2$. Consequently, we can choose a positive constant $\gamma$ such that

$$pq\gamma(\tilde{x}_1 - \zeta) + \beta(\tilde{x}_2 - \zeta) - (d_2 + (1 - c_2)a_2(\tilde{x}_2 - \zeta) + \gamma + \mu) - (1 - c_2)a_2 \xi > \gamma,$$

For the $\zeta$ there is a $T_1 > 0$ such that $x_1(t) \geq \tilde{x}_1 - \zeta, S(t) \geq \tilde{x}_2 - \zeta$ and $I(t) \leq \zeta$, for all $t \geq T_1$. Combing the above inequations with the equation in (2), we have

$$\dot{I} \geq I(pq\gamma(\tilde{x}_1 - \zeta) + \beta(\tilde{x}_2 - \zeta) - (d_2 + (1 - c_2)a_2(\tilde{x}_2 - \zeta) + \gamma + \mu) - (1 - c_2)a_2 \xi) > \zeta I,$$

for all $t \geq T_0$. Consequently, $I(t) \to \infty$ as $t \to \infty$, which contradicts with $I(t) \to 0$ as $t \to \infty$. Therefore, $\limsup_{t \to \infty} I(t) > 0$.

Finally, we go to prove $S$ is uniformly persistent. Since $x_1$ and $I$ are uniformly persistent and $\limsup_{t \to \infty} x_2(t) \leq v_0 + \varepsilon u_0/\gamma$ (see Lemma 2.3), for any small positive constant $\delta$ there is $T_0$ such that $x_2(t) \leq v_0 + \varepsilon u_0/\gamma + \delta, x_1(t) \geq \delta$ and $I(t) \geq \delta$ for all $t \geq T_0$. By the $S$ equation in system (2), it has

$$\dot{S} \geq \gamma \delta - S((d_2 + (1 - c_2)a_2 + \beta)(v_0 + \varepsilon u_0/\gamma + \delta)) - \varepsilon \eta \delta).$$

Therefore, by the comparison theory, we have

$$\liminf_{t \to \infty} S(t) \geq \frac{\gamma \delta}{(d_2 + (1 - c_2)a_2 + \beta)(v_0 + \varepsilon u_0/\gamma + \delta) - \varepsilon \eta \delta} > 0.$$

This completes the proof. \square

**Theorem 2.10.** Suppose $v_0 \geq r_1/\eta > \tilde{S} + q\tilde{I}$ and $R_0 > 1$ (case (d) in Theorem 2.6). Then system (2) is uniformly persistent in $X_0$, i.e. there is a $\delta > 0$ such that

$$\liminf_{t \to \infty} x_1(t) > \delta, \quad \liminf_{t \to \infty} S(t) > \delta \quad and \quad \liminf_{t \to \infty} I(t) > \delta,$$

for any solution with initial value in $X_0$.

**Proof.** Let $(x_1(t), S(t), I(t))$ be any solution of system (2) with initial value in $X_0$. In the similar way as in the discussion of Theorem 2.9 one derives that $x_1$ and $I$ are uniformly persistent in $X_0$ when they are weakly persistent in $X_0$.

We claim that $\limsup_{t \to \infty} x_1(t) > 0$. Otherwise, suppose that $\limsup_{t \to \infty} x_1(t) = 0$. Then equation (4) is the limiting equation of $S$ and $I$ equations in system (2). By Lemma 2.2, (4) has a global asymptotical equilibrium point $(\tilde{S}, \tilde{I})$ in $((S, I) : S \geq 0, I > 0)$ when $R_0 > 1$. Therefore, by Lemma 2.5, we have $\lim_{t \to \infty} S(t) = \tilde{S}$ and $\lim_{t \to \infty} I(t) = \tilde{I}$. Then for any $\xi > 0$, there is a $T_1 > 0$ such that $S(t) \leq \tilde{S} + \xi/2$ and $I(t) \leq \tilde{I} + \xi/2q$ for all $t \geq T_1$. Consequently, the $x_1$ equation satisfies

$$\dot{x}_1 \geq x_1(r_1 - a_1x_1 - \eta(\tilde{S} + q\tilde{I} + \xi)).$$

(10)

Since $r_1/\eta > \tilde{S} + q\tilde{I}$, we can restrict $\xi$ small enough such that $r_1 - \eta(\tilde{S} + q\tilde{I} + \xi) > \xi$. Combine with (10), we have $\dot{x}_1 \geq x_1(\xi - a_1x_1).$ Hence, $\liminf_{t \to \infty} x_1(t) \geq \xi/a_1,$ which contradicts with $\limsup_{t \to \infty} x_1(t) = 0$. This proves the claim.
We now claim $\limsup_{t \to \infty} I(t) > 0$. If this claim does not arise, then $\limsup_{t \to \infty} I(t) = 0$. Consequently, equation (1) is the limiting equation of the first and second equations of system (2). It follows from (d) of Lemma 2.1 that the limiting equation (1) has a global asymptotical equilibrium point $(0, v_0)$. Therefore, by Lemma 2.5, we have $\lim_{t \to \infty} x_1(t) = 0$ and $\lim_{t \to \infty} S(t) = v_1$. Since $R_0 > 1$, we can choose a positive constant $\zeta$ such that

$$\beta (v_0 - \zeta) - (d_2 + (1 - c_2)a_2(v_0 - \zeta) + \gamma + \mu) - (1 - c_2)a_2\zeta > \zeta.$$ 

For the $\zeta$ there is a $T_2 > 0$ such that $S(t) \geq v_0 - \zeta$ and $I(t) \leq \zeta$ for all $t \geq T_2$. Hence, $I(t)$ satisfies

$$\dot{I} \geq I(\beta (v_0 - \zeta) - (d_2 + (1 - c_2)a_2(v_0 - \zeta) + \gamma + \mu) - (1 - c_2)a_2\zeta) > \zeta I,$$

for all $t \geq T_2$. Consequently, it has $\limsup_{t \to \infty} I(t) = +\infty$, which contradicts with $\limsup_{t \to \infty} I(t) = 0$. Therefore, by the Theorem 4.6 in [38], $\partial X_0$ is a strong repeller for $X_0$, i.e. there is a positive constant $\delta$ such that $\liminf_{t \to \infty} x_1(t) > \delta$ and $\limsup_{t \to \infty} I(t) > \delta$ for any initial value in $X_0$. In the same way as in the proof of the Theorem 2.9 one derives that $S(t)$ also is strong persistent with any initial value in $X_0$. This completes the proof.

**Remark 2.** Suppose $-\varepsilon \eta_0/a_2 < v_0 < r_1/\eta$. Then by the Lemma 2.1, the predator and prey species can coexist in the disease-free system (1). Further, from the cases (b) and (c) of Theorem 2.6 and Theorem 2.9, we can obtain a threshold value of whether a small number of infected predator species can invade into the disease-free predator-prey system. That is the disease invasion happens when $R_1 > 1$, and does not happen when $R_1 < 1$.

**Remark 3.** We consider whether a small number of prey species can invade into an SIS form prey-absent predator species system (4). Suppose $r_2 > 0$ and $R_0 > 1$, by the Lemma 2.2, the disease becomes to be an endemic in the prey-absent predator species system. Further, by the Theorems 2.6 and 2.10, a small number of prey species is (not) able to invade into prey-absent predator species system when $r_1/\eta > (\varepsilon)S + q\dot{I}$.

### 2.3. Biological interpretation.

Here, we will give the biological explanation for the threshold parameters, which are obtained from the local stability analysis of the equilibrium points. The parameters are divided into four cases:

(a) $v_0 \leq -\varepsilon \eta_0/a_2$. Here the $u_0$ stands for the carrying of the prey species. Hence, $\varepsilon \eta_0$ is the conversion rate of one predator when the prey species $x_1$ arrives at the carrying $u_0$. $r_2 + \varepsilon \eta_0$ is the growth rate of the predator species $x_2$. When $v_0 \leq -\varepsilon \eta_0/a_2$ (i.e. $r_2 + \varepsilon \eta_0 \leq 0$), it means that the population size of predator species will decrease in spite of the population of prey $x_1$ arrived at the environmental capacity. Therefore, $x_2$ will be extinct and $E_1(u_0, 0, 0)$ is locally stable.

(b) $-\varepsilon \eta_0/a_2 < v_0 < 0$. The predator species $x_2$ can survive with positive growth rate $r_2 + \varepsilon \eta_0$ even though the intrinsic growth rate $r_2$ is negative. Hence, $E_0(0, 0, 0)$ and $E_1(u_0, 0, 0)$ are unstable. The local stability of $E_3(\bar{x}_1, \bar{x}_2, 0)$ is determined by the basic reproduction number

$$R_1 = \frac{\beta \bar{x}_2}{d_2 + \gamma + \mu + (1 - c_2)a_2\bar{x}_2 - pq\varepsilon \eta \bar{x}_1}.$$ 

Here, $\beta \bar{x}_2$ is the infection rate of a new infective predator appearing in a totally susceptible predator population, and $1/(d_2 + \gamma + \mu + (1 - c_2)a_2\bar{x}_2 - pq\varepsilon \eta \bar{x}_1)$ is the
duration of infectivity of an infective predator, the product of which is the disease basic reproduction number at $E_3(\tilde{x}_1, \tilde{x}_2, 0)$. $R_1 < 1$ implies that the disease will be eliminated in the predator population with the initial value near the disease-free equilibrium point $E_3(\tilde{x}_1, \tilde{x}_2, 0)$, hence $E_3(\tilde{x}_1, \tilde{x}_2, 0)$ is locally stable; when $R_1 > 1$, the disease will be an endemic, the prey species, susceptible predator and infected predator can coexist, $E_3(\tilde{x}_1, \tilde{x}_2, 0)$ is unstable and system (2) is uniformly persistent.

(c) $0 < v_0 < r_1/\eta$. $v_0$ is the carrying of the predator species $x_2$ with the absence of prey $x_1$, $\eta$ is the predation rate of $x_2$. So $v_0\eta$ is the predation rate when susceptible predator population size is $v_0$. Since the hunting ability of infected predator is weaker than the healthy predator, the predation rate of predator species $x_2$ is not exceeding $v_0\eta$. Thus, the prey $x_1$ and predator $x_2$ can coexist, $E_0(0, 0, 0)$, $E_1(u_0, 0, 0)$ and $E_2(0, v_0, 0)$ are unstable for this case. The local stability of $E_3(\tilde{x}_1, \tilde{x}_2, 0)$ is the same with case (b), which also is determined by the basic reproduction number $R_1$.

(d) $v_0 \geq r_1/\eta$, which means the predation rate $v_0\eta$ is over the intrinsic growth rate $r_1$ of the prey. Hence, the prey species will be extinct if it is in the disease-free system for this case. Further, the predator species $x_2$ can survive even if the prey species $x_1$ is extinct. Hence, $E_0(0, 0, 0)$ and $E_1(u_0, 0, 0)$ are unstable. The local stability of $E_2(0, v_0, 0)$ is determined by the basic reproduction number

$$R_0 = \frac{\beta v_0}{d_2 + \gamma + \mu + (1 - c_2)a_2v_0}.$$  

Here, $\beta v_0$ is the infection rate of a new infected predator, and $d_2 + \gamma + \mu + (1 - c_2)a_2v_0$ is the removal rate of the infected predator. Consequently, $R_0$ is the disease basic reproduction number at $E_2(0, v_0, 0)$. $R_0 < 1$ implies that the disease will be wiped out from the predator population and $E_2(0, v_0, 0)$ is locally stable; when $R_0 > 1$, the disease will be an endemic and $E_2(0, v_0, 0)$ is unstable. Since the abilities of predation and conservation of the infected predator are weaker than the susceptible predator, there exists a threshold value $\bar{S} + q\bar{I}$ of the survival of prey species when $R_0 > 1$. $\bar{S}$ and $\bar{I}$ are the equilibrium states of the susceptible and infected predator in the prey-absent predator system (4), $\eta(\bar{S} + q\bar{I})$ is the predation rate of the predator species $x_2$ at $E_4(0, S, \bar{I})$. When $S + q\bar{I} > r_1/\eta$, means the predation rate $(S + q\bar{I})\eta$ is exceeding the intrinsic growth rate $r_1$ of $x_1$, thus, the prey species $x_1$ will be extinct and the equilibrium point $E_4(0, S, \bar{I})$ is locally stable. When $S + q\bar{I} < r_1/\eta$, it means the predation rate $(S + q\bar{I})\eta$ is less than the intrinsic growth rate $r_1$ of $x_1$, thus, the prey species $x_1$, susceptible predator species $S$ and infected predator species $I$ can coexist, $E_4$ is unstable and system (2) is uniformly persistent.

3. CTMC model. Let the discrete random vector $Y(t) = (x_1(t), S(t), I(t))$ be a CTMC, which takes values in a set $\mathbb{K} \subset \mathbb{N}_0^3$, with $\mathbb{N}_0$ the set of non-negative integers. For simplicity, the same notations for the random variables are used as in the deterministic model. The infinitesimal transition probabilities for the process $Y(t)$ are given by

$$\text{Prob}\{Y(t + \Delta t) = b|Y(t) = a\} = P(a, b)\Delta t + o(\Delta t),$$

where the transitions and their rates are summarized in Table 1. Because of the Markov assumption, the inter-event time of $Y(t)$ is exponentially distributed with a parameter $\sum_{i=1}^{7} P_i$. In the following, we will argue to compute the approximate probability of a disease outbreak and the approximate probability of prey species invasion based on the branching process theory (see Theorem 4.1 and Corollary 4.1 in [1]).
3.1. Probability of an outbreak. We consider the disease-free predator-prey system (1), in which the prey and predator species can coexist, i.e. assume $-\varepsilon \eta u_0/a_2 < v_0 < r_1/\eta$. By cases (b) and (c) of Lemma 2.1, all the positive solutions of the disease-free predator-prey system (1) go to $N_3(\tilde{x}_1, \tilde{x}_2)$. Hence, let $x_1 = \tilde{x}_1$ and $S = \tilde{x}_2$ be approximately constants and the events associated with $I(t)$ are independent of each other. We consider whether an infected predator can invade into the disease-free predator-prey system (1). In order to derive a stochastic threshold for disease outbreak, we apply the branching process theory to approximate the Markov chain process (listed in Table 1) near the disease-free equilibrium point $E_0(\tilde{x}_1, \tilde{x}_2, 0)$ with small initial number of infected predator. Given $I(0) = i$, the branching process has a limiting probability of extinction $P_0 = \lim_{t \to \infty} \text{Prob}\{I(t) = 0\}$, which can be estimated by the offspring probability-generating function (pgf) of $I$. Here, the offspring of the infected predator $I$ is the new infections. The value $1 - P_0$ is a good approximation for the probability of an outbreak, assuming that a small infected individuals is introduced.

In the continuous-time process, a birth is not accompanied by a death. Hence, the offspring pgf for infected predator $I$ given the $x_1(0) = \tilde{x}_1$, $S(0) = \tilde{x}_2$ and $I(0) = 1$ is $h(u) = E[u^I] = p_0 + p_2u^2$, where

$$p_0 = \frac{P_4 + P_5}{P_3 + P_4 + P_5} = \frac{(1 - c_2)a_2\tilde{x}_2 + d_2 + \mu + \gamma - pq\varepsilon\eta\tilde{x}_1}{(\beta + (1 - c_2)a_2)\tilde{x}_2 + d_2 + \mu + \gamma - pq\varepsilon\eta\tilde{x}_1}$$

is the probability of a death or recovery of an infected individual, and

$$p_2 = \frac{P_3}{P_3 + P_4 + P_5} = \frac{\beta\tilde{x}_2}{(\beta + (1 - c_2)a_2)\tilde{x}_2 + d_2 + \mu + \gamma - pq\varepsilon\eta\tilde{x}_1}$$

is the probability of a successful transmission of an infected individual.

The mean number of offspring per infected individual is given by $m_I = h'(1) = 2p_2$. By the theory of Galton-Watson branching process, if $m_I \leq 1$ (i.e. $R_1 \leq 1$) then $P_0 = \lim_{t \to \infty} \text{Prob}\{I(t) = 0\} = 1$ that means the probability of ultimate extinction is one. And if $m_I > 1$ (i.e. $R_1 > 1$), there is a unique fixed point $u^* = 1/R_1 \in (0, 1)$ of $h$ and such that $P_0 = \lim_{t \to \infty} \text{Prob}\{I(t) = 0\} = u^{*i}$, where $I(0) = i$. That is a positive probability $1 - 1/R_1$ that the disease can outbreak when $m_I > 1$. Hence, we have following result.

**Theorem 3.1.** Suppose $-\varepsilon \eta u_0/a_2 < v_0 < r_1/\eta$. If $R_1 \leq 1$, a small number of infected predator can not invade into the disease-free predator-prey system (1) in probability one; if $R_1 > 1$, the probability of an outbreak given $I(0) = i$ is approximately $1 - 1/R_1$.

### Table 1. State transitions and the infinitesimal probabilities for the CTMC epidemic model.

| Description | State transition $a \to b$ | Rate $P(a, b)$ |
|-------------|-----------------------------|----------------|
| 1 Birth of $S$ | $(x_1, S, I) \to (x_1, S + 1, I)$ | $P_1 = x_2(c_2a_2x_2)$ |
| 2 Death of $S$ | $(x_1, S, I) \to (x_1, S - 1, I)$ | $P_2 = S(d_2 + (1 - c_2)a_2x_2 - \varepsilon \eta x_1)$ |
| 3 Infection | $(x_1, S, I) \to (x_1, S - 1, I + 1)$ | $P_3 = \beta SI$ |
| 4 Death of $I$ | $(x_1, S, I) \to (x_1, S, I - 1)$ | $P_4 = I(d_2 + (1 - c_2)a_2x_2 + \mu - pq\varepsilon\eta x_1)$ |
| 5 Recover of $I$ | $(x_1, S, I) \to (x_1, S + 1, I - 1)$ | $P_5 = \gamma I$ |
| 6 Birth of $x_1$ | $(x_1, S, I) \to (x_1 + 1, S, I)$ | $P_6 = x_1(c_1a_1x_1)$ |
| 7 Death of $x_1$ | $(x_1, S, I) \to (x_1 - 1, S, I)$ | $P_7 = x_1(d_1 + (1 - c_1)a_1x_1 + \eta(S + qI))$ |
Remark 4. Suppose $-\varepsilon \eta u_0/a_2 < v_0 < r_1/\eta$ and $R_1 > 1$. The Remark 2 points out that a small number of infected predator species can invade into the disease-free predator-prey system (1). But, by the Theorem 3.1, a small number of infected predator species has a probability of successful invasion which is approximately $1 - 1/R_1^i$ with $I(0) = i$. This means, the sample paths of the CTMC process of infected population either hit zero with approximate probability $(1/R_1)^i$ or increase exponentially with approximate probability $1 - 1/R_1^i$. Therefore, the properties of CTMC model are totally different to the ODE model.

3.2. Probability of species invasion. Consider the prey-absent predator species system (4) with disease of SIS form transmission, in which the disease becomes an endemic. By (b) of Lemma 2.2, all positive solutions $(S(t), I(t))$ of system (4) go to the unique positive equilibrium point $(\bar{S}, \bar{I})$ when $r_2 > 0$ and $R_0 > 1$. Hence, we assume that $r_2 > 0$, $R_0 > 1$ and the predator species with disease has arrived at the equilibrium state, i.e., $S(t) = \bar{S}$ and $I(t) = \bar{I}$ for all $t > 0$. In the following, we will consider whether a small number of prey species can invade into the predator system. Assuming the invading species is $x_1$ with $x_1(0) = 1$, $S(0) = \bar{S}$ and $I(0) = \bar{I}$, consequently, by Table 1 the approximate pgf for the offspring of $x_1$ is

$$k(u) = \frac{P_6 u^2 + P_7}{P_6 + P_7} = \frac{(b_1 - c_1 a_1) u^2 + d_1 + (1-c_1) a_1 + \eta(\bar{S} + q \bar{I})}{b_1 + d_1 + (1 - 2c_1) a_1 + \eta(\bar{S} + q \bar{I})},$$

where $P_6$ and $P_7$ are defined by Table 1, $P_6/(P_6 + P_7)$ is the probability of a birth of a prey individual and $P_7/(P_6 + P_7)$ is the probability of a death of a prey individual. The mean number of offspring per predator individual is given by $m_p = k'(1) = 2 P_6/(P_6 + P_7)$. By the Galton-Watson branching process theory, if $m_p \leq 1$, i.e. $(r_1 - a_1)/\eta \leq \bar{S} + q \bar{I}$, then $P'_6 := \lim_{t \to \infty} \text{Prob}(x_1(t) = 0) = 1$, that is the prey species cannot invade by probability one. And if $m_p > 1$, i.e. $(r_1 - a_1)/\eta > \bar{S} + q \bar{I}$, there is a unique fixed point $u_* \in (0,1)$ of $k(u)$, such that $P'_6 = \lim_{t \to \infty} \text{Prob}(x_1(t) = 0) = u'_*,$ where $x_1(0) = i$. That is the probability of prey species invasion is approximately $1 - u'_*$ when $m_p > 1$. In addition, the fixed point is

$$u_* = \frac{d_1 + (1-c_1) a_1 + \eta(\bar{S} + q \bar{I})}{b_1 - c_1 a_1}.$$

Hence, we have the following result:

Theorem 3.2. Suppose $r_2 > 0$ and $R_0 > 1$. The prey species cannot invade into the predator system when $(r_1 - a_1)/\eta \leq \bar{S} + q \bar{I}$; the probability of an invasion given $x_1(0) = i$ is approximately $1 - u'_*$ when $(r_1 - a_1)/\eta > \bar{S} + q \bar{I}$.

Remark 5. These results also are different to the deterministic model. Suppose $r_2 > 0$ and $R_0 > 1$. In the CTMC model, when a small number of prey species are introduced, the prey species has a probability $P'_6$ to fail invasion if $(r_1 - a_1)/\eta > \bar{S} + q \bar{I}$. But it follows from the Remark 3 that the prey species can successfully invade into the predator system under the same condition for the ODE model.

Remark 6. By Theorems 2.10 and 3.2, (d) of Theorem 2.6, we find an interesting phenomenon. Suppose $v_0 > r_1/\eta$, $R_0 > 1$ and $(r_1 - a_1)/\eta \leq \bar{S} + q \bar{I} < r_1/\eta$. Then by (d) of the Theorem 2.6 and Theorem 2.10, we have that the prey-absent equilibrium point $E_0(0, \bar{S}, \bar{I})$ is unstable and the system (2) is uniformly persistent. That is, a small number of prey species can successfully invade into the prey-absent predator species system. But, under the above assumption, the Theorem 3.2 points out that the prey species will fail to invade into the prey-absent predator species system.
Table 2. Parameter Values for Cases 1, 2 and 3.

| Parameter          | Interpretation                        | Case 1  | Case 2  | Case 3  |
|--------------------|---------------------------------------|---------|---------|---------|
| $a_1$              | Density dependent of prey             | 0.0005  | 0.0005  | 0.0005  |
| $a_2$              | Density dependent of predator         | 0.0006  | 0.0006  | 0.0006  |
| $b_1$              | Intrinsic birth rate of prey          | 2       | 0.7795  | 2       |
| $b_2$              | Intrinsic birth rate of predator      | 1       | 1       | 1       |
| $c_1$              | Density dependence effects of prey    | 0.5     | 0.5     | 0.5     |
| $c_2$              | Density dependence effects of predator| 0.5     | 0.5     | 0.5     |
| $d_1$              | Natural mortality of prey             | 0.1     | 0.1     | 0.1     |
| $d_2$              | Natural mortality of predator         | 0.8     | 0.8     | 0.8     |
| $\eta$             | Predation rate of susceptible predator| 0.005   | 0.005   | 0.005   |
| $\varepsilon$      | Conversion rate of susceptible predator| 0.01   | 0.01   | 0.01   |
| $p\eta$            | Predation rate of infected predator   | 0.0005  | 0.0005  | 0.0005  |
| $q\varepsilon$     | Conversion rate of infected predator  | 0.002   | 0.002   | 0.001   |
| $\beta$            | Transmission                          | 0.01    | 0.01    | 0.01    |
| $\gamma$           | Recover rate                          | 0.01    | 0.01    | 0.01    |
| $\mu$              | Disease related mortality             | 0.02    | 0.02    | 0.02    |

with probability one for the CTMC model. Is there a contradiction? According to the biological significance, when $x_1(0) = 1$, $S(0) = \bar{S}$ and $I(0) = \bar{I}$, $r_1 - a_1$ is the increasing rate of prey species at $t = 0$, $\eta(\bar{S} + q\bar{I})$ is the sum of predation rate of all predator individuals at $t = 0$. By $(r_1 - a_1)/\eta < \bar{S} + q\bar{I}$, the small number of prey species is easy preyed by the predator species and will be extinct in a short period of time. Therefore, the CTMC model is more accordance with the biological phenomenon than the ODE model for this case. The numerical example will be illustrated by Case 2 in the following section.

4. Numerical simulations. In this section, we present three numerical examples of the ODE and CTMC models to illustrate the analytical results. The CTMC models are simulated by using the Stochastic Simulation Algorithm (see [13, 27]). Case 1 illustrates disease invasion, where the presence of an infected predator causes a change in a stable disease-free predator-prey system. Cases 2 and 3 illustrate prey species invasion, where a prey species invades into a stable predator species system with SIS form disease infection. Parameter values for the three cases are presented in Table 2, the existence and local stability results for the equilibria of ODE model (2) are summarized in Table 3.

Remark 7. Firstly, we consult the parameter values in the Table 2 in [2], and fix the parameter $a_i$, $b_i$, $c_i$, $d_i$, $\eta$, $\beta$, $\gamma$, $\mu$ ($i = 1, 2$) such that $R_0 > 1$. Then, we choose suitable $b_1$ and $\varepsilon$ such that $v_0 < r_1/\eta$ and $R_1 > 1$ (Cases 1 and 3) or $v_0 > r_1/\eta$ (Case 2).

4.1. Case 1: Probability of an outbreak. From Table 2, we have $v_0 \approx 333.3333 < r_1/\eta = 380$ and $R_1 \approx 3.7867 > 1$. By (c) of Lemma 2.1, there is a globally asymptotically stable positive equilibrium point $(x_1, x_2) = (255, 355)$ of the disease-free predator-prey system (1). However, by (c) of Theorem 2.6, the corresponding disease-free equilibrium point $E_3(255, 355, 0)$ is unstable in the model (2) (see Table 3), while the infected coexistence equilibrium point $E_5(2328, 94, 268)$ is locally
A COMPARISON OF DETERMINISTIC AND STOCHASTIC MODELS

Table 3. Equilibria in the form \((x_1, S, I)\) and their local stability for the ODE model (2) with parameters given in Table 2, \(U={\text{unstable}}, S={\text{stable}}\).

| Case 1     | Equilibria | Case 2     | Equilibria | Case 3     | Equilibria |
|------------|------------|------------|------------|------------|------------|
|            | \((0, 0, 0)\) | \((0, 0, 0)\) | \((0, 0, 0)\) | \((0, 0, 0)\) |
|            | \((3800, 0, 0)\) | \((1359, 0, 0)\) | \((3800, 0, 0)\) |
|            | \((0, 333, 0)\) | \((0, 333, 0)\) | \((0, 333, 0)\) |
|            | \((255, 355, 0)\) | \((0, 92.298, 217.629)\) | \((255, 355, 0)\) |
|            | \((0, 92, 218)\) | \((0.724, 92.298, 217.647)\) | \((0, 92, 218)\) |
|            | \((2327, 94, 268)\) | | \((2590, 94, 272)\) |

Table 4. Case 1: Probability of an outbreak \(1 - P_0\) computed from the theory of branching processes, and based on 5000 sample paths of the CTMC model for initial values of \(I(0) = 1\) and \(I(0) = 2\) at \(t = 50\).

| Cases | Initial value | \(1 - P_0\) | CTMC |
|-------|--------------|--------------|------|
| 1     | \(I(0) = 1\) | 0.7359       | 0.7440 |
|       | \(I(0) = 2\) | 0.9303       | 0.9288 |

stable and the system (2) is uniformly persistent. In the ODE model, the presence of the disease in predator species results in a new stable infected coexistence equilibrium with increased both population densities.

For the CTMC model, using the parameter values in Table 2 and applying the Theorem 3.1 to compute the approximate probability of an outbreak, \(1 - P_0 \approx 0.7359\) when \(I(0) = 1\), \(1 - P_0 \approx 0.9303\) when \(I(0) = 2\) (see Table 4). We verify the probability of an outbreak \(1 - P_0\) by computing the proportion of sample paths out of 5000 in which an outbreak occurs. That is, the CTMC model is simulated 5000 times over a given time period, \([0, t_{\text{end}}]\) \((t_{\text{end}} = 50)\) with the initial conditions \(x_1(0) = 255\), \(S(0) = 355\) and a small number of infected predator individuals. In each run of the model, the condition \(I(t_{\text{end}}) > 0\) is checked. The probability of an outbreak is computed as the fraction of simulations in which the number of infected individual of predator was above 0 at \(t_{\text{end}}\). The simulation results presented in Table 4 show good agreement with the predicted value of the probability of an outbreak.

Sample paths of the CTMC model are compared with the ODE solution in Fig. 2 for the initial condition \(x_1(0) = 255\), \(S(0) = 355\) and \(I(0) = 1\), i.e. we start at the disease-free equilibrium point and add one infected predator individual into the disease-free predator-prey system. In difference to the ODE solution, the CTMC sample paths have a probability \((P_0 = 0.2641)\) to choose disease extinction in a short time. Further, the tendency of success invasion paths of the CTMC model follows the ODE solution at the beginning, then it oscillates around the positive equilibrium after the ODE solution arrives at the equilibrium point. 5000 sample paths of CTMC model were run until \(t_{\text{end}} = 50\) with the initial values \(x_1(0) = 255\), \(S(0) = 355\) and small \(I(0)\). We study the distributions of an outbreak at \(t = 30, 40, 50\) by using some statistical methods. The distributions of an outbreak
Table 5. Case 1: Using the Kolmogorov-Smirnov test to compare the distributions of an outbreak sample paths of $x_1$, $S$ and $I$ at $t = 30, 40, 50$ based on 5000 sample paths, where $(n_i, m_j)$ means test whether the sample paths at $t = n$ with $I(0) = i$ and $t = m$ with $I(0) = j$ are from the same distribution.

|      | $(30, 40_1)$ | $(40, 50_1)$ | $(30_1, 30_2)$ | $(40_1, 40_2)$ | $(50_1, 50_2)$ |
|------|-------------|-------------|----------------|----------------|----------------|
| $p$  | 0.9571      | 0.8605      | 0.9115         | 0.81           | 0.7619         |
| $S$  | 0.3127      | 0.1434      | 0.9989         | 0.2852         | 0.9961         |
| $I$  | 0.3411      | 0.5824      | 0.5220         | 0.4625         | 1              |
| $D$  | 0.0118      | 0.014       | 0.0123         | 0.014          | 0.0147         |
| $I$  | 0.0223      | 0.0266      | 0.0083         | 0.0217         | 0.009          |

Table 6. Case 1: Using the Shapiro-Wilk normality test to verify whether an outbreak sample paths of $x_1$, $S$ and $I$ with initial value $I(0) = 1$ at time $t = 50$ follow the normal distribution.

|      | $x_1$ | $S$ | $I$ |
|------|-------|-----|-----|
| $W$  | 0.9996| 0.9974| 0.9995|
| p-value | 0.6331| 4.672e-06| 0.454|

Figure 2. Comparison of ODE and CTMC (3 sample paths) solutions for Case 1. We start the simulation at $x_1(0) = 255$, $S(0) = 355$, $I(0) = 1$.

at $t = 30, 40, 50$ with $I(0) = 1$ and $I(0) = 2$ are compared in pairs by the two sample Kolmogorov-Smirnov test (see Table 5). Since the p-values in Table 5 are all above 0.05, we can believe that the three moments with different initial values are following the same distributions. Therefore, the CTMC model has a quasi-stationary distribution with the initial values near the $E_3(255, 355, 0)$ (see Fig. 8 and Fig. 10 in Appendix B). This means, the initial value of $I(0)$ can affect the invasion probability, but after successful invasion the paths of the CTMC model oscillate around the equilibrium state of ODE model and have a quasi-stationary distribution. Furthermore, using the well-known Shapiro-Wilk normality test, we find that the sample paths for $x_1$ and $I$ follow the normal distributions (see Table 6 and Fig. 3).

4.2. Case 2: Prey species fail invasion with probability 1. In this case, we consider the prey species fails to invade into a predator species system with SIS form disease infection. Using the parameter values in Table 2, we have $v_0 \approx$
A COMPARISON OF DETERMINISTIC AND STOCHASTIC MODELS

Figure 3. Qqplot (see R statistical software) of an outbreak sample paths for $x_1$, $S$ and $I$ of Case 1 when $I(0) = 1$ based on 5000 sample paths at $t = 50$.

Figure 4. Solution of ODE model for Case 2. We start the simulation at $x_1(0) = 1, S(0) = 92, I(0) = 218$.

Figure 5. Comparison of ODE and CTMC (3 sample paths) solutions for Case 2. We start the simulation at $x_1(0) = 1, S(0) = 92, I(0) = 218$.

333.333, $r_1/\eta = 135.9$, $R_0 \approx 3.584 > 1$. By (b) of Lemma 2.2, there is a globally asymptotically stable equilibrium point $(\bar{S}, \bar{I})$ of system (4), where $\bar{S} \approx 92.298$ and $\bar{I} \approx 217.629$ (see Table 3). Then $v_0 > r_1/\eta > \bar{S} + q\bar{I} \approx 135.824$. However, by (d) of Theorem 2.6 and Theorem 2.10, the corresponding equilibrium point $E_4(0, \bar{S}, \bar{I})$ is unstable, while the positive equilibrium point $E_5(0.724, 92.298, 217.647)$ is locally stable and system (2) is uniformly persistent.

For the CTMC model, it follows from $(r_1 - a_1)/\eta = 135.8 < \bar{S} + q\bar{I} \approx 135.824$ and Theorem 3.2 that a small number of prey species fail to invade into the predator system (4) with probability one. Sample paths of the CTMC model are compared with the ODE model in Fig. 5 for the initial value $x_1(0) = 1, S(0) = 92, I(0) = 218$, i.e. we start at the disease-free equilibrium point and introduce a prey individual. The trajectory of the prey species costs a long time to arrive the equilibrium state. And the three species can coexist (see Fig. 4). But all three sample paths of prey species of the CTMC model go to zero in a short time, and all the sample paths of susceptible and infected predator species oscillate around the ODE solution.
Table 7. Case 3: Probability of prey species invasion \((1 - P'_0)\) computed from the theory of branching processes, and based on 5000 sample paths of the CTMC model for initial values of \(x_1(0) = 1\) and \(x_1(0) = 2\) at \(t = 50\).

| Cases | Initial value | \(1 - P'_0\) | CTMC   |
|-------|---------------|---------------|--------|
| 3     | \(x_1(0) = 1\) | 0.6647        | 0.6784 |
|       | \(x_1(0) = 2\) | 0.8876        | 0.8922 |

4.3. Case 3: Probability of prey species invasion. In this case, we also consider the prey species invasion. Using the parameter values in Table 2, we have \(R_0 \approx 3.5842 > 1\). Hence, by (b) of Lemma 2.2, the predator species system (4) has a global stable equilibrium point \((\bar{S}, \bar{I}) \approx (92, 218)\). Further, we have \(v_0 \approx 333.3333 < r_1/\eta = 380\) and \(R_1 \approx 3.7865 > 1\). By Theorem 2.6, the corresponding equilibrium point \(E_4(0, \bar{S}, \bar{I})\) of system (2) is unstable, meanwhile, there is a locally stable positive equilibrium point \(E_5(2590, 94, 272)\) (Table 3). Furthermore, from Theorem 2.9, system (2) is uniformly persistent, i.e. the prey species can invade into the prey-absent predator system for this case.

For the CTMC model, applying the parameter values in Table 2, we calculate that \(\bar{S} + q\bar{I} \approx 113.8 < (r_1 - a_1)/\eta = 379.9\). By Theorem 3.2, there is a probability \(1 - P'_0\) for the species successful invasion. That is \(1 - P'_0 \approx 0.6647\) when \(x_1(0) = 1\), \(1 - P'_0 \approx 0.8876\) when \(x_1(0) = 2\) (see Table 7). 5000 sample paths of CTMC model were run until \(t_{end} = 50\) with the initial values \(S(0) = 92\), \(I(0) = 218\) and small \(x_1(0)\). The probability of species invasion was computed as the fraction of simulations in which the number of prey individual was above 0 at \(t_{end}\). The simulation results presented in Table 7 show good agreement with the predicted value of the probability of invasion.

Sample paths of the CTMC model are compared with the ODE solution in Fig. 6 for the initial condition \(x_1(0) = 1\), \(S(0) = 92\) and \(I(0) = 218\), i.e. we start at the disease-free equilibrium point and introduce one prey individual into the predator system with disease. Difference from the ODE solution, the CTMC sample paths have a probability \((P'_0 = 0.3353)\) to choose prey species extinction in a short time. Further, the tendency of successful invasion paths of the CTMC model follows the ODE solution at the beginning, then it oscillates around the positive equilibrium after the ODE solution arrives at the equilibrium point. 5000 sample paths of CTMC model were run until \(t_{end} = 50\) with the initial values \(S(0) = 92\), \(I(0) = 218\) and small \(x_1(0)\). The distribution of a successful invasion at \(t = 30, 40, 50\) is studied by using some statistical methods in the following. The distributions of a successful invasion at \(t = 30, 40, 50\) with \(x_1(0) = 1\) and \(x_1(0) = 2\) are compared in pairs by the two sample Kolmogorov-Smirnov test (see Table 8). Since the \(p\)-values in Table 8 are all above 0.05, we can believe that the three moments with different initial values are following the same distribution. Therefore, the CTMC model has a quasi-stationary distribution with the initial values near the \(E_4(0, 92, 218)\) (see Fig. 9 and Fig. 11 in Appendix B). This means, the initial value of \(x_1(0)\) can affect the invasion probability, but after successful invasion the paths of CTMC model oscillate around the equilibrium state of ODE model and have a quasi-stationary distribution. Furthermore, using the well-known Shapiro-Wilk normality test, we
A COMPARISON OF DETERMINISTIC AND STOCHASTIC MODELS

Table 8. Case 3: Using the Kolmogorov-Smirnov test to compare the distributions of an outbreak sample paths of $x_1$, $S$ and $I$ at $t = 30, 40, 50$ based on 5000 sample paths, where $(n_i, m_j)$ means test whether the sample paths at $t = n$ with $x_1(0) = i$ and $t = m$ with $x_1(0) = j$ are from the same distribution.

| $p$ | $(30_1, 40_1)$ | $(40_1, 50_1)$ | $(30_1, 30_2)$ | $(40_1, 40_2)$ | $(50_1, 50_2)$ |
|-----|----------------|----------------|----------------|----------------|----------------|
| $x_1$ | 0.9481 | 0.7244 | 0.5647 | 0.8352 | 0.7135 |
| $S$ | 0.3127 | 0.1434 | 0.9989 | 0.2852 | 0.9961 |
| $I$ | 0.0684 | 0.6225 | 0.0022 | 0.6726 | 0.3696 |
| $D$ | $x_1$ | 0.0127 | 0.0168 | 0.0179 | 0.0141 | 0.0159 |
| $S$ | 0.0223 | 0.0266 | 0.0083 | 0.0217 | 0.009 |
| $I$ | 0.0315 | 0.0183 | 0.0421 | 0.0165 | 0.0209 |

Table 9. Case 3: Using the Shapiro-Wilk normality test to verify whether the sample paths of $x_1$, $S$ and $I$ with initial value $x_1(0) = 1$ at time $t = 50$ follow the normal distribution.

| | $x_1$ | $S$ | $I$ |
|-----|-----|-----|-----|
| $W$ | 0.9996 | 0.9972 | 0.9992 |
| p-value | 0.7315 | 6.957e-06 | 0.1244 |

Figure 6. Comparison of ODE and CTMC (3 sample paths) solutions for Case 3. We start the simulation at $x_1(0) = 1$, $S(0) = 92$, $I(0) = 218(P'_0 = 0.335)$.

Figure 7. Qqplot of successful invasion sample paths for $x_1$, $S$ and $I$ of Case 3 when $x_1(0) = 1$ based on 5000 sample paths at $t = 50$.

find that the sample paths for $x_1$ and $I$ follow the normal distributions (see Table 9 and Fig. 7).

Remark 8. The above examples show that system (2) is uniformly persistent under the parameter values in the Cases 1 and 3. It should be noted that the system (2)
has a globally asymptotically stable positive equilibrium point in the Cases 1 and 3 by the numerical simulation. However, the issues on whether and how varies values of the parameter may influence the behaviour of the system must be further considered, such as how stable is the behaviour against changes? Still qualitatively the same, or larger changes / bifurcations etc. taking place? These topics will be addressed in our future study.

5. Conclusions and future directions. In this paper, we mainly study the dynamics of deterministic and stochastic models for a predator-prey model with an SIS form of parasitic infection in the predator. For the deterministic model, the local stability of boundary equilibria and the uniform persistence results are obtained. Applying the Branching process theory, the approximate probabilities of the disease outbreak and prey species invasion are obtained for the CTMCs model. For the SDE model, we only build the model and use the numerical simulations to compare the dynamics with the ODE model. But the dynamic properties of the SDE model also are an interesting directions. Recently, many scholars used the Itô formula and stochastic analysis to study the dynamics of stochastic population models or epidemic models (see [6, 7, 17, 31, 32, 33, 34] and the references therein). Therefore, in our future works, we will study the existence of the global positive solution, stochastic permanence, existence of the stationary distribution of the SDE model by the tools of the stochastic analysis.

Appendix A. Proof of Lemma 2.2. We convert the system (4) into the following system:

\[
\begin{aligned}
\dot{x}_2 &= x_2(r_2 - a_2x_2) - \mu I, \\
\dot{I} &= \beta(x_2 - I)I - (d_2 + (1 - c_2)a_2x_2)I - \gamma I - \mu I.
\end{aligned}
\]  

(A.1)

System (A.1) has two disease-free equilibrium points \( N_0(0,0) \) which exists for all parameter values, and \( N_1(v_0,0) \) which exists for \( r_2 > 0 \). We assume that (A.1) has a positive equilibrium \( N_2(\bar{x}_2, \bar{I}) \), which is a positive solution of the following equations

\[
\begin{aligned}
\bar{x}_2(r_2 - a_2\bar{x}_2) - \mu \bar{I} &= 0, \\
\beta(\bar{x}_2 - \bar{I}) - (d_2 + (1 - c_2)a_2\bar{x}_2) - \gamma - \mu &= 0.
\end{aligned}
\]

(A.2)

It is the same discussion as the existence condition of \( E_4(0, \bar{S}, \bar{I}) \) in Section 2.2, we can obtain that \( N_2(\bar{x}_2, \bar{I}) \) exits when \( R_0 > 1 \), does not exit when \( R_0 \leq 1 \).

If \( r_2 \leq 0 \), \( \lim_{t \to \infty} x_2(t) = 0 \). Hence, the unique equilibrium \( N_0(0,0) \) is globally asymptotically stable. In the following, we consider the case \( r_2 > 0 \). From the first equation of (A.1), we have \( \dot{x}_2 \leq x_2(r_2 - a_2x_2) \). Hence, \( 0 \leq \liminf_{t \to \infty} x_2(t) \leq \limsup_{t \to \infty} x_2(t) \leq v_0 \). Furthermore, system (4) is positively invariant in the bounded set \( \Gamma = \{(S,I) : 0 \leq S + I \leq v_0\} \), which attracts every solution with initial conditions in \( \mathbb{R}^2_+ \). In the following, we study the dynamic property of system (4) in the bounded set \( \Gamma \setminus \{N_0\} \).

For the Case (a), i.e. \( R_0 \leq 1 \), let \( i = I/x_2 \). From system (A.1), we consider the following system

\[
\begin{aligned}
\dot{x}_2 &= x_2(r_2 - a_2x_2 - \mu i), \\
\dot{i} &= i(\beta x_2(1 - i) - (d_2 + \gamma + \mu(1 - i) + r_2 - c_2a_2x_2)).
\end{aligned}
\]

(A.3)

Define the Lyapunov function \( V(x_2, i) = i \). Then the time derivative of \( V \) along a solution of model (A.3) is
\[
\begin{align*}
\dot{V} \leq & (\beta v_0(1-i) - (d_2 + \gamma + \mu(1-i) + (1-c_2)a_2v_0)) \\
& \leq \beta v_0(1-i) \left( 1 - \frac{1}{R_0} \right). \tag{A.4}
\end{align*}
\]

It follows from (A.4) and \( R_0 \leq 1 \), that \( \dot{V} \leq 0 \). Furthermore, \( \dot{V} = 0 \) if and only if \( i = 0 \) (from the second inequality of (A.4)). Therefore, \( \{ i = 0 \} \) is the maximal invariant set in \( \{(x_2, i) : \dot{V} = 0\} \). By the LaSalle’s invariance principle, \( \lim_{t \to \infty} i(t) = 0 \). Therefore, the limiting equation of the first equation of system (A.3) is \( \dot{x}_2 = x_2(r_2 - a_2x_2) \), which has a globally asymptotically stable equilibrium \( x_2 = x_0 \). By Lemma 2.5, \( N_1(v_0, 0) \) is a globally asymptotically stable equilibrium of system (A.3). Consequently, the disease-free equilibrium point \((v_0, 0)\) of system (4) is globally asymptotically stable in \( \mathbb{R}^2_+ \setminus \{N_0\} \), if \( R_0 \leq 1 \).

For the Case (b), i.e. \( R_0 > 1 \), we separate the proof into three claims.

**Claim 1.** There exists a \( \delta > 0 \) such that \( \lim inf_{t \to \infty} I(t) > \delta \), for any solutions \((S(t), I(t))\) of system (4) with initial conditions \( S(0) \geq 0 \) and \( I(0) > 0 \).

We can easily know that system (A.1) has three equilibria, that is \( N_0(0, 0), N_1(v_0, 0) \) and positive equilibrium \( N_2(\bar{x}_2, \bar{I}) \). Let \( J(\bar{x}_2, \bar{I}) \) be the Jacobian matrix of system (A.1). Then

\[
J(N_0) = \begin{pmatrix} r_2 & -\mu \\ 0 & -(d_2 + \gamma + \mu) \end{pmatrix} \quad \text{and} \quad J(N_1) = \begin{pmatrix} -r_2 & -\mu \\ 0 & \beta v_0(-1 - \frac{1}{R_0}) \end{pmatrix}.
\]

Obviously, \( N_0(0, 0) \) is a saddle and always is unstable; when \( R_0 > 1 \), \( N_1(v_0, 0) \) also is a saddle and it is unstable. Consequently, \( N_0(0, 0) \) and \( N_1(v_0, 0) \) also are unstable equilibria in system (4). Define

\[
X_0 = \{(S, I) : S \geq 0, I > 0\}, \quad \partial X_0 = \mathbb{R}^2_+ \setminus X_0.
\]

It is easy to see that \( \mathbb{R}^2_+ \) and \( X_0 \) are all positively invariant. Clearly, \( \partial X_0 \) is positively invariant and relatively closed in \( \mathbb{R}^2_+ \). Assume that \( \Omega_0 = \bigcup_{z \in Y_0} \omega(z), \quad Y_0 = \{(S(0), I(0)) \in \partial X_0 : (S(t), I(t)) \in \partial X_0, \forall t \geq 0\} \), where \( \omega(z) \) is the omega limit set of solution \((S(t), I(t))\) with initial value \( z \). Then it is easy to see that \( \Omega_0 = \{N_0, N_1\} \).

Let \((S(t), I(t))\) be any solution of system (4) with initial value in \( X_0 \). We claim \( \lim \sup_{t \to \infty} I(t) > 0 \). Otherwise, we have \( \lim \sup_{t \to \infty} I(t) = 0 \). Then the \( S \) component of system (4) has a limiting equation \( \dot{S} = S(r_2 - a_2S) \), whose solution paths with positive initial values go to \( v_0 \). By Lemma 2.5 and \( \dot{S}(t) = (\beta_2 - c_2a_2I(t)) + \gamma I(t) > 0 \) when \( S(t) = 0 \) and \( I(t) > 0 \), we have \( \lim_{t \to \infty} S(t) = v_0 \). Then for any \( \xi > 0 \) there is a \( T > 0 \) such that \( I(t) < \xi \) and \( S(t) > v_0 - \xi \) for all \( t \geq T \). Since \( R_0 > 1 \), we can choose \( \xi \) small enough to make the following inequality holds

\[
(\beta - (1-c_2)a)(v_0 - \xi) - (d_2 + \gamma + \mu) - (1-c_2)a_2 \xi > \xi.
\]

By the \( I \) component equation of system (4), yields

\[
\dot{I} = I(\beta S - (d_2 + \gamma + \mu + (1-c_2)a_2S) - (1-c_2)aI) \\
\geq I((\beta - (1-c_2)a)(v_0 - \xi) - (d_2 + \gamma + \mu) - (1-c_2)a_2 \xi) \\
> \xi I, \quad \text{for all} \ t \geq T.
\]

Hence, \( \lim_{t \to \infty} I(t) = \infty \), which is a contradiction. Therefore, \( \lim \sup_{t \to \infty} I(t) > 0 \).
It is obvious that the $S$-axis is invariant. Furthermore, any solution $(S(t),0)$ of system (4) approaches to $N_1(v_0,0)$ as $t \to \infty$ when $S(0) > 0$. Therefore, the set \{\$N_0, N_1$\} is an acyclic isolated covering of $\Omega_0$. By Theorem 4.6 in [38], the weak persistent of $I(t)$ can conclude the uniformly persistent of $I(t)$. This completes the proof of Claim 1.

**Claim 2.** There is no limit cycle in $\Gamma$ of system (4).

Let

$$P(S,I) = x_2(b_2 - c_2a_2x_2) - S(d_2 + (1 - c_2)a_2x_2) - \beta SI + \gamma I,$$

and

$$Q(S,I) = \beta SI - I(d_2 + (1 - c_2)a_2x_2) - \gamma I - \mu I,$$

where $x_2 = S + I$. We construct a Dulac function as $B(S,I) = 1/SI$. Then we obtain

$$\frac{\partial(BP)}{\partial S} = -\frac{1}{S^2I} \left( x_2(b_2 - c_2a_2x_2) - S(d_2 + (1 - c_2)a_2x_2) - \beta SI + \gamma I \right)$$

$$+ \frac{1}{SI} \left( b_2 - c_2a_2x_2 - c_2a_2x_2 - (d_2 + (1 - c_2)a_2x_2) - (1 - c_2)a_2S - \beta I \right)$$

$$= \frac{1}{SI} \left( -(b_2 + \gamma)\frac{I}{S} + c_2a_2\frac{I^2}{S} - a_2S \right)$$

and

$$\frac{\partial(BQ)}{\partial I} = -\frac{1}{SI^2} \left( \beta SI - I(d_2 + (1 - c_2)a_2x_2) - \gamma I - \mu I \right)$$

$$+ \frac{1}{SI} \left( \beta S - (d_2 + (1 - c_2)a_2x_2 + \gamma + \mu) - (1 - c_2)a_2I \right)$$

$$= \frac{1}{SI} \left( -(1 - c_2)a_2I \right).$$

Hence

$$\frac{\partial(BP)}{\partial S} + \frac{\partial(BQ)}{\partial I} = \frac{1}{SI^2} \left( -(b_2 + \gamma)I + c_2a_2I^2 - a_2S^2 - (1 - c_2)a_2SI \right).$$

Let $h(I) = -(b_2 + \gamma)I + c_2a_2I^2$. Obviously, $h(I) < 0$ for all $I \in (0, (b_2 + \gamma)/c_2a_2)$. Since $(b_2 + \gamma)/c_2a_2 = v_0/c_2 + (d_2 + \gamma)/c_2a_2 > v_0$, we have $h(I) < 0$ for all $I \in (0, v_0]$. Furthermore,

$$\frac{\partial(BP)}{\partial S} + \frac{\partial(BQ)}{\partial I} < 0, \text{ for all } S, I \in (0, v_0].$$

Therefore, system (4) has no limit cycle in $(0, v_0] \times (0, v_0]$.

**Claim 3.** The positive equilibrium point $(\bar{S}, \bar{I})$ of system (4) is global asymptotical stable.

By Claim 2, we only need to prove the positive equilibrium point $(\bar{S}, \bar{I})$ is locally stable. We suppose $(\bar{S}, \bar{I})$ is unstable. Since $\bar{S}(t) = I(t)(b_2 - c_2a_2I(t)) + \gamma I(t) > 0$ with $\bar{S}(0) = 0$ and $I(t) > 0$, the paths with initial value $S(0) = 0$ and $I(0) > 0$ go to the inner of $X$. Combine with Claim 1 and the well known Poincaré-Bendixson Theorem, there exists a limit cycle in $(0, v_0] \times (\delta, v_0] \cap \Gamma$, which contradicts with Claim 2. Therefore, $(\bar{S}, \bar{I})$ is locally stable. This completes the proof.
Figure 8. Case 1: Boxplot of an outbreak sample paths for prey species $x_1$, susceptible predator species $S$ and infected predator species $I$ with initial value $I(0) = 1$ (first line) and $I(0) = 2$ (second line) based on 5000 sample paths at time $t = 30, 40, 50$.

Figure 9. Case 3: Boxplot of successful invasion sample paths for prey species $x_1$, susceptible predator species $S$ and infected predator species $I$ with initial value $x_1(0) = 1$ (first line) and $x_2(0) = 2$ (second line) based on 5000 sample paths at time $t = 30, 40, 50$.

Appendix B. Figures in Cases 1 and 3.

Acknowledgments. The authors would like to thank the anonymous reviewers for their insightful comments that have helped improve the quality of the paper. The work is supported by the National Natural Science Foundation of China under grants 11401382, 11501518 and 11461073.
Figure 10. Case 1: Histogram of the probability density function for an outbreak sample paths for prey species $x_1$ (first and second lines), susceptible predator species $S$ (third and fourth lines) and infected predator species $I$ (fifth and sixth lines) of Case 1 when $I(0) = 1$ (odd number lines) and when $I(0) = 2$ (even number lines) based on 5000 sample paths at $t = 30$ (left column), $t = 40$ (middle column), $t = 50$ (right column).
Figure 11. Case 3: Histogram of the probability density function for successful invasion sample paths for prey species $x_1$ (first and second lines), susceptible predator species $S$ (third and fourth lines) and infected predator species $I$ (fifth and sixth lines) of Case 3 when $x_1(0) = 1$ (odd number lines) and when $x_1(0) = 2$ (even number lines) based on 5000 sample paths at $t = 30$ (left column), $t = 40$ (middle column), $t = 50$ (right column).
REFERENCES

[1] L. J. S. Allen, An Introduction to Stochastic Processes with Applications to Biology, 2nd edition, CRC Press, Boca Raton, FL, 2011.
[2] L. J. S. Allen and V. A. Bokil, Stochastic models for competing species with a shared pathogen, *Math. Biosci. Eng.*, 9 (2012), 461–485.
[3] L. J. S. Allen and N. Kirupaharan, Asymptotic dynamics of deterministic and stochastic epidemic models with multiple pathogens, *Int. J. Numer. Anal. Modeling*, 2 (2005), 329–344.
[4] L. J. S. Allen and G. E. Lahodny, Extinction thresholds in deterministic and stochastic epidemic models, *J. Biol. Dyn.*, 6 (2012), 590–611.
[5] R. M. Anderson and R. M. May, The invasion, persistence, and spread of infectious diseases within animal and plant communities, *Phil. Trans. R. Soc. London B*, 314 (1986), 533–570.
[6] Y. L. Cai, Y. Cai, M. Banerjee and W. M. Wang, A stochastic sirs epidemic model with infectious force under intervention strategies, *J. Differential Equations*, 259 (2015), 7463–7502.
[7] Y. L. Cai, Y. Kang and W. M. Wang, A stochastic sirs epidemic model with nonlinear incidence rate, *Appl. Math. Comput.*, 305 (2017), 221–240.
[8] J. Chattopadhyay and O. Arino, A predator-prey model with disease in the prey, *Nonlinear Anal.*, 36 (1999), 747–766.
[9] K. P. Das, A study of chaotic dynamics and its possible control in a predator-prey model with disease in the predator, *J. Dyn. Control Syst.*, 21 (2015), 605–624.
[10] K. P. Das, A study of harvesting in a predator-prey model with disease in both populations, *Math. Methods Appl. Sci.*, 39 (2016), 2853–2870.
[11] K. S. Dorman, J. S. Sinsheimer and K. Lange, In the garden of branching processes, *SIAM Rev.*, 46 (2004), 202–229.
[12] R. Durrett, Special invited paper: Coexistence in stochastic spatial models, *Ann. Appl. Probab.*, 19 (2009), 477–496.
[13] D. T. Gillespie, *Markov Processes: An Introduction for Physical Scientists*, Academic Press, Inc., Boston, MA, 1992.
[14] B. S. Goh, *Management and Analysis of Biological Populations*, Elsevier Sci. Pub. Com., Amsterdam, 1980.
[15] B. S. Goh, Global stability in two species interactions, *J. Math. Biol.*, 3 (1976), 313–318.
[16] F. M. D. Gulland, The impact of infectious diseases on wild animal populations—a review, *Ecology of Infectious Diseases in Natural Populations*. (B. T. Grenfell and A. P. Dobson, eds). Cambridge: Cambridge University Press, 1995, 20–51.
[17] W. J. Guo, Y. L. Cai, Q. M. Zhang and W. M. Wang, Stochastic persistence and stationary distribution in an sis epidemic model with media coverage, *Physica A: Statistical Mechanics and its Applications*, 492 (2018), 2220–2236.
[18] P. Haccou, P. Jagers and V. A. Vatutin, *Branching Processes Variation, Growth, and Extinction of Populations*, Cambridge University Press, Cambridge; IIAAS, Laxenburg, 2007.
[19] K. P. Hadeler and H. I. Freedman, Predator-prey populations with parasitic infection, *J. Math. Biol.*, 27 (1989), 609–631.
[20] J. K. Hale and P. Waltman, Persistence in infinite-dimensional systems, *SIAM J. Math. Anal.*, 20 (1989), 388–395.
[21] L. T. Han and Z. E. Ma, Four predator prey models with infectious diseases, *Math. Comput. Modelling*, 34 (2001), 849–858.
[22] L. T. Han, Z. E. Ma and T. Shi, An sirs epidemic model of two competitive species, *Math. Comput. Modelling*, 37 (2003), 87–108.
[23] L. T. Han and A. Pugliese, Epidemics in two competing species, *Nonlinear Anal.*, 10 (2009), 723–744.
[24] M. Haque, A predator-prey model with disease in the predator species only, *Nonlinear Anal.: Real World Appl.*, 11 (2010), 2224–2236.
[25] M. Haque and E. Venturino, An ecoepidemiological model with disease in predator: The ratio-dependent case, *Math. Meth. Appl. Sci.*, 30 (2007), 1791–1809.
[26] H. W. Hethcote, W. D. Wang, L. T. Han and Z. E. Ma, A predator-prey model with infected prey, *Theor. Popul. Biol.*, 66 (2004), 259–268.
[27] D. J. Higham, Modeling and simulating chemical reactions, *SIAM Rev.*, 50 (2008), 347–368.
[28] M. Kimmel and D. Axelrod, *Branching Processes in Biology*, Springer-Verlag, New York, 2002.
[29] N. Lanchier and C. Neuhauser, A spatially explicit model for competition among specialists and generalists in a heterogeneous environment, *Ann. Appl. Probab.*, 16 (2006), 1385–1410.

[30] N. Lanchier and C. Neuhauser, Stochastic spatial models of host-pathogen and host-mutualist interactions, i, *Ann. Appl. Probab.*, 16 (2006), 448–474.

[31] Q. Liu, D. Q. Jiang, N. Z. Shi, T. Hayat and A. Alsaeedi, The threshold of a stochastic sis epidemic model with imperfect vaccination, *Math. Comput. Simulation*, 144 (2018), 78–90.

[32] M. Liu, C. Bai and Y. Jin, Population dynamical behavior of a two-predator one-prey stochastic model with time delay, *Discrete Contin. Dyn. Syst.*, 37 (2017), 2513–2538.

[33] M. Liu, X. He and J. Yu, Dynamics of a stochastic regime-switching predator-prey model with harvesting and distributed delays, *Nonlinear Anal. Hybrid Syst.*, 28 (2018), 87–104.

[34] M. Liu and M. Fan, Stability in distribution of a three-species stochastic cascade predator-prey system with time delays, *IMA J. Appl. Math.*, 82 (2017), 396–423.

[35] R. K. McCormack and L. J. S. Allen, Disease emergence in multi-host epidemic models, *Math. Med. Biol.*, 24 (2007), 17–34.

[36] S. Sarwardi, M. Haque and E. Venturino, Global stability and persistence in ëg-holling type ii diseased predator ecosystems, *J. Biol. Phys.*, 37 (2011), 91–106.

[37] H. R. Thieme, Covergence results and a Poincaré-Bendixson trichotomy for asymptotically autonomous differential equations, *J. Math. Biol.*, 30 (1992), 755–763.

[38] H. R. Thieme, Persistence under relaxed point-dissipativity (with application to an endemic model), *SIAM J. Math. Anal.*, 24 (1993), 407–435.

[39] E. Venturino, The influence of diseases on lotka-volterra systems, *Rocky Mountain J. Math.*, 24 (1994), 381–402.

[40] E. Venturino, Epidemics in predator-prey models: disease in the predators, *IMA J. Math. Appl. Med. Biol.*, 19 (2002), 185–205.

[41] P. Whittle, The outcome of a stochastic epidemic: A note on bailey’s paper, *Biometrika*, 42 (1955), 116–122.

[42] Y. N. Xiao and L. S. Chen, Modeling and analysis of a predator-prey model with disease in the prey, *Math. Biosci.*, 171 (2001), 59–82.

[43] R. Xu and S. H. Zhang, Modelling and analysis of a delayed predator-prey model with disease in the predator, *Appl. Math. Comput.*, 224 (2013), 372–386.

[44] Y. Yuan and L. J. S. Allen, Stochastic models for virus and immune system dynamics, *Math. Biosci.*, 234 (2011), 84–94.

Received July 2017; 1st revision December 2017; final revision June 2018.

E-mail address: hhxiao1@126.com
E-mail address: xlg132@126.com
E-mail address: wangkaimath@sina.com