



Available online at <http://scik.org>

Commun. Math. Biol. Neurosci. 2016, 2016:12

ISSN: 2052-2541

## POPULATION AND EVOLUTIONARY ADAPTIVE DYNAMICS OF A STOCHASTIC PREDATOR-PREY MODEL

TAO FENG<sup>1</sup>, XINZHU MENG<sup>1,2,\*</sup>

<sup>1</sup>College of Mathematics and Systems Science, Shandong University of Science and Technology,  
Qingdao 266590, PR China

<sup>2</sup>State Key Laboratory of Mining Disaster Prevention and Control Co-founded by Shandong Province and the  
Ministry of Science and Technology, Shandong University of Science and Technology,  
Qingdao 266590, PR China

Copyright © 2016 Tao Feng and Xinzhu Meng. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

**Abstract.** This paper intends to develop a theoretical framework for investigating the evolutionary adaptive dynamics of a stochastic differential system. The key to the question is how to build an evolutionary fitness function. Firstly, we propose a stochastic predator-prey model with disease in the prey and discuss the asymptotic behavior around the positive equilibrium of its deterministic equation. Secondly, by using stochastic population dynamics and adaptive dynamics methods, we propose a fitness function based on stochastic impact and investigate the conditions for evolutionary branching and the evolution of pathogen strains in the infective prey. Our results show that (1) large stochastic impact can lead to rapidly stable evolution towards smaller toxicity of pathogen strains, which implies that stochastic disturbance is beneficial to epidemic control; (2) stochastic disturbance can go against evolutionary branching and promote evolutionary stability. Finally, we carry on the evolutionary analysis and make some numerical simulations to illustrate our main results. The developed methodologies could potentially be used to investigate the evolutionary adaptive dynamics of the stochastic differential systems.

**Keywords:** stochastic predator-prey model; evolution branching; stationary distribution; disease in the prey; population dynamics.

**2010 AMS Subject Classification:** 34K50, 92D15.

---

\*Corresponding author

E-mail address: [mxz721106@sdust.edu.cn](mailto:mxz721106@sdust.edu.cn)

Received May 3, 2016

## 1. Introduction

After the pioneering work of Haderler and Freedman [1] on three species eco-epidemiological systems, namely, sound prey (susceptible), infected prey (infective) and predator have been studied extensively by researchers. The three species eco-epidemiological system describes a predator-prey model where the prey is infected, and the predator preys on only the infected prey since infected prey may be weaker or less active. There are so many references in this context, we have just cited here some of them (e.g. see, [2-8] and the references therein). The main works include Hopf bifurcation [2-3], the stability of equilibrium points [4-6], extinction and permanence [7], and so on.

Recently, the evolution of predator-prey community has received much attention from scientists (e.g. see, [9-13] and the references therein). The adaptive dynamics method provides a useful tool to investigate the long-term evolutionary outcomes of a small mutation in the traits expressing the phenotypes. A classical method of building the evolutionary fitness function is based on a globally asymptotically stable positive equilibrium point of the autonomous differential system (e.g. see, [9-12]). Moreover, many researchers are interested in the adaptive evolution based on nonequilibrium population dynamics. Meng et al. [13] have constructed an invasion fitness function, which involves the average growth rate and settles in a nonequilibrium attractor of non-autonomous differential system.

The species in the natural world are often affected by environmental noises. For better understanding the dynamic behaviors of the predator-prey system, many authors formulated some stochastic systems of the predator-prey models and discussed the effects of the environmental disturbance on the dynamic behaviors of the stochastic models (e.g. see, [14-20] and the references therein). Zhang et al. [15] introduced a stochastic predator-prey system with infected prey. They studied the stationary distribution and the ergodicity of the predator-prey model. Recently, Dieckmann and Law [21] introduced environmental stochastic impacts on the evolutionary process. But little attention has been paid so far to merge these two important areas of research. Taking all above mentioned into account, the research on evolutionary dynamics for stochastic differential systems is very interesting. However, to the best of our knowledge, the research on evolutionary analysis of a stochastic predator-prey system is not too much yet.

Therefore, according to a deterministic predator-prey system, this paper proposes a stochastic predator-prey evolutionary model with infected prey, to explore the influence of stochastic disturbance on the evolution of toxicity of pathogen strains.

Our main goal of this paper is to build an evolutionary fitness function which is based on stochastic differential system and investigates the adaptive dynamics of the proposed model. To this end, the rest of the paper is organized as follows. In the next section, we first propose a stochastic predator-prey system with infected prey, then we study the global asymptotic behavior around the equilibrium of the deterministic predator-prey system. In Section 3, we build a fitness function to study the evolutionary dynamics of a single state. In the last section, we summarize our main results and focus our discussion on biological meaning.

## 2. Model and demographic properties

### 2.1. Population dynamics

We first consider a deterministic predator-prey system [2, 5], which reads

$$\begin{cases} \frac{dS}{dt} = rS(t) \left(1 - \frac{S(t)}{K}\right) - \beta S(t)I(t), \\ \frac{dI}{dt} = \beta S(t)I(t) - cI(t) - pI(t)Y(t) - aI^2(t), \\ \frac{dY}{dt} = kpI(t)Y(t) - dY(t), \end{cases} \quad (2.1)$$

where  $S(t)$  and  $I(t)$ , respectively, stand for the density of susceptible prey and infected prey at time  $t$ ,  $Y(t)$  represents the population density of predator at time  $t$ .  $K$  represents the environmental maximum capacity,  $\beta$  is the infection rate from susceptible prey  $S(t)$  to infected prey  $I(t)$ ,  $k(0 \leq k \leq 1)$  is the conversion rate from prey to a predator,  $r$  is the intrinsic growth rate of  $S(t)$ ,  $p$  is the probability that infected prey is attacked by predator,  $a$  is the intraspecific competition coefficient of  $I(t)$ ,  $c$  is the diseased death rate of  $I(t)$ ,  $d$  represents the natural death rate of predator.

Easy, if

$$R = \frac{kp}{d} \frac{r - \frac{cr}{\beta K}}{\beta + \frac{a}{\beta K}} > 1,$$

system (2.1) has a positive equilibrium  $(S^*, I^*, Y^*)$  with

$$S^* = \frac{K(rkp - \beta d)}{rkp}, I^* = \frac{d}{kp}, Y^* = \frac{\beta K(rkp - \beta d) - rkpc - rda}{rkp^2}.$$

In the real world, biological populations are often disturbed by environmental interference. Therefore, it is important to investigate the effects of environmental noises on the properties of the systems. In this paper, we firstly study the following stochastic population dynamics system

$$\begin{cases} dS = \left[ rS(t) \left( 1 - \frac{S(t)}{K} \right) - \beta S(t)I(t) \right] dt + \sigma_1 S(t) dB_1(t), \\ dI = \left[ \beta S(t)I(t) - cI(t) - pI(t)Y(t) - aI^2(t) \right] dt + \sigma_2 I(t) dB_2(t), \\ dY = \left[ kpI(t)Y(t) - dY(t) \right] dt + \sigma_3 Y(t) dB_3(t), \end{cases} \quad (2.2)$$

where  $B_i(t) (i = 1, 2, 3)$  is independent Brownian motions with intensity  $\sigma_i^2 (i = 1, 2, 3)$ .

Throughout this paper, let  $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathcal{P})$  be a complete probability space with a filtration  $\{\mathcal{F}_t\}_{t \geq 0}$  satisfying the usual conditions (i.e. it is increasing and right continuous while  $\mathcal{F}_0$  contains all  $\mathcal{P}$ -null sets. Function  $B_i(t) (i = 1, 2, 3)$  is a Brownian motion defined on the complete probability space  $\Omega$ . For an integrable function  $f$  on  $[0, +\infty)$ , we define  $\langle f(t) \rangle = t^{-1} \int_0^t f(s) ds$ .

## 2.2. Asymptotic behavior around the equilibrium $E = (S^*, I^*, Y^*)$ of system (2.1)

In this subsection, we consider the asymptotic behavior around the equilibrium  $E = (S^*, I^*, Y^*)$  of system (2.1).

$X(t)$  is a temporally homogeneous Markov process in  $E_l$ , which is given by the stochastic differential equation

$$dX(t) = b(X)dt + \sum_{m=1}^k \sigma_m(x) dB_m(t),$$

where  $E_l \subset R^l$  represents a  $l$ -dimensional Euclidean space.

The diffusion matrix of  $X(t)$  is given by

$$\Lambda(x) = (a_{i,j}(x)), a_{i,j}(x) = \sum_{m=1}^k \sigma_m^i(x) \sigma_m^j(x).$$

**Assumption 2.1.** [22] Assume that there is a bounded domain  $U \subset E_l$  with regular boundary, satisfying the following conditions:

- (1) In the domain  $U$  and some of its neighbors, the minimum eigenvalue of the diffusion matrix  $A(x)$  is nonezero.
- (2) When  $x \in E_l \setminus U$ , the mean time  $\tau$  at which a path starting from  $x$  to the set  $U$  is limited, and  $\sup_{x \in H} E_x \tau < \infty$  for every compact subset  $H \subset E_l$ .

**Lemma 2.1.** [22] When Assumption 2.1 holds, the Markov process  $X(t)$  has a stationary distribution  $\mu(\cdot)$  with density in  $E_l$ . Let  $f(x)$  be a function integrable with respect to the measure  $\mu$ , where  $x \in E_l$ , then for any Borel set  $B \subset E_l$ , we have

$$\lim_{t \rightarrow \infty} P(t, x, B) = \mu(B),$$

and

$$P_x \left\{ \lim_{T \rightarrow \infty} \frac{1}{T} \int_0^T f(x(t)) dt = \int_{E_l} f(x) \mu(dx) \right\} = 1.$$

**Theorem 2.1.** Let  $(S(t), I(t), Y(t))$  be the solution of model (2.2). If  $R > 1$ ,  $\sigma_i > 0$ ,  $1 \leq i \leq 3$  and  $0 < \delta < \min\{m_1 S^{*2}, m_2 I^{*2}, m_3 Y^{*2}\}$ , then model (2.2) has a unique stationary distribution  $\mu$  and it is ergodic. Here  $E = (S^*, I^*, Y^*)$  is the positive equilibrium of system (1),  $(S(0), I(0), Y(0)) \in R_+^3$  is the initial value,

$$\eta = \frac{2K}{r} \left( r - \frac{r}{K} S^* + \frac{r}{K} I^* + \frac{1}{k} \frac{r}{K} Y^* + \frac{(r - \frac{2r}{K} S^* - c - 2aI^*)^2}{2c} + \frac{(r - \frac{2r}{K} S^* - d)^2}{2d} \right),$$

$$\varpi = a\eta + aI^* + \frac{c}{2} - aS^* - \frac{a}{k} Y^*,$$

$$m_1 = \frac{r\eta}{2K} - \sigma_1^2, m_2 = \varpi - \sigma_2^2, m_3 = \frac{1}{2k^2} (d - 2\sigma_3^2)$$

and

$$\delta = \sigma_1^2 S^{*2} + \sigma_2^2 I^{*2} + \frac{1}{k^2} \sigma_3^2 Y^{*2} + \frac{(c + 2aI^* + d) \sigma_3^2 Y^*}{2k^2 p} + \frac{\eta}{2} (\sigma_1^2 S^* + \sigma_2^2 I^* + \frac{1}{k} \sigma_3^2 Y^*).$$

Also we have

$$\limsup_{t \rightarrow \infty} \frac{1}{t} E \int_0^t [m_1 (S(s) - S^*)^2 + m_2 (I(s) - I^*)^2 + m_3 (Y(s) - Y^*)^2] ds \leq \delta.$$

**Proof.** Noting that  $(S^*, I^*, Y^*)$  is the positive equilibrium of model (2.1), one sees that

$$r \left( 1 - \frac{S^*}{K} \right) = \beta I^*, \beta S^* - c - aI^* = pY^*, kpI^* = d.$$

Let us now define

$$\begin{aligned}
V(S, I, Y) &= \eta \left[ S - S^* - S^* \log \frac{S}{S^*} + I - I^* - I^* \log \frac{I}{I^*} + \frac{1}{k} \left( Y - Y^* - Y^* \log \frac{Y}{Y^*} \right) \right] \\
&\quad + \frac{1}{2} \left[ S - S^* + I - I^* + \frac{1}{k} (Y - Y^*) \right]^2 + \frac{1}{k^2} \frac{c + 2aI^* + d}{p} \left( Y - Y^* - Y^* \log \frac{Y}{Y^*} \right) \\
&:= \eta V_1 + V_2 + \frac{1}{k^2} \frac{c + 2aI^* + d}{p} V_3.
\end{aligned}$$

Applying Itô's formula to stochastic differential system (2.2) yields

$$\begin{aligned}
dV_1 &= \left\{ (S - S^*) \left[ r \left( 1 - \frac{S}{K} \right) - \beta I \right] + \frac{S^* \sigma_1^2}{2} \right\} dt + \sigma_1 (S - S^*) dB_1(t) \\
&\quad + \left\{ (I - I^*) [\beta S - c - aI - pY] + \frac{I^* \sigma_2^2}{2} \right\} dt + \sigma_2 (I - I^*) dB_2(t) \\
&\quad + \frac{1}{k} \left\{ (Y - Y^*) [kpI - d] + \frac{Y^* \sigma_3^2}{2} \right\} dt + \sigma_3 (Y - Y^*) dB_3(t) \\
&:= LV_1 dt + \sigma_1 (S - S^*) dB_1(t) + \sigma_2 (I - I^*) dB_2(t) + \frac{1}{k} \sigma_3 (Y - Y^*) dB_3(t),
\end{aligned}$$

where

$$\begin{aligned}
LV_1 &= (S - S^*) \left[ r \left( 1 - \frac{S}{K} \right) - \beta I \right] + \frac{S^* \sigma_1^2}{2} + (I - I^*) [\beta S - c - aI - pY] + \frac{I^* \sigma_2^2}{2} \\
&\quad + \frac{1}{k} \left[ (Y - Y^*) (kpI - d) + \frac{Y^* \sigma_3^2}{2} \right] \\
&= r(S - S^*) - \frac{r}{K} (S - S^*)^2 - \frac{r}{K} (S - S^*) S^* - \beta (S - S^*) (I - I^*) - \beta (S - S^*) I^* \\
&\quad + \frac{S^* \sigma_1^2}{2} - a(I - I^*)^2 - a(I - I^*) I^* + \beta (S - S^*) (I - I^*) + \beta (I - I^*) S^* \\
&\quad - c(I - I^*) - p(I - I^*) (Y - Y^*) + \frac{I^* \sigma_2^2}{2} - pY^* (I - I^*) \\
&\quad + \frac{1}{k} \left[ kp(Y - Y^*) (I - I^*) + kpI^* (Y - Y^*) - d(Y - Y^*) + \frac{Y^* \sigma_3^2}{2} \right] \\
&= -\frac{r}{K} (S - S^*)^2 - a(I - I^*)^2 + \frac{1}{2} \left( \sigma_1^2 S^* + \sigma_2^2 I^* + \frac{1}{k} \sigma_3^2 Y^* \right). \tag{2.3}
\end{aligned}$$

Let

$$\begin{aligned}
dV_3 &= \left[ (Y - Y^*) (kpI - d) + \frac{Y^* \sigma_3^2}{2} \right] dt + (Y - Y^*) \sigma_3 dB_3(t) \\
&:= LV_3 dt + (Y - Y^*) \sigma_3 dB_3(t),
\end{aligned}$$

then

$$LV_3 = (Y - Y^*)(kpI - d) + \frac{Y^* \sigma_3^2}{2} = kp(Y - Y^*)(I - I^*) + \frac{Y^* \sigma_3^2}{2}.$$

Let

$$Z = S - S^* + I - I^* + \frac{1}{k}(Y - Y^*),$$

then

$$dZ = \left[ rS - \frac{r}{K}S^2 - cI - aI^2 - \frac{d}{k}Y \right] dt + \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \frac{1}{k} \sigma_3 Y dB_3(t).$$

Easy,

$$\begin{aligned} dV_2 &= \left[ S - S^* + I - I^* + \frac{1}{k}(Y - Y^*) \right] \left\{ \left[ rS - \frac{r}{K}S^2 - cI - aI^2 - \frac{d}{k}Y \right] dt + \sigma_1 S dB_1(t) \right. \\ &\quad \left. + \sigma_2 I dB_2(t) + \frac{1}{k} \sigma_3 Y dB_3(t) \right\} + \frac{1}{2} \left( \sigma_1^2 S^2 + \sigma_2^2 I^2 + \frac{1}{k^2} \sigma_3^2 Y^2 \right) dt \\ &:= LV_2 dt + \left[ S - S^* + I - I^* + \frac{1}{k}(Y - Y^*) \right] \left( \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \frac{1}{k} \sigma_3 Y dB_3(t) \right), \end{aligned}$$

where

$$\begin{aligned} LV_2 &= \left[ S - S^* + I - I^* + \frac{1}{k}(Y - Y^*) \right] \left[ rS - \frac{r}{K}S^2 - cI - aI^2 - \frac{d}{k}Y \right] \\ &\quad + \frac{1}{2} \left( \sigma_1^2 S^2 + \sigma_2^2 I^2 + \frac{1}{k^2} \sigma_3^2 Y^2 \right). \end{aligned}$$

Since

$$\begin{aligned} &rS - \frac{r}{K}S^2 - cI - aI^2 - \frac{d}{k}Y \\ &= rS - \frac{r}{K}(S - S^*)^2 + \frac{r}{K}S^{*2} - \frac{2r}{K}SS^* - cI - a(I - I^*)^2 + aI^{*2} - 2aII^* - \frac{d}{k}Y \\ &= -\frac{r}{K}(S - S^*)^2 + \left( r - \frac{2r}{K}S^* \right) S + \frac{r}{K}S^{*2} - cI - a(I - I^*)^2 + aI^{*2} - 2aII^* - \frac{d}{k}Y \\ &= -\frac{r}{K}(S - S^*)^2 + \left( r - \frac{2r}{K}S^* \right) (S - S^*) + \left( r - \frac{r}{K}S^* \right) S^* - c(I - I^*) - cI^* \\ &\quad - a(I - I^*)^2 - aI^{*2} - 2a(I - I^*)I^* - \frac{d}{k}(Y - Y^*) - \frac{d}{k}Y^* \\ &= -\frac{r}{K}(S - S^*)^2 + \left( r - \frac{2r}{K}S^* \right) (S - S^*) - (c + 2aI^*)(I - I^*) - a(I - I^*)^2 - pI^*(Y - Y^*), \end{aligned}$$

we have

$$\begin{aligned}
LV_2 &= \left[ S - S^* + I - I^* + \frac{1}{k}(Y - Y^*) \right] \left[ -\frac{r}{K}(S - S^*)^2 + \left( r - \frac{2r}{K}S^* \right) (S - S^*) \right. \\
&\quad \left. - pI^*(Y - Y^*) - (c + 2aI^*)(I - I^*) - a(I - I^*)^2 \right] \\
&\quad + \frac{1}{2} \left( \sigma_1^2 S^2 + \sigma_2^2 I^2 + \frac{1}{k^2} \sigma_3^2 Y^2 \right) \\
&= -\frac{r}{K} \left( S + I + \frac{1}{k}Y \right) (S - S^*)^2 + \left( r - \frac{r}{K}S^* + \frac{r}{K}I^* + \frac{1}{k} \frac{r}{K}Y^* \right) (S - S^*)^2 \\
&\quad - a \left( S + I + \frac{1}{k}Y \right) (I - I^*)^2 + \left( aS^* - aI^* + \frac{a}{k}Y^* - c \right) (I - I^*)^2 \\
&\quad + \left( r - \frac{2r}{K}S^* - c - 2aI^* \right) (S - S^*)(I - I^*) \\
&\quad + \frac{1}{k} \left( r - \frac{2r}{K}S^* - d \right) (S - S^*)(Y - Y^*) - \frac{1}{k} (c + 2aI^* + d) (I - I^*)(Y - Y^*) \\
&\quad - \frac{d}{k^2} (Y - Y^*)^2 + \frac{1}{2} \left( \sigma_1^2 S^2 + \sigma_2^2(x) I^2 + \frac{1}{k^2} \sigma_3^2 Y^2 \right) \\
&\leq \left( r - \frac{r}{K}S^* + \frac{r}{K}I^* + \frac{1}{k} \frac{r}{K}Y^* \right) (S - S^*)^2 + \left( aS^* - aI^* + \frac{a}{k}Y^* - c \right) (I - I^*)^2 \\
&\quad + \left( r - \frac{2r}{K}S^* - c - 2aI^* \right) (S - S^*)(I - I^*) + \frac{1}{2} \left( \sigma_1^2 S^2 + \sigma_2^2 I^2 + \frac{1}{k^2} \sigma_3^2 Y^2 \right) \\
&\quad + \frac{1}{k} \left( r - \frac{2r}{K}S^* - d \right) (S - S^*)(Y - Y^*) - \frac{1}{k} (c + 2aI^* + d) (I - I^*)(Y - Y^*) \\
&\quad - \frac{d}{k^2} (Y - Y^*)^2.
\end{aligned} \tag{2.4}$$

By the Cauchy inequality, it is easy to show that

$$\left( r - \frac{2r}{K}S^* - c - 2aI^* \right) (S - S^*)(I - I^*) \leq \frac{c}{2} (I - I^*)^2 + \frac{\left( r - \frac{2r}{K}S^* - c - 2aI^* \right)^2}{2c} (S - S^*)^2$$

and

$$\frac{1}{k} \left( r - \frac{2r}{K}S^* - d \right) (S - S^*)(Y - Y^*) \leq \frac{d}{2k^2} (Y - Y^*)^2 + \frac{\left( r - \frac{2r}{K}S^* - d \right)^2}{2d} (S - S^*)^2.$$



We can show from (2.4) that

$$\begin{aligned}
LV_2 \leq & \left( r - \frac{r}{K}S^* + \frac{r}{K}I^* + \frac{1}{k} \frac{r}{K}Y^* + \frac{(r - \frac{2r}{K}S^* - c - 2aI^*)^2}{2c} + \frac{(r - \frac{2r}{K}S^* - d)^2}{2d} + \sigma_1^2 \right) (S - S^*)^2 \\
& + \left( aS^* - aI^* + \frac{a}{k}Y^* - \frac{c}{2} + \sigma_2^2 \right) (I - I^*)^2 - \frac{1}{2k^2}(d - 2\sigma_3^2)(Y - Y^*)^2 \\
& - \frac{1}{k}(c + 2aI^* + d)(I - I^*)(Y - Y^*) + \sigma_1^2 S^{*2} + \sigma_2^2(x)I^{*2} + \frac{1}{k^2}\sigma_3^2 Y^{*2}.
\end{aligned} \tag{2.5}$$

Easy, we have

$$\begin{aligned}
LV_4 = & LV_2 + \frac{c + 2aI^* + d}{k^2 p} LV_3 \\
\leq & \left( r - \frac{r}{K}S^* + \frac{r}{K}I^* + \frac{1}{k} \frac{r}{K}Y^* + \frac{(r - \frac{2r}{K}S^* - c - 2aI^*)^2}{2c} + \frac{(r - \frac{2r}{K}S^* - d)^2}{2d} + \sigma_1^2 \right) (S - S^*)^2 \\
& + \left( aS^* - aI^* + \frac{a}{k}Y^* - \frac{c}{2} + \sigma_2^2 \right) (I - I^*)^2 - \frac{1}{2k^2}(d - 2\sigma_3^2)(Y - Y^*)^2 \\
& + \sigma_1^2 S^{*2} + \sigma_2^2(x)I^{*2} + \frac{1}{k^2}\sigma_3^2 Y^{*2} + \frac{(c + 2aI^* + d)\sigma_3^2 Y^*}{2k^2 p}.
\end{aligned} \tag{2.6}$$

Consequently, from (2.3) and (2.6), one has

$$\begin{aligned}
LV = & \eta LV_1 + LV_4 \\
\leq & - \left( \frac{r\eta}{2K} - \sigma_1^2 \right) (S - S^*)^2 - (\varpi - \sigma_2^2) (I - I^*)^2 - \frac{1}{2k^2}(d - 2\sigma_3^2)(Y - Y^*)^2 \\
& + \sigma_1^2 S^{*2} + \sigma_2^2 I^{*2} + \frac{1}{k^2}\sigma_3^2 Y^{*2} + \frac{(c + 2aI^* + d)\sigma_3^2 Y^*}{2k^2 p} + \frac{\eta}{2}(\sigma_1^2 S^* + \sigma_2^2 I^* + \frac{1}{k}\sigma_3^2 Y^*) \\
= & -m_1(S - S^*)^2 - m_2(I - I^*)^2 - m_3(Y - Y^*)^2 + \delta.
\end{aligned} \tag{2.7}$$

For any  $\delta < \min\{m_1 S^*, m_2 I^*, m_3 Y^*\}$ , the ellipsoid

$$-m_1(S - S^*)^2 - m_2(I - I^*)^2 - m_3(Y - Y^*)^2 + \delta = 0$$

lies entirely in  $R_+^3$ . Let  $U$  to be any neighborhood of the ellipsoid with  $\bar{U} \subseteq E_3 = R_+^3$ , thus for any  $x \in U \setminus E_l, LV \leq -\bar{M}$  ( $\bar{M}$  is a positive constant). Therefore, condition (2) in Assumption 2.1 is satisfied. Moreover, there exists a  $G = \min\{\sigma_1^2 x_1^2, \sigma_2^2 x_2^2, \sigma_3^2 x_3^2, (x_1, x_2, x_3) \in \bar{U}\} > 0$  such that

$$\sum_{i,j=1}^3 \left( \sum_{k=1}^3 a_{ik}(x)a_{jk}(x) \right) \xi_i \xi_j = \sigma_1^2 x_1^2 \xi_1^2 + \sigma_2^2 x_2^2 \xi_2^2 + \sigma_3^2 x_3^2 \xi_3^2 \geq G \|\xi\|^2$$

for all  $x \in \bar{U}$ ,  $\xi \in \mathbb{R}^3$ , which means condition (1) in Assumption 2.1 is satisfied. Therefore, the stochastic model (2.2) has a unique stationary distribution  $\mu(\cdot)$ , it also has the ergodic property.

Since

$$\begin{aligned} dV = & LV dt + \eta \left[ (S - S^*)\sigma_1 dB_1(t) + (I - I^*)\sigma_2 dB_2(t) + \frac{1}{k}(Y - Y^*)\sigma_3 dB_3(t) \right] \\ & + \left[ S - S^* + I - I^* + \frac{1}{k}(Y - Y^*) \right] \left[ \sigma_1 S dB_1(t) + \sigma_2 I dB_2(t) + \frac{1}{k}\sigma_3 Y dB_3(t) \right] \\ & + \frac{c + 2aI^* + d}{k^2 p} \sigma_3 (Y - Y^*) dB_3(t). \end{aligned} \quad (2.8)$$

Integrating system (2.8) from 0 to  $t$  and taking the expectation on both sides yields

$$\begin{aligned} EV(t) - EV(0) \leq & -m_1 E \int_0^t (S(s) - S^*)^2 ds - m_2 E \int_0^t (I(s) - I^*)^2 ds \\ & - m_3 E \int_0^t (Y(s) - Y^*)^2 ds + \delta t. \end{aligned} \quad (2.9)$$

Dividing both sides of (2.9) by  $t$  and let  $t \rightarrow +\infty$  yields

$$\begin{aligned} & \lim_{t \rightarrow \infty} \frac{EV(t) - EV(0)}{t} \\ \leq & -\frac{1}{t} \left[ m_1 E \int_0^t (S(s) - S^*)^2 ds + m_2 E \int_0^t (I(s) - I^*)^2 ds + m_3 E \int_0^t (Y(s) - Y^*)^2 ds \right] + \delta. \end{aligned}$$

Easy,

$$\limsup_{t \rightarrow \infty} \frac{1}{t} E \int_0^t [m_1 (S(s) - S^*)^2 + m_2 (I(s) - I^*)^2 + m_3 (Y(s) - Y^*)^2] ds \leq \delta. \quad (2.10)$$

This completes the proof.

**Theorem 2.2.** *Assume that the conditions in Theorem 2.1 are met. Let  $(S(t), I(t), Y(t))$  be a solution of model (2.2) with initial value  $(S(0), I(0), Y(0)) \in \mathbb{R}_+^3$ . If*

$$r > \frac{\sigma_1^2}{2}$$

and

$$\frac{kp}{\beta(d + \frac{\sigma_3^2}{2})} \left[ r - \frac{\sigma_1^2}{2} - \frac{r}{K\beta} \left( \frac{a(d + \frac{\sigma_2^2}{2})}{kp} + c + \frac{\sigma_2^2}{2} \right) \right] > 1,$$

then the solution  $(S(t), I(t), Y(t))$  of model (2.2) satisfies

$$\begin{cases} P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) ds = \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) = \tilde{S}^* \right\} = 1, \\ P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) ds = \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) = \tilde{I}^* \right\} = 1, \\ P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Y(s) ds = \int_{R_+^3} z_3 \mu(dz_1, dz_2, dz_3) = \tilde{Y}^* \right\} = 1, \end{cases} \quad (2.11)$$

where

$$\begin{cases} \tilde{S}^* = \frac{K}{r} \left[ r - \frac{\sigma_1^2}{2} - \frac{\beta \left( d + \frac{\sigma_3^2}{2} \right)}{kp} \right], \\ \tilde{I}^* = \frac{d + \frac{\sigma_3^2}{2}}{kp}, \\ \tilde{Y}^* = \frac{1}{p} \left[ \frac{K\beta}{r} \left( r - \frac{\sigma_1^2}{2} - \frac{\beta \left( d + \frac{\sigma_3^2}{2} \right)}{kp} \right) - \frac{a \left( d + \frac{\sigma_3^2}{2} \right)}{kp} - c - \frac{\sigma_2^2}{2} \right]. \end{cases}$$

**Proof.** By the definition of ergodic property, for any  $w$  ( $w$  is a positive constant), one has

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t (S^2(s) \wedge w) ds = \int_{R_+^3} (z_1^2 \wedge w) \mu(dz_1, dz_2, dz_3) \text{ a.s.} \quad (2.12)$$

Using the dominated convergence theorem and equation (2.12), one can see that

$$E \left[ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t (S^2(s) \wedge w) ds \right] = \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t E \left( S^2(s) \wedge w \right) ds < \infty, \quad (2.13)$$

Easy,

$$\int_{R_+^3} (z_1^2 \wedge w) \mu(dz_1, dz_2, dz_3) < \infty.$$

Let  $w \rightarrow \infty$ , one has

$$\int_{R_+^3} z_1^2 \mu(dz_1, dz_2, dz_3) < \infty.$$

In other words,  $S^2$  is integrable with respect to the measure  $\mu$ . By using the property of ergodicity, one sees that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) ds = \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) \text{ a.s.} \quad (2.14)$$

Similarly, one can derive that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) ds = \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) \text{ a.s.}$$

and

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Y(s) ds = \int_{R_+^3} z_3 \mu(dz_1, dz_2, dz_3) \text{ a.s.}$$

An application of Itô's formula implies

$$d \log S = \left[ r - \frac{1}{2} \sigma_1^2 - \frac{r}{K} S - \beta I \right] dt + \sigma_1 dB_1(t),$$

Integrating the above equation from 0 to  $t$  yields

$$\log S(t) - \log S(0) = \left( r - \frac{1}{2} \sigma_1^2 \right) t - \frac{r}{K} \int_0^t S(s) ds - \beta \int_0^t I(s) ds + \sigma_1 B_1(t).$$

Dividing both sides of the above equation by  $t$  and let  $t \rightarrow +\infty$ , we have

$$\lim_{t \rightarrow \infty} \frac{\log S(t)}{t} = r - \frac{1}{2} \sigma_1^2 - \frac{r}{K} \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) - \beta \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) := \rho_1.$$

If  $\rho_1 > 0$ , there exists a  $T = T(\omega)$  for any  $t > T$  one has

$$\log S(t) > \frac{\rho_1}{2} t.$$

Then

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) ds \rightarrow \infty,$$

which is in contradiction with equation (2.14).

If  $\rho_1 < 0$ , there exists a  $T = T(\omega)$  for  $t > T$  one gets that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) d < 0,$$

which is in contradiction with equation (2.14).

Therefore

$$\lim_{t \rightarrow \infty} \frac{\log S(t)}{t} = 0.$$

Similarly, one can derive that

$$\lim_{t \rightarrow \infty} \frac{\log I(t)}{t} = 0, \lim_{t \rightarrow \infty} \frac{\log Y(t)}{t} = 0.$$

So we have

$$\begin{cases} r - \frac{\sigma_1^2}{2} - \frac{r}{K} \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) - \beta \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) = 0, \\ -c - \frac{\sigma_2^2}{2} + \beta \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) - a \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) - p \int_{R_+^3} z_3 \mu(dz_1, dz_2, dz_3) = 0, \\ -d - \frac{\sigma_3^2}{2} + kp \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) = 0. \end{cases}$$

Moreover, there exists a positive solution  $(\tilde{S}^*, \tilde{I}^*, \tilde{Y}^*)$  of equation

$$\begin{cases} r - \frac{\sigma_1^2}{2} - \frac{r}{K}S - \beta I = 0, \\ -c - \frac{\sigma_2^2}{2} + \beta S - aI - pY = 0, \\ -d - \frac{\sigma_3^2}{2} + kpI = 0, \end{cases} \quad (2.15)$$

if  $r > \frac{\sigma_1^2}{2}$  and  $\frac{kp}{\beta(d + \frac{\sigma_3^2}{2})} \left[ r - \frac{\sigma_1^2}{2} - \frac{r}{K\beta} \left( \frac{a(d + \frac{\sigma_2^2}{2})}{kp} + c + \frac{\sigma_2^2}{2} \right) \right] > 1$  are fulfilled.

The above discussion gives

$$\begin{cases} P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) ds = \int_{R_+^3} z_1 \mu(dz_1, dz_2, dz_3) = \tilde{S}^* \right\} = 1, \\ P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t I(s) ds = \int_{R_+^3} z_2 \mu(dz_1, dz_2, dz_3) = \tilde{I}^* \right\} = 1, \\ P \left\{ \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Y(s) ds = \int_{R_+^3} z_3 \mu(dz_1, dz_2, dz_3) = \tilde{Y}^* \right\} = 1. \end{cases} \quad (2.16)$$

This completes the proof.

### 3. Evolutionary adaptive dynamics

#### 3.1. The model

When the resident population(the infected prey with trait  $x$ ) was invaded by a rare mutant prey population with a markedly different trait  $y$ , the predator-prey system (2.2) becomes

$$\begin{cases} dS = S(t) \left[ r \left( 1 - \frac{S(t)}{K} \right) - \beta(x)I(t) - \beta(y)I_{mut}(t) \right] dt + \sigma_1 S(t) dB_1(t), \\ dI = I(t) [\beta(x)S(t) - c(x) - p(x)Y(t) - a(x,x)I(t) - a(x,y)I_{mut}(t)] dt + \sigma_2(x)I(t) dB_2(t), \\ dI_{mut} = I_{mut}(t) [\beta(y)S(t) - c(y) - p(y)Y(t) - a(y,y)I_{mut}(t) - a(y,x)I(t)] dt \\ \quad + \sigma_2(y)I_{mut}(t) dB_2(t), \\ dY = Y(t) [kp(x)I(t) + k'p(y)I_{mut}(t) - d] dt + \sigma_3 Y(t) dB_3(t), \end{cases} \quad (3.1)$$

where the phenotypic traits  $x, y$  stand for the severity of the disease in  $I(t)$  and  $I_{mut}(t)$ , respectively.  $S(t), I(t)$  and  $I_{mut}(t)$ , respectively, stand for the densities of susceptible prey, infected prey with trait  $x$ , infected prey with trait  $y$  at time  $t$ ,  $Y(t)$  represents the population density of

predator at time  $t$ .  $\beta(x)$  and  $\beta(y)$  stand for the infection rates from susceptible  $S(t)$  to infected  $I(t)$  with trait  $x$  and infected  $I_{mut}(t)$  with trait  $y$ , respectively.  $c(x)$  and  $c(y)$  are the diseased death rates of  $I(t)$  and  $I_{mut}(t)$ , respectively.  $p(x)$  and  $p(y)$  represent the attack rates on infected prey  $I(t)$  and infected prey  $I_{mut}(t)$ , respectively. The competition coefficient  $a(x,y)$  denotes the influence of trait  $x$  on trait  $y$ ,  $a(x,x)$  and  $a(y,y)$  are the intraspecific competition coefficients of  $I(t)$  and  $I_{mut}(t)$ , respectively.  $B_i(t)(i = 1, 2, 3)$  are independent Brownian motions with intensity  $\sigma_i^2(i = 1, 2, 3)$ ,  $K$  represents the environmental maximum capacity,  $r$  is the intrinsic growth rate of  $S(t)$ ,  $d$  represents the natural death rate of predator.  $k$  and  $k'$  ( $0 \leq k, k' \leq 1$ ) stand for the conversion rates from  $I(t)$  to  $Y(t)$  and from  $I_{mut}(t)$  to  $Y(t)$ , respectively.

Generally speaking, the more severe the disease is, the weaker the population's ability to resist disturbance is. That is to say, the more severe the disease is, the more the dead diseased prey is. Therefore, we choose the following interference intensity function:

$$\sigma_2(x) = v \left[ 1 - (1 + 0.2\delta x) \exp(-0.2\delta x) \right], \quad (3.2)$$

where  $\delta$  is a coefficient that is positively correlated with the intensity of stochastic perturbation,  $v > 0$  is a constant. See Fig. 1.

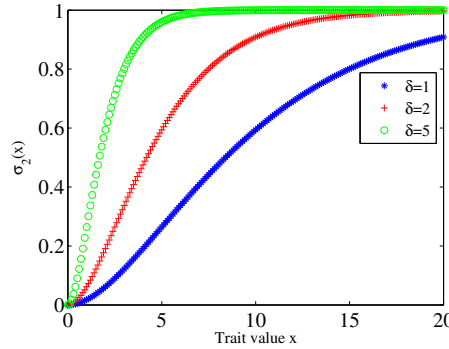


FIG. 1. Convex-concave shape of the intensity of noises  $\sigma_2(x_i)$  as given by system (3.2)( $v = 1$ ).

The greater pathogenic toxicity of the virus in infected prey implies the greater infection rate, diseased death rate and predation rate. Therefore, in system (3.1), we choose the following infection rate function, diseased death rate function, attack rate function:

$$\beta(x) = x + l, c(x) = e_1 x, p(x) = e_2 x,$$

where  $l, e_1, e_2$  are positive constants.

It is widely accepted that prey with serious disease is at a disadvantage to survive when competes with prey who has light disease. Therefore, the competition coefficient  $a(x_i, x_j)$  which denotes the influence of trait  $x_j$  on trait  $x_i$  is an increasing function, which is given by

$$a(x_i, x_j) = f \left( 1 - \frac{1}{1 + m \exp(w(x_i - x_j))} \right), \quad (3.3)$$

where  $f, m, w$  are positive constants. See Fig. 2.

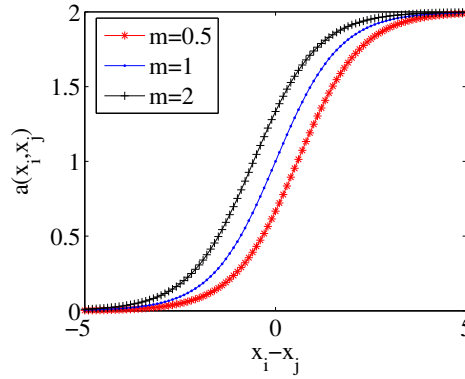


FIG. 2. Concave-convex shape of  $a(x_i, x_j)$  as given by (3.3) ( $m = 0.5; 1; 2; f = 2; w = 1.2$ ).

Letting  $I_{mut} = 0$  and using the well-known Itô's formula in the third equation of model (3.1), we get

$$\frac{dI_{mut}}{I_{mut}} = d \ln I_{mut} = \left[ \beta(y)S(t) - c(y) - p(y)Y(t) - a(y, x)I(t) - \frac{\sigma_2^2(y)}{2} \right] dt + \sigma_2(y)dB_2(t).$$

Let

$$F(x, y) = \left[ \beta(y)S(t) - c(y) - p(y)Y(t) - a(y, x)I(t) - \frac{\sigma_2^2(y)}{2} \right] dt + \sigma_2(y)dB_2(t). \quad (3.4)$$

Integrating both sides of equation (3.4) from 0 to  $t$  leads to

$$\begin{aligned} \int_0^t F(x, y) dt &= \beta(y) \int_0^t S(s) ds - \int_0^t c(y) ds - p(y) \int_0^t Y(s) ds - a(y, x) \int_0^t I(s) ds - \int_0^t \frac{\sigma_2^2(y)}{2} ds \\ &\quad + \sigma_2(y) (B_2(t) - B_2(0)). \end{aligned} \quad (3.5)$$

Dividing both sides of equation (3.5) by  $t$  yields

$$\langle F(x, y) \rangle = \beta(y) \langle S(t) \rangle - c(y) - p(y) \langle Y(t) \rangle - a(y, x) \langle I(t) \rangle - \frac{\sigma_2^2(y)}{2} + \frac{\sigma_2(y) (B_2(t) - B_2(0))}{t}.$$

Letting  $t \rightarrow \infty$ , using  $\lim_{t \rightarrow \infty} t^{-1}B(t) = 0$  and  $\lim_{t \rightarrow \infty} t^{-1}B(0) = 0$ , we see that

$$f(x, y) \equiv \lim_{t \rightarrow \infty} \langle F(x, y) \rangle = \beta(y)\tilde{S}^* - c(y) - p(y)\tilde{Y}^* - a(y, x)\tilde{I}^* - \frac{\sigma_2^2(y)}{2}, \text{ a.s.} \quad (3.6)$$

Note that  $f(x, y)$  is the long-term average exponential growth rate of the mutant population. Hence, if  $f(x, y)$  is positive, then the mutant prey can invade, otherwise the resident prey can not be invaded and the mutant prey will die out. So  $f(x, y)$  is the fitness function[23]. Then we can obtain a local fitness gradient  $D(x)$  which determines the direction of evolution, and  $D(x)$  is given by

$$\begin{aligned} D(x) &= \left. \frac{\partial f(x, y)}{\partial y} \right|_{y=x} \\ &= \frac{K\beta'(x)}{r} \left[ r - \frac{\sigma_1^2}{2} - \frac{\beta(x) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x)} \right] - c'(x) - d'(x, x) \frac{d + \frac{\sigma_3^2}{2}}{kp(x)} - \frac{p'(x)}{p(x)} \\ &\quad \times \left[ \frac{K\beta(x)}{r} \left( r - \frac{\sigma_1^2}{2} - \frac{\beta(x) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x)} \right) - \frac{a(x, x) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x)} - c(x) - \frac{\sigma_2^2(x)}{2} \right] \\ &\quad - \sigma_2(x)\sigma_2'(x). \end{aligned} \quad (3.7)$$

If  $D(x) > 0$ , the mutant infectious prey with trait  $y$  which is obviously bigger than  $x$  can invade and replace the resident infectious prey population, otherwise if the fitness gradient  $D(x) < 0$ , the resident infectious prey population can be invaded and took over by the mutant infectious prey with trait  $y$  which is obviously smaller than  $x$ . Thus, we know that the direction of evolution is determined by  $D(x)$ . Since mutations are random and sufficiently small, so the evolutionary dynamics of trait  $x$  can expressed as [9]

$$\frac{dx}{dt} = \frac{1}{2} \mu \sigma^2 \widetilde{I(x)}^* D(x), \quad (3.8)$$

where  $D(x)$  is the fitness gradient in equation (3.7),  $\widetilde{I(x)}^*$  is the ecological equilibrium population size of infected prey which is given by (2.16),  $\mu$  represents the rate that a resident infectious prey give birth to a mutant infectious prey,  $\frac{1}{2} \mu \sigma^2$  is the mutation rate of the infectious prey.

### 3.2. Evolutionary analysis



When  $D(x^*) = 0$ , the trait  $x^*$  is called an evolutionary singular strategy. At the evolutionary singular strategy point  $x^*$ , whether the evolution continues depends on its evolutionary stability and convergence stability. In the neighborhood of the evolutionary singular strategy  $x^*$ , if the resident population with strategy  $x$  was invaded by the nearby mutant strategy  $y$ , then  $x^*$  is called the convergence stable strategy.

According to (3.7), we know that in the vicinity of  $x^*$ , if  $x < x^*$ , then  $D(x) > 0$ , otherwise if  $x > x^*$ , then  $D(x) < 0$ . Therefore, the condition for convergence stable is as follows

$$\begin{aligned}
\left. \frac{dD(x)}{dx} \right|_{x=x^*} &= -\frac{K\beta'(x^*)}{rk^2p^2(x^*)} \left[ \beta'(x^*) \left( d + \frac{\sigma_3^2}{2} \right) kp(x^*) - \beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right) kp'(x^*) \right] \\
&\quad - \frac{p''(x^*)p(x^*) - p'^2(x^*)}{p^2(x^*)} \left[ \frac{K\beta(x^*)}{r} \left( r - \frac{\sigma_1^2}{2} - \frac{\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right) \right. \\
&\quad \left. - \frac{a(x^*, x^*) \left( d + \frac{\sigma_3^2}{2} \right) - c(x^*) - \frac{\sigma_2(x^*)^2}{2}}{kp(x^*)} - c''(x^*) - a''(x^*, x^*) \frac{d + \frac{\sigma_3^2}{2}}{kp(x^*)} \right. \\
&\quad \left. + a'(x^*, x^*) \frac{d + \frac{\sigma_3^2}{2}}{kp^2(x^*)} - \frac{p'(x^*)}{p(x^*)} \left[ \frac{K\beta'(x^*)}{r} \left( r - \frac{\sigma_1^2}{2} - \frac{\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right) \right. \right. \\
&\quad \left. \left. - \frac{K\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{rkp^2(x^*)} \left( \beta'(x^*)p(x^*) - \beta(x^*)p'(x^*) \right) \right. \right. \\
&\quad \left. \left. - \frac{\left( d + \frac{\sigma_3^2}{2} \right)}{kp^2(x^*)} \left( a'(x^*, x^*)p(x^*) - a(x^*, x^*)p'(x^*) \right) - c'(x^*) - \sigma_2(x^*)\sigma_2'(x^*) \right] \right. \\
&\quad \left. - \sigma_2'^2(x^*) - \sigma_2(x^*)\sigma_2''(x^*) + \frac{K\beta''(x^*)}{r} \left[ r - \frac{\sigma_1^2}{2} - \frac{\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right] \right] < 0.
\end{aligned} \tag{3.9}$$

The singular strategy  $x^*$  is evolutionary stable (i.e. ESS) if it satisfies

$$\begin{aligned}
\left. \frac{\partial f^2(x, y)}{\partial y} \right|_{y=x=x^*} &= \frac{K\beta''(x^*)}{r} \left[ r - \frac{\sigma_1^2}{2} - \frac{\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right] - c''(x^*) - a''(x^*, x^*) \frac{d + \frac{\sigma_3^2}{2}}{kp(x^*)} \\
&\quad - \frac{p''(x^*)}{p(x^*)} \left[ \frac{K\beta(x^*)}{r} \left( r - \frac{\sigma_1^2}{2} - \frac{\beta(x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right) - \frac{a(x^*, x^*) \left( d + \frac{\sigma_3^2}{2} \right)}{kp(x^*)} \right. \\
&\quad \left. - c(x^*) - \frac{\sigma_2(x^*)^2}{2} \right] - \sigma_2'^2(x^*) - \sigma_2(x^*)\sigma_2''(x^*) < 0.
\end{aligned} \tag{3.10}$$

The singular strategy  $x^*$  is a continuously stable strategy (i.e. CSS) if it is both convergence stable and evolutionary stable, which will bring the endpoint of the evolution. If the evolutionary singular point  $x^*$  satisfies convergence stable but lacks evolutionary stable, it will bring evolutionary branching. In other words, the single population will become two different species. Otherwise, if the evolutionary singular strategy  $x^*$  is both convergence stable and evolutionary stable, the evolution will stop.

**Proposition 3.1.** *If the singularity  $x^*$  of (3.8) satisfies inequalities (3.9) and (3.10), the evolutionary singular point  $x^*$  is a continuously stable strategy (CSS); If the singularity  $x^*$  of (3.8) satisfies (3.9) but does not satisfy (3.10), the evolutionary singular point  $x^*$  is a branching point.*

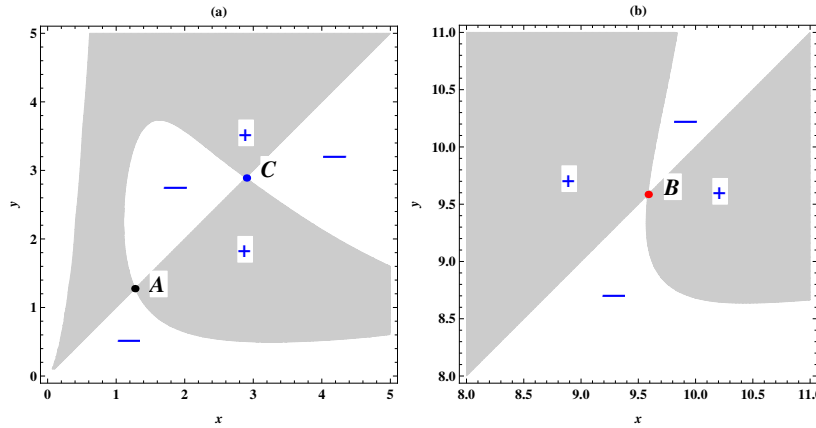


FIG. 3. Pairwise invasibility plots. The dashed areas marked with ‘+’ indicate that the fitness function  $f(x,y) > 0$ , in contrast, the areas marked with ‘-’ say  $f(x,y) < 0$ . A is an evolutionary stable point, B is a branching point, C is a repeller. (a) Multisingular strategies when  $\delta = 5$ ; (b) The singular strategy is an evolutionary branching point when  $\delta = 0.4$ ;

We utilize “pairwise invasibility plot” (PIP) to investigate how the intensity of noises affects the results of evolution. Therefore, we use two different values of  $\delta$  and obtained two PIPs, see Fig. 3. Here we choose  $K = 1, k = 0.1, d = 0.1, r = 0.1, \sigma_1 = 0.1, \sigma_3 = 0.2, v = 1, m = 2, w = 1.2, f = 1, l = 0.5, e_1 = 0.05, e_2 = 0.05$ . In Fig. 3(a), we choose  $\delta = 5$ , one sees that the singular strategy  $x^*$  is both evolutionarily stable and convergence. Thus  $x^*$  is the endpoint of the evolutionary process and is a CSS. In Fig. 3(b), we choose  $\delta = 0.4$ , the singular strategy  $x^*$

is convergence stable but evolutionarily stable. Thus  $x^*$  is an evolutionary branching point, and all nearby mutant population can invade.

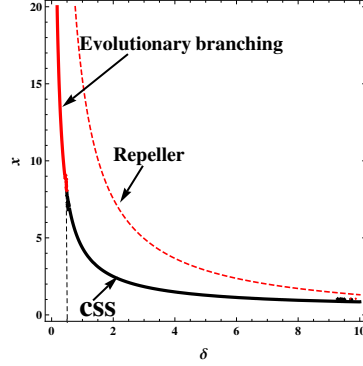


FIG. 4. Bifurcation diagram for evolutionary singular strategy  $x$  and the intensity of noise  $\delta$ . Red solid line represents unstable singular strategy  $x$ , black solid line represents the CSS, and read dashed line represents the repeller. The parameter values are  $K = 1, k = 0.1, d = 0.1, r = 0.1, \sigma_1 = 0.1, \sigma_3 = 0.2, v = 1, m = 2, w = 1.2, f = 1, l = 0.5, e_1 = 0.05, e_2 = 0.05$ .

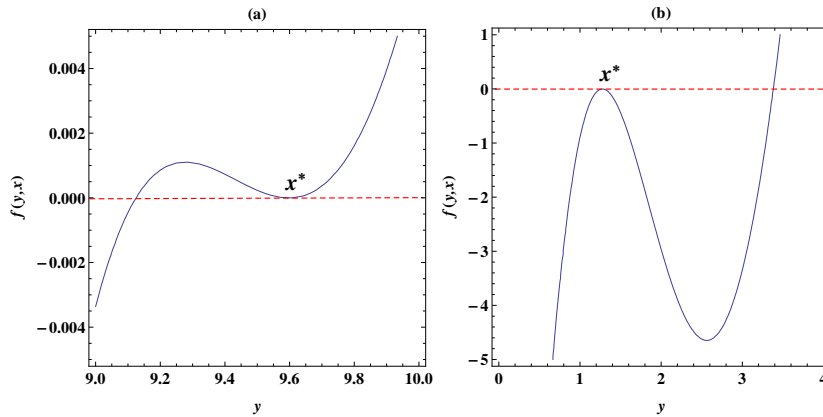


FIG. 5. Fitness landscape at the singular  $x^*$ . (a)  $\delta = 0.4$ ; (b)  $\delta = 5$ ; Other parameter values:  $K = 1, k = 0.1, d = 0.1, r = 0.1, \sigma_1 = 0.1, \sigma_3 = 0.2, v = 1, m = 2, w = 1.2, f = 1, l = 0.5, e_1 = 0.05, e_2 = 0.05$ .

We next study the effect of different intensity of noise to the evolutionary stability and evolutionary branching of the singular strategy. We first plot a bifurcation diagram, see Fig. 4. From Fig. 4, an increase of the intensity of noise will lead to the decrease of singular strategy  $x$ . Once the intensity of the noise exceeds the threshold value  $\delta = 0.448$ , the evolutionary stability of

the singular strategy will be changed. That is to say, noise with small intensity can lead to the evolutionary branching, in contrast, noise with large intensity may cause a continuous stability strategy (CSS). In Fig.5 (a), we choose  $\delta = 0.4$ , it is easy to see that  $f(y, x)$  near strategy  $x^*$  is positive and convex, thus the strategy can experience evolutionary branching. In Fig.5(b), we choose  $\delta = 5$ , we can see that  $f(y, x)$  near strategy  $x^*$  is negative and concave, thus the strategy can not experience evolutionary branching.

## 4. Discussion

This paper considers a stochastic predator-prey model with disease in the prey under white noise disturbances and this paper shows that the stochastic model has a unique stationary distribution with ergodic property. Furthermore, we investigate asymptotic behavior of the stochastic system around the endemic equilibrium of the deterministic model and we explored the evolution of pathogen virulence of diseased prey with phenotype trait  $x$ . By modeling population dynamics under these conditions, we gain the fitness function, then we give the conditions under which the resident diseased prey population experienced evolutionary branching. The biological significance of the results shows that an increase in the intensity of noise will cause the decrease in the singular strategy  $x$ . In addition, noise with small intensity can lead to the evolutionary branching, in contrast, noise with large intensity may cause a continuous stability strategy (CSS), which implies that the white noise stochastic disturbance is advantage for the control of the epidemic disease.

This paper intends to develop a theoretical framework for investigating the evolutionary adaptive dynamics of a stochastic differential system. We apply our theoretical method to understand the evolutionary dynamics under stochastic differential equations. As a consequence, this paper proposes a new theoretical method for evolutionary adaptive dynamics based on stochastic differential equations. A promising extension of this work is to consider the environment with disturbance of Lévy jumps or Markov process.

### Conflict of Interests

The authors declare that there is no conflict of interests.

## Acknowledgements

This work is supported by the National Natural Science Foundation of China (11371230, 11501331), the SDUST Research Fund (2014TDJH102), Shandong Provincial Natural Science Foundation, China (ZR2015AQ001, BS2015SF002), Joint Innovative Center for Safe And Effective Mining Technology and Equipment of Coal Resources, Shandong Province, a Project of Shandong Province Higher Educational Science and Technology Program of China (J13LI05).

## REFERENCES

- [1] K.P. Hadeler, H.I. Freedman, Predator-prey populations with parasitic infection, *J. Math. Biol.* 27 (1989), 609-631.
- [2] J. Chattopadhyay, O. Arino, A predator-prey model with disease in the prey, *Nonlinear. Anal.* 36 (1999), 747-766.
- [3] Y.N. Xiao, L.S. Chen, Modeling and analysis of a predator-prey model with disease in the prey, *Math. Biosci.* 171 (2001), 59-82.
- [4] B. Mukhopadhyay, R. Bhattacharyya, Role of predator switching in an eco-epidemiological model with disease in the prey, *Ecol. Model.* 220 (2009), 931-939.
- [5] Y.N. Xiao, L.S. Chen, Analysis of a Three Species Eco-Epidemiological Model, *J. Math. Anal. Appl.* 258 (2001), 733-754.
- [6] B.W. Kooi, G.A.V. Voorn, K.P. Das, Stabilization and complex dynamics in a predator-prey model with predator suffering from an infectious disease, *Ecol. Complex.* 8 (2011), 113-122.
- [7] H.W. Hethcote, W.D. Wang, L.T. Han, Z.E. Ma, A predator-prey model with infected prey, *Theor. Popul. Biol.* 66 (2004), 259-268.
- [8] L.T. Han, Z.E. Ma, Four Predator Prey Models with Infectious Diseases, *Math. Comput. Model.* 34 (2001), 849-858.
- [9] U. Dieckmann, P. Marrowb, R. Law, Evolutionary cycling in predator-prey interactions: population dynamics and the red queen, *J. Theor. Biol.* 176 (1995), 91-102.
- [10] J. Zu, M. Mimura, Y. Takeuchi, Adaptive evolution of foraging-related traits in a predator-prey community, *J. Theor. Biol.* 268 (2011), 14-29.
- [11] J. Zu, J.L. Wang, Adaptive evolution of attack ability promotes the evolutionary branching of predator species, *Theor. Popul. Biol.* 89 (2013), 12-23.
- [12] X.Z. Meng, R.Liu, L.D. Liu, T.H. Zhang, Evolutionary analysis of a predator-prey community under natural and artificial selections, *Appl. Math. Model.* 39 (2015), 574-585.

- [13] X.Z. Meng, R. Liu, T.H. Zhang, Adaptive dynamics for a non-autonomous Lotka-Volterra model with size-selective disturbance, *Nonlinear. Anal.* 16 (2014), 202-213.
- [14] M. Liu, H. Qiu, K. Wang, A remark on a stochastic predator-prey system with time delays, *Appl. Math. Lett.* 26 (2013), 318-323.
- [15] Q. M. Zhang, D.Q. Jiang, Z.W. Liu, D. O'Regan, The long time behavior of a predator-prey model with disease in the prey by stochastic perturbation, *Appl. Math. Comput.* 245 (2014), 305-320.
- [16] C.Y. Ji, D.Q. Jiang, N.Z. Shi, Analysis of a predator-prey model with modified Leslie-Gower and Holling-type II schemes with stochastic perturbation, *J. Math. Anal. Appl.* 359 (2009), 482-498.
- [17] J.L. Liu, K. Wang, Asymptotic properties of a stochastic predator-prey system with Holling II functional response, *Commun. Nonlinear. Sci.* 16 (2011), 4037-4048.
- [18] M. Liu, K. Wang, Global stability of a nonlinear stochastic predator-prey system with Beddington-DeAngelis functional response, *Commun. Nonlinear. Sci.* 16 (2011), 1114-1221.
- [19] C.Y. Ji, D.Q. Jiang, X.Y. Li, Qualitative analysis of a stochastic ratio-dependent predator-prey system, *J. Comput. Appl. Math.* 235 (2011), 1326-1341.
- [20] M. Costa, C. Hauzy, N. Loeuille, S. Méléard, Stochastic eco-evolutionary model of a prey-predator community, *J. Math. Biol.* 72 (2016), 573-622.
- [21] U. Dieckmann, R. Law, The Dynamical Theory of Coevolution: A Derivation from Stochastic Ecological Processes, *J. Math. Biol.* 34 (1996), 579-612.
- [22] R. Z. Hasminskij, G.N. Milstejn, M.B. Nevelson, Stochastic stability of differential equations, Springer-Verlag, 2012.
- [23] J.A.J. Metz, R.M. Nisbet, S.A.H. Geritz, How should we define fitness for general ecological scenarios, *Trends. Ecol. Evol.* 7 (1992), 198-202.