Contents lists available at ScienceDirect

Commun Nonlinear Sci Numer Simulat

journal homepage: www.elsevier.com/locate/cnsns

Research paper

Stability of a stochastic one-predator-two-prey population model with time delays

Jing Geng^a, Meng Liu^{a,*}, Youqiang Zhang^b

^a School of Mathematical Science, Huaiyin Normal University, Huaian 223300, PR China
 ^b Department of Mathematics, Harbin Institute of Technology (Weihai), Weihai, 264209, PR China

ARTICLE INFO

Article history: Received 25 October 2016 Revised 6 March 2017 Accepted 23 April 2017 Available online 3 May 2017

Keywords: Two-prey-one-predator model Random perturbations Delays Stability in distribution

ABSTRACT

This paper is concerned with the stability in distribution of a delay stochastic population model with two competing preys (X_1 and X_2) and one predator (X_3). Under some assumptions we prove that there are three numbers $\gamma_1 > \gamma_2 > \gamma_3$ which have the following properties: if $\gamma_1 < 1$, then all the populations go to extinction almost surely (a.s.), i.e., $\lim_{t\to+\infty} X_i(t) = 0$ a.s., i = 1, 2, 3; If $\gamma_i > 1 > \gamma_{i+1}$, i = 1, 2, then the distribution of ($X_1(t), \ldots, X_i(t)$)^T converges weakly to a unique ergodic invariant distribution and $\lim_{t\to+\infty} X_j(t) = 0$ a.s., $j = i + 1, \ldots, 3$; If $\gamma_3 > 1$, then the distribution of ($X_1(t), X_2(t), X_3(t)$)^T converges weakly to a unique ergodic invariant distribution as. The influence of random perturbations on the stability are discussed and some numerical simulations are given to illustrate the main results.

© 2017 Elsevier B.V. All rights reserved.

1. Introduction

In the natural world, it is a common phenomenon that a predator feeds on some competing preys, for example, Hyenas, Zebra and Connochaetes in Africa. On the other hand, time delays should be taken into account in biological models [1]. Therefore in the past few decades, delay population models with two competing preys and one predator have received great attention and have been studied extensively. Among various types of delay population models with two competing preys and one predator, we should specially mention the following Lotka–Volterra system, which has been widely studied in [2–6]:

$$\begin{cases} dX_1(t) = X_1(t) \Big[b_1 - c_{11}X_1(t) - c_{12}X_2(t - \tau_{12}) - c_{13}X_3(t - \tau_{13}) \Big] dt, \\ dX_2(t) = X_2(t) \Big[b_2 - c_{21}X_1(t - \tau_{21}) - c_{22}X_2(t) - c_{23}X_3(t - \tau_{23}) \Big] dt, \\ dX_3(t) = X_3(t) \Big[-b_3 + c_{31}X_1(t - \tau_{31}) + c_{32}X_2(t - \tau_{32}) - c_{33}X_3(t) \Big] dt. \end{cases}$$

with initial data

 $X_i(\theta) = \xi_i(\theta), \quad \theta \in [-\bar{\tau}, 0], \quad \bar{\tau} = \max\{\tau_{ij}\},$

* Corresponding author.

http://dx.doi.org/10.1016/j.cnsns.2017.04.022 1007-5704/© 2017 Elsevier B.V. All rights reserved.





CrossMark

(1)

E-mail addresses: liumeng0557@sina.com, liumeng0557@163.com (M. Liu).

where $X_i(t)$ is the size of the prey *i*, i = 1, 2, and $X_3(t)$ is the size of the predator. b_i is the growth rate of species *i*, $i = 1, 2, b_3$ is the death rate of the predator. c_{ii} is the intra-specific competition rate, i = 1, 2, 3. c_{12} and c_{21} are the inter-specific competition rates between species 1 and 2, c_{13} and c_{23} are the capture rates, c_{31} and c_{32} stand for the efficiency of food conversion. All coefficients mentioned above are positive constants. $\tau_{ij} \ge 0$ represents the time delay. $\xi(\theta) = (\xi_1(\theta), \xi_2(\theta), \xi_3(\theta))^T \in \mathbb{U}$, \mathbb{U} represents the space of all the continue functions from $[-\bar{\tau}, 0]$ to $R_+^3 = \{x = (x_1, x_2, x_3) \in R^3 | x_i > 0, i = 1, 2, 3\}$.

On the other hand, the natural growth of species in the real world is inevitably subject to environmental fluctuations [7]. It is therefore important to study the stochastic population models and reveal the effects of stochasticity on the dynamics of the models. There are several approaches to introduce the stochastic fluctuations into population models. Here we follow the approach adopted in [8–30], i.e., we assume that the stochastic factors mainly affect the growth/death rates of the species, with

$$b_i \rightarrow b_i + \beta_i W_i(t), \quad i = 1, 2, 3,$$

where $\{W(t)\}_{t\geq 0} = \{W_1(t), W_2(t), W_3(t)\}_{t\geq 0}$ is a three-dimensional Brownian motion defined on a complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}\}_{t\geq 0}, \mathcal{P})$ with a filtration $\{\mathcal{F}\}_{t\geq 0}$ satisfying the usual conditions, and β_i^2 is the intensity of the noise, i = 1, 2, 3. Thus we derive the following stochastic delay model:

$$dX_{1}(t) = X_{1}(t) \begin{bmatrix} b_{1} - c_{11}X_{1}(t) - c_{12}X_{2}(t - \tau_{12}) - c_{13}X_{3}(t - \tau_{13}) \end{bmatrix} dt + \beta_{1}X_{1}(t)dW_{1}(t),$$

$$dX_{2}(t) = X_{2}(t) \begin{bmatrix} b_{2} - c_{21}X_{1}(t - \tau_{21}) - c_{22}X_{2}(t) - c_{23}X_{3}(t - \tau_{23}) \end{bmatrix} dt + \beta_{2}X_{2}(t)dW_{2}(t),$$

$$dX_{3}(t) = X_{3}(t) \begin{bmatrix} -b_{3} + c_{31}X_{1}(t - \tau_{31}) + c_{32}X_{2}(t - \tau_{32}) - c_{33}X_{3}(t) \end{bmatrix} dt + \beta_{3}X_{3}(t)dW_{3}(t),$$
(2)

with initial value (1).

In the investigation of population model, an interesting question is to consider the stability of positive equilibrium state [1]. However, lots of stochastic population models (for example, model (2)) do not have the traditional positive equilibrium state. Several researchers (see e.g., [8–16]) have suggested that one can consider the stability in distribution of the solutions (SDS) of stochastic population models. As far as we are concerned, no results relative to the SDS of model (2) have been reported. One reason is that the classical approaches could not be used.

One classical approach to investigate the SDS of stochastic population models is to solve the corresponding Fokker-Planck equations explicitly (see, e.g. [8]). However, this is almost impossible for stochastic population models with time delays. Another approach is the Markov semigroup theory, see, e.g., [9,10,27]. However, this approach requires some standard measure. The standard measure of the phase space of ordinary differential equations is the Lebesgue measure. But it is difficult to decide the standard measure of the phase space of delay differential equations (communicated with Professor Ryszard Rudnick). The third approach is the Lyapunov function method proposed in [31] which has been widely used in literatures, for example, by using this approach, Ji et al. [11,12] studied the SDS of predator-prey models perturbed by white noises; Mao [13] explored the SDS of a *n*-species Lotka–Volterra mutualism system with random perturbations; Jiang et al. [14] considered the SDS of a *n*-species stochastic Lotka–Volterra competitive model; Zou et al. [15] investigated the SDS of a predator-prey model perturbed by white noises; Zhao et al. [16] analyzed the SDS of a three-species stochastic Lotka– Volterra competitive system in polluted environments. However, the Lyapunov function approach needs the Markov property of the solutions which the stochastic delay models do not own. The fourth approach is the basic theory of SDS for stochastic functional differential equations (see, e.g., Hu and Wang [32]). However, this approach requires that the coefficients of the models must obey the linear growth condition, while most population systems do not obey.

In this paper, we use an asymptotic approach [27] to analyze the SDS of model (2). Under some assumptions we show that there are three numbers $\gamma_1 > \gamma_2 > \gamma_3$ which are represented by the coefficients of the model. We prove that: if $\gamma_1 < 1$, then $\lim_{t\to+\infty} X_i(t) = 0$ almost surely (a.s.), i = 1, 2, 3; If $\gamma_i > 1 > \gamma_{i+1}$, i = 1, 2, then the distribution of $(X_1(t), \ldots, X_i(t))^T$ converges weakly to a unique ergodic invariant distribution (UEID) and $\lim_{t\to+\infty} X_j(t) = 0$ a.s., $j = i + 1, \ldots, 3$; If $\gamma_3 > 1$, then the distribution of $(X_1(t), X_2(t), X_3(t))^T$ converges weakly to a UEID a.s..

The organization of this paper is outlined as follows. In Section 2, we give the main theorem and its proof. In Section 3, we introduce some numerical simulations to illustrate the main results. In Section 4, we discuss the theoretical results and their biological interpretations.

2. Main results

For the sake of simplification, we define some notations:

$$\begin{aligned} a_i &= b_i - \frac{\beta_i^2}{2}, \quad i = 1, 2, \quad a_3 = b_3 + \frac{\beta_3^2}{2}, \\ \Gamma &= \begin{vmatrix} c_{11} & b_1 & \beta_1^2/2 \\ c_{21} & b_2 & \beta_2^2/2 \\ -c_{31} & -b_3 & \beta_3^2/2 \end{vmatrix}, \quad C = \begin{vmatrix} c_{11} & c_{12} & c_{13} \\ c_{21} & c_{22} & c_{23} \\ -c_{31} & -c_{32} & c_{33} \end{vmatrix} , \end{aligned}$$

Download English Version:

https://daneshyari.com/en/article/5011289

Download Persian Version:

https://daneshyari.com/article/5011289

Daneshyari.com