Effects of environmental fluctuation on plankton allelopathy

P. K. Tapaswi[1], **A. Mukhopadhyay**[2]

[1]Embryology Research Unit, Indian Statistical Institute, 203, B. T. Road, Calcutta 700035, India. e-mail: tapaswi@isical.ac.in
[2]Kanailal Vidyamandir, Barobazar, Chandannagar, Pin 712136, India

Abstract. This paper studies a two-dimensional system that arises in plankton allelopathy involving discrete time delays and environmental fluctuations. The environmental parameters are assumed to be perturbed by white noise characterized by a Gaussian distribution with mean zero and unit spectral density. The dynamic behaviour of the stochastic system is studied and the fluctuations in population are measured both analytically and numerically by computer simulation.

Key words: Phytoplankton – Allelopathy – Time delay – Environmental fluctuation – White noise

1. Introduction

Fluctuations in size and density of many phytoplankton communities depend on several factors, some physical, others involving variation of necessary nutrients, or a combination of these. Several workers have noted that the increased population of one species of phytoplankton might affect the growth of one or several other species by the production of allelopathic toxins or stimulators, influencing bloom, pulses and seasonal succession. For detailed literature studies on allelopathic interactions in the phytoplanktonic world, see the elegant review of Hellebust (1974) and the book by Rice (1984).

Maynard Smith (1974) incorporated the effect of toxic substances in a two species Lotka–Volterra competitive system by assuming that each species produces a substance toxic to the other, but only when the other is present. Chattopadhyay (1996) investigated the stability

properties of the above system and Mukhopadhyay et al. (1998) sug-
gested that a species needs some time to mature before producing
a substance which will be toxic (or stimulatory) to the other, i.e., the
production of an allelopathic substance by the competing species will
not be instantaneous, but will occur after some time lag required for
maturity of the species.

Environmental fluctuation is an important component in an eco-
system. Most natural phenomena do not follow strictly deterministic
laws, but rather oscillate randomly about some average so that the
deterministic equilibrium is not an absolutely fixed state; instead it
is a "fuzzy" value around which the biological system fluctuates
(Renshaw, 1995). The environmental parameters are time dependent
randomly varying, and should be taken as stochastic. From this view-
point, we have modified our deterministic work (Mukopadhyay et al.,
1998) by incorporating a white noise which fluctuates with the environ-
mental carrying capacity in the growth regulating terms. In this
paper we study the plankton ecology with the help of a logistic model
with delayed allelopathic inhibition effect in a random fluctuating
environment.

2. The mathematical model

Maynard Smith (1974) modified the Lotka-Volterra two species com-
petition model by considering that each species produces a substance
toxic to the other, but only when the other is present. The modified
system can be written as:

$$\frac{dN_1}{dt} = N_1[K_1 - \alpha_1 N_1 - \beta_{12} N_2 - \gamma_1 N_1 N_2],$$

$$\frac{dN_2}{dt} = N_2[K_2 - \alpha_2 N_2 - \beta_{21} N_1 - \gamma_2 N_1 N_2],.$$

(2.1)

where $N_1(t)$, $N_2(t)$ are the population densities (number of cells per
litre) of two competing species; K_1, K_2 are the rates of cell proliferation
per hour; α_1, α_2 are the rates of intra-specific competition of the first
and second species respectively; β_{12}, β_{21} are the rates of inter-specific
competition of the first and second species respectively and $\frac{K_i}{\alpha_i}$ $(i = 1, 2)$
are environmental carrying capacities (representing the number of cells
per litre). The units of α_1, α_2, β_{12} and β_{21} are per hour per cell, and the
unit of time is in hours. γ_1 and γ_2 are the respective rates of toxic
inhibition of the first species by the second and vice versa.

It also seems reasonable to assume that the production of the toxic substance allelopathic to the competing species, simply termed "allelochemic", will not be instantaneous, but delayed by different discrete time lags required for the maturity of both species. The system of equations (2.1) is then reduced to

$$\frac{dN_1}{dt} = N_1[K_1 - \alpha_1 N_1 - \beta_{12} N_2 - \gamma_1 N_1 N_2(t - \tau_2)],$$

$$\frac{dN_2}{dt} = N_2[K_2 - \alpha_2 N_2 - \beta_{21} N_1 - \gamma_2 N_1(t - \tau_1) N_2], \tag{2.2}$$

where $\tau_i > 0$, $i = 1, 2$ are the times (in hours) required to produce the respective allelopathic inhibition effect of the first species on the second, and vice versa (Mukopadhyay et al., 1998).

Generalizing the result in Mukopadhyay et al. (1998) it can be shown that if $\frac{\alpha_i}{\beta_{ij}} > \max(\frac{\gamma_i}{\gamma_j}, \frac{K_i}{K_j})$, the system (2.2) has a unique positive equilibrium E^* which is always locally asymptotically stable for all $\tau_i \geq 0$. Hence it is evident that the allelopathic deterministic delay system does not undergo Hopf bifurcation and cannot evolve into a limit cycle exhibiting bloom, pulses and seasonal succession in the phytoplankton population.

We then consider the growth of the phytoplankton community in a stochastic environment incorporating a white noise in each of the equations the system (2.2). We investigate whether random fluctuation in the environment can drive the system into quasi-cyclic equilibrium as a result of non-zero constant fluctuation in the abundance of this aquatic population.

3. Basic equations

To take into account the influence of a random environment, we modify the delay system (2.2) to the form

$$\frac{dN_1}{dt} = N_1[K_1 + \xi_1(t) - \alpha_1 N_1 - \beta_{12} N_2 - \gamma_1 N_1 N_2(t - \tau_2)],$$

$$\frac{dN_2}{dt} = N_2[K_2 + \xi_2(t) - \alpha_2 N_2 - \beta_{21} N_1 - \gamma_2 N_1(t - \tau_1) N_2], \tag{3.1}$$

where the environmental parameters have been perturbed by standard white noise characterized by

$$\overline{\xi_i(t)} = 0 \quad \text{and} \quad \overline{\xi_i(t)\xi_j(t')} = \delta_{ij}\delta(t - t') \quad i, j = 1, 2. \tag{3.2}$$

The overbar in equation (3.2) denotes the ensemble average due to the effect of the randomly fluctuating environment, δ_{ij} is the Kronecker delta (representing the spectral density of the white noise) and δ is the Dirac delta function with t and t' being distinct times.

Substituting $N_i(t) = \exp(x_i(t))$, the system (3.1) reduces to the form

$$\frac{dx_1}{dt} = [K_1 - \alpha_1 \exp(x_1(t)) - \beta_{12} \exp(x_2(t))$$

$$- \gamma_1 \exp(x_1(t)) \exp(x_2(t - \tau_2))] + \xi_1(t),$$

$$\frac{dx_2}{dt} = [K_2 - \alpha_2 \exp(x_2(t)) - \beta_{21} \exp(x_1(t))$$
$$(3.3)$$

$$- \gamma_2 \exp(x_1(t - \tau_1)) \exp(x_2(t))] + \xi_2(t).$$

To analyze the behaviour of the system around the non-zero steady state solution $(N_1^*, N_2^*) = (\exp(x_1^*), \exp(x_2^*))$ of (2.2) we substitute

$$x_i(t) = x_i^* + \eta_i(t) \tag{3.4}$$

in (3.3), reducing the equations to the form

$$\frac{d\eta_1}{dt} = [\xi_1(t) - (\alpha_1 \exp(x_1^*) + \gamma_1 \exp(x_1^* + x_2^*))\eta_1(t) - \beta_{12} \exp(x_2^*)\eta_2(t)$$

$$- \gamma_1 \exp(x_1^* + x_2^*)\eta_2(t - \tau_2)],$$

$$\frac{d\eta_2}{dt} = [\xi_2(t) - \beta_{21} \exp(x_1^*)\eta_1(t) - (\alpha_2 \exp(x_2^*) + \gamma_2 \exp(x_1^* + x_2^*))\eta_2(t)$$

$$- \gamma_2 \exp(x_1^* + x_2^*)\eta_1(t - \tau_1)], \tag{3.5}$$

(to a first order approximation).

Hence the linearized system without delays around the interior equilibrium $(N_1^*, N_2^*) = (\exp(x_1^*), \exp(x_2^*))$ reduces to the equations

$$\frac{d\eta_1}{dt} = A\eta_1 + B\eta_2 + \xi_1(t),$$

$$\frac{d\eta_2}{dt} = C\eta_1 + D\eta_2 + \xi_2(t),$$
$$(3.6)$$

where

$$A = -(\alpha_1 + \gamma_1 \exp(x_2^*)) \exp(x_1^*),$$

$$B = -(\beta_{12} + \gamma_1 \exp(x_1^*)) \exp(x_2^*),$$

$$C = -(\beta_{21} + \gamma_2 \exp(x_2^*)) \exp(x_1^*),$$
$$(3.7)$$

$$D = -(\alpha_2 + \gamma_2 \exp(x_1^*)) \exp(x_2^*).$$

Note that A, B, C, D are all negative as $\gamma_i > 0, i = 1, 2$.

The corresponding characteristic equation of the system (3.6) without environmental fluctuation is given by

$$\Delta(\lambda) \equiv \lambda^2 - (A + D)\lambda + (AD - BC) = 0, \tag{3.8}$$

and the corresponding characteristic roots are

$$\lambda = \tfrac{1}{2}\{(A + D) \pm \sqrt{(A + D)^2 - (AD - BC)}\}. \tag{3.9}$$

Here $(A + D) < 0$ and $(A + D)^2 - (AD - BC) > 0$. Since $AD - BC > 0$, which is a requisite condition for existence of the non zero interior equilibrium, there exist two negative real values of λ for which the system is locally asymptotically stable.

3.1. The system without time delays

Consider the system (3.1) when $\tau_i = 0$, $i = 1, 2$ and write the Langevin equations (3.6) in the matrix form

$$\dot{\mathbf{X}}(t) = \mathbf{P}\mathbf{X}(t) + \mathbf{I}\xi(t), \tag{3.10}$$

where $\dot{\mathbf{X}}(t)$ represents the derivative with respect to time t, $\mathbf{I}$ is the unit matrix and

$$\mathbf{X} = \begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix}, \qquad \mathbf{P} = \begin{pmatrix} A & C \\ B & D \end{pmatrix}, \qquad \xi = \begin{pmatrix} \xi_1(t) \\ \xi_2(t) \end{pmatrix}. \tag{3.11}$$

The solution of the above equation, with the initial condition describing a sharp value $\mathbf{X}_0$ at $t = 0$, is given by

$$\mathbf{X}(t) = e^{\mathbf{P}t}\mathbf{X}_0 + \int_0^t e^{\mathbf{P}(t - t')}\mathbf{I}\xi(t')\,dt'. \tag{3.12}$$

Here we have assumed that the system started off at the sharp (corresponding to the delta function initial condition in the associated Fokker–Plank equation) phase point

$$\mathbf{X}_0 = \mathbf{X}(0) = (\eta_1(0), \eta_2(0)), \tag{3.13}$$

and $e^{\mathbf{P}t}$ from the matrix equation (3.12) is given by

$$e^{\mathbf{P}t} = L^{-1}\{(s\mathbf{I} - \mathbf{P})^{-1}\}, \tag{3.14}$$

with $L\{f(t)\} = \int_0^\infty e^{-st}f(t)\,dt$ as the Laplace transform of $f(t)$, $\mathbf{I}$ as the identity matrix and L^{-1} as the inverse Laplace transform (Coffey et al., 1997). Now,

$$(s\mathbf{I} - \mathbf{P}) = \begin{pmatrix} s - A & B \\ C & s - D \end{pmatrix},$$

and

$$\det(s\mathbf{I} - \mathbf{P}) \equiv \Delta(s) = s^2 - (A + D)s + (AD - BC).$$

Let λ_1, λ_2 (assuming that $\lambda_1 > \lambda_2$) be the roots (3.8), then

$$\Delta(s) = s^2 - (\lambda_1 + \lambda_2)s + \lambda_1\lambda_2 = \Delta \text{ (say) and}$$

$$(s\mathbf{I} - \mathbf{P})^{-1} = \frac{1}{\Delta}\begin{pmatrix} s - D & -B \\ -C & s - A \end{pmatrix}. \tag{3.15}$$

Now,

$$L^{-1}\left(\frac{s - D}{\Delta}\right) = e^{((\lambda_1 + \lambda_2)/2)t}\cosh\frac{(\lambda_1 - \lambda_2)}{2}t$$

$$+ \frac{(A - D)}{(\lambda_1 - \lambda_2)}e^{((\lambda_1 + \lambda_2)/2)t}\sinh\frac{(\lambda_1 - \lambda_2)}{2}t,$$

$$L^{-1}\left(\frac{s - A}{\Delta}\right) = e^{((\lambda_1 + \lambda_2)/2)t}\cosh\frac{(\lambda_1 - \lambda_2)}{2}t$$

$$\tag{3.16}$$

$$- \frac{(A - D)}{(\lambda_1 - \lambda_2)}e^{((\lambda_1 + \lambda_2)/2)t}\sinh\frac{(\lambda_1 - \lambda_2)}{2}t,$$

$$L^{-1}\left(\frac{-B}{\Delta}\right) = -\frac{B}{(\lambda_1 - \lambda_2)}e^{((\lambda_1 + \lambda_2)/2)t}\sinh\frac{(\lambda_1 - \lambda_2)}{2}t,$$

$$L^{-1}\left(\frac{-C}{\Delta}\right) = -\frac{C}{(\lambda_1 - \lambda_2)}e^{((\lambda_1 + \lambda_2)/2)t}\sinh\frac{(\lambda_1 - \lambda_2)}{2}t.$$

So, on simplification, we have

$$e^{\mathbf{P}t} = L^{-1}(s\mathbf{I} - \mathbf{P})^{-1} = \begin{pmatrix} (\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t}) & \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t}) \\ \rho_4(e^{\lambda_1 t} - e^{\lambda_2 t}) & (\rho_2 e^{\lambda_1 t} + \rho_1 e^{\lambda_2 t}) \end{pmatrix}, \tag{3.17}$$

where

$$\rho_1 = \frac{1}{2}\left(1 + \frac{A - D}{\lambda_1 - \lambda_2}\right),$$

$$\rho_2 = \frac{1}{2}\left(1 - \frac{A - D}{\lambda_1 - \lambda_2}\right),$$

$$\tag{3.18}$$

$$\rho_3 = \frac{-B}{\lambda_1 - \lambda_2},$$

$$\rho_4 = \frac{-C}{\lambda_1 - \lambda_2}.$$

Therefore, we may write

$$\begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} = \begin{pmatrix} (\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t}) & \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t}) \\ \rho_4(e^{\lambda_1 t} - e^{\lambda_2 t}) & (\rho_2 e^{\lambda_1 t} + \rho_1 e^{\lambda_2 t}) \end{pmatrix} \begin{pmatrix} \eta_1(0) \\ \eta_2(0) \end{pmatrix}$$

$$+ \int_0^t \begin{pmatrix} (\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t}) & \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t}) \\ \rho_4(e^{\lambda_1 t} - e^{\lambda_2 t}) & (\rho_2 e^{\lambda_1 t} + \rho_1 e^{\lambda_2 t}) \end{pmatrix} \begin{pmatrix} \xi_1(t') \\ \xi_2(t') \end{pmatrix} dt'. \quad (3.19)$$

Taking the averages over the stochastic variable $\xi(t)$ and noting from (3.2) that $\overline{\xi_i(t)} = 0, i = 1, 2$ we obtain

$$\overline{X(t)} = e^{P t} X_0. \quad (3.20)$$

Using the fact that $\overline{\xi_i(t')\xi_j(t'')} = \delta_{ij}\delta(t' - t'')$ we have

$$\overline{\eta_1^2(t)} = [(\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t})\eta_1(0) + \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t})\eta_2(0)]^2$$

$$+ 2[(\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t})\eta_1(0) + \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t})\eta_2(0)]$$

$$\times \int_0^t [(\rho_1 e^{\lambda_1(t-t')} + \rho_2 e^{\lambda_2(t-t')})\overline{\xi_1(t')} + \rho_3(e^{\lambda_1(t-t')} - e^{\lambda_2(t-t')})\overline{\xi_2(t')}] dt'$$

$$+ \int_0^t [(\rho_1 e^{\lambda_1(t-t')} + \rho_2 e^{\lambda_2(t-t')})\overline{\xi_1(t')}$$

$$+ \rho_3(e^{\lambda_1(t-t')} - e^{\lambda_2(t-t')})\overline{\xi_2(t')}] dt' \int_0^t [(\rho_1 e^{\lambda_1(t-t')}$$

$$+ \rho_2 e^{\lambda_2(t-t')})\overline{\xi_1(t'')} + \rho_3(e^{\lambda_1(t-t')} - e^{\lambda_2(t-t')})\overline{\xi_2(t'')}] dt''$$

$$= [(\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t})\eta_1(0) + \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t})\eta_2(0)]^2$$

$$+ \int_0^t \int_0^t [\{(\rho_1 e^{\lambda_1(t-t')} + \rho_2 e^{\lambda_2(t-t')})\}\{(\rho_1 e^{\lambda_1(t-t')} + \rho_2 e^{\lambda_2(t-t')})\}$$

$$+ \{\rho_3(e^{\lambda_1(t-t')} - e^{\lambda_2(t-t')})\}\{\rho_3(e^{\lambda_1(t-t')} - e^{\lambda_2(t-t')})\}]\delta(t' - t'') dt' dt'',$$

$$= [(\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t})\eta_1(0) + \rho_3(e^{\lambda_1 t} - e^{\lambda_2 t})\eta_2(0)]^2$$

$$- \left[\frac{\rho_1^2}{2\lambda_1}(1 - e^{2\lambda_1 t}) + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) \right.$$

$$+ \frac{\rho_2^2}{2\lambda_2}(1 - e^{2\lambda_2 t}) + \rho_3^2 \left\{ \frac{1}{2\lambda_1}(1 - e^{2\lambda_1 t}) - \frac{2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) \right.$$

$$+ \left. \left. \frac{1}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right\} \right], \quad (3.21)$$

by using the properties of the Dirac delta function i.e., $\int_p^q f(t)\delta(t-a)\,dt = f(a),\; p < a < q$.

Similarly,

$$\overline{\eta_2^2(t)} = [\rho_4(e^{\lambda_1 t} - e^{\lambda_2 t})\eta_1(0) + (\rho_1 e^{\lambda_1 t} + \rho_2 e^{\lambda_2 t})\eta_2(0)]^2$$

$$- \left[\frac{\rho_2^2}{2\lambda_1}(1 - e^{2\lambda_1 t}) + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) + \frac{\rho_1^2}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right.$$

$$+ \rho_4^2 \left\{ \frac{1}{2\lambda_1}(1 - e^{2\lambda_1 t}) - \frac{2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) \right.$$

$$\left. \left. + \frac{1}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right\} \right]. \tag{3.22}$$

So the population variances are given by

$$\sigma_{\eta_1}^2 = \overline{\eta_1^2(t)} - (\bar\eta_1(t))^2$$

$$= - \left[\frac{\rho_1^2}{2\lambda_1}(1 - e^{2\lambda_1 t}) + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) + \frac{\rho_2^2}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right.$$

$$+ \rho_3^2 \left\{ \frac{1}{2\lambda_1}(1 - e^{2\lambda_1 t}) - \frac{2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) \right.$$

$$\left. \left. + \frac{1}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right\} \right], \tag{3.23a}$$

$$\sigma_{\eta_2}^2 = \overline{\eta_2^2(t)} - (\bar\eta_2(t))^2$$

$$= - \left[\frac{\rho_2^2}{2\lambda_1}(1 - e^{2\lambda_1 t}) + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) + \frac{\rho_1^2}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right.$$

$$+ \rho_4^2 \left\{ \frac{1}{2\lambda_1}(1 - e^{2\lambda_1 t}) - \frac{2}{(\lambda_1 + \lambda_2)}(1 - e^{(\lambda_1 + \lambda_2)t}) \right.$$

$$\left. \left. + \frac{1}{2\lambda_2}(1 - e^{2\lambda_2 t}) \right\} \right]. \tag{3.23b}$$

Each population variance is an increasing function of t, reaching a steady state value as $t \to +\infty$ given by

$$\sigma_{\eta_1}^2 = - \left[\frac{\rho_1^2}{2\lambda_1} + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)} + \frac{\rho_2^2}{2\lambda_2} + \rho_3^2 \left\{ \frac{1}{2\lambda_1} - \frac{2}{(\lambda_1 + \lambda_2)} + \frac{1}{2\lambda_2} \right\} \right].$$

$$\tag{3.24a}$$

$$\sigma_{\eta_2}^2 = -\left[\frac{\rho_2^2}{2\lambda_1} + \frac{2\rho_1\rho_2}{(\lambda_1 + \lambda_2)} + \frac{\rho_1^2}{2\lambda_2} + \rho_4^2\left\{\frac{1}{2\lambda_1} - \frac{2}{(\lambda_1 + \lambda_2)} + \frac{1}{2\lambda_2}\right\}\right].$$

(3.24b)

Substituting $\rho_i(i = 1 \cdots 4)$ from (3.18) in (3.24) gives

$$\sigma_{\eta_1}^2 = -\left[\frac{B^2 + D^2 + (AD - BC)}{2(A + D)(AD - BC)}\right],$$

(3.25a)

$$\sigma_{\eta_2}^2 = -\left[\frac{A^2 + C^2 + (AD - BC)}{2(A + D)(AD - BC)}\right],$$

(3.25b)

in terms of the original parameters. Details of the calculations involved are available to interested readers.

3.2. The system with time delays

When time delays are present in the system, i.e., $\tau_i > 0$, $i = 1, 2$, it is not possible to obtain explicit expressions of the variances by the above method. Instead, we proceed to obtain the spectral density of each population by Fourier transform methods.

3.2.1. Fourier transforms: spectral density

Let us consider a continuous function $\mathbf{Y}(t)$, known over the interval $-T/2 \leq t \leq T/2$, and define another function $\tilde{\mathbf{Y}}(\omega)$ which is related to it by

$$\tilde{\mathbf{Y}}(\omega) = \int_{-T/2}^{T/2} \mathbf{Y}(t)e^{-i\omega t}\,dt.$$

(3.26)

As $\tilde{\mathbf{Y}}(\omega)$ is the Fourier transform of $\mathbf{Y}(t)$, we know that

$$\mathbf{Y}(t) = \frac{1}{2\pi}\int_{-\infty}^{\infty}\tilde{\mathbf{Y}}(\omega)e^{i\omega t}\,d\omega.$$

(3.27)

Equation (3.27) implies that $\frac{1}{2\pi}\tilde{\mathbf{Y}}(\omega)$ is the amplitude density of the components of $\mathbf{Y}(t)$ in the angular frequency interval ω to $\omega + d\omega$. Thus $\frac{1}{2\pi}\tilde{\mathbf{Y}}(\omega)\,d\omega$ is a crude estimate of the amplitude of the component of $\mathbf{Y}(t)$ with angular frequency ω (Nisbet and Gurney, 1982).

So, the Fourier transforms of the system equations (3.5) can be written as

$$\tilde{\xi}_1(\omega) = r_{11}\tilde{\eta}_1(\omega) + r_{12}\tilde{\eta}_2(\omega),$$

$$\tilde{\xi}_2(\omega) = r_{21}\tilde{\eta}_1(\omega) + r_{22}\tilde{\eta}_2(\omega),$$

(3.28)

where

$$\int_{-\infty}^{\infty} \xi_i(t - \tau_i)\exp(-i\omega t)\,dt = \exp(-i\omega\tau_i)\tilde{\xi}_i(\omega) \quad i = 1, 2, \qquad (3.29)$$

$$
\begin{aligned}
r_{11} &= -A + i\omega, \\
r_{12} &= -B + (\exp(-i\omega\tau_2) - 1)\gamma_1 \exp(x_1^* + x_2^*), \\
r_{21} &= -C + (\exp(-i\omega\tau_1) - 1)\gamma_2 \exp(x_1^* + x_2^*), \\
r_{22} &= -D + i\omega,
\end{aligned}
\qquad (3.30)
$$

and A, B, C, D are as in (3.7). The system equations (3.28) can be written in the matrix form

$$\mathbf{R}\tilde{\eta} = \tilde{\xi}, \qquad (3.31)$$

where

$$\mathbf{R} = \begin{pmatrix} r_{11} & r_{12} \\ r_{21} & r_{22} \end{pmatrix}, \qquad \tilde{\eta} = \begin{pmatrix} \tilde{\eta}_1 \\ \tilde{\eta}_2 \end{pmatrix}, \qquad \tilde{\xi} = \begin{pmatrix} \tilde{\xi}_1 \\ \tilde{\xi}_2 \end{pmatrix}, \qquad (3.32)$$

and $\tilde{\eta}$ and $\tilde{\xi}$ are the Fourier transforms of η and ξ respectively.

Let

$$R^{-1} = \begin{pmatrix} \kappa_{11} & \kappa_{12} \\ \kappa_{21} & \kappa_{22} \end{pmatrix}; \qquad (3.33)$$

then

$$\tilde{\eta}_i = \sum_{j=1}^{2} \kappa_{ij}\tilde{\xi}_j \quad i = 1, 2. \qquad (3.34)$$

If the function $\mathbf{Y}(t)$ has zero mean value then the fluctuation intensity (variance) of the components in the frequency band ω and $\omega + d\omega$ is $S_Y(\omega)d\omega$ where the spectral density $S_Y(\omega)$ is formally defined, as in Nisbet and Gurney (1982), by

$$S_Y(\omega)d\omega = \lim_{T \to \infty} \frac{\overline{|\tilde{\mathbf{Y}}(\omega)|^2}}{T}.$$

Hence,

$$
\begin{aligned}
S_\xi(\omega)d\omega &= \lim_{T \to \infty} \frac{\overline{|\tilde{\xi}(\omega)|^2}}{T} \\
&= \lim_{T \to \infty} \frac{1}{T} \int_{-T/2}^{T/2} \int_{-T/2}^{T/2} \overline{\xi(t)\,\xi(t')} \exp(i\omega(t' - t))\,dt\,dt'. \quad (3.35)
\end{aligned}
$$

Therefore, from (3.34) and (3.35), we have

$$S_{\eta_i}(\omega) = \sum_{j=1}^{2} |\kappa_{ij}|^2 S_{\xi_j}(\omega), \quad i = 1, 2, \qquad (3.36)$$

because $\overline{\xi_i(t)} = 0$ and $\overline{\xi_i(t)\xi_j(t')} = \delta_{ij}\delta(t - t')$. Therefore the fluctuation intensity (variance) in η_i is given by Nisbet and Gurney (1982) as

$$\sigma_{\eta_i}^2 = \frac{1}{2\pi} \int_{-\infty}^{\infty} S_{\eta_i}(\omega)\,d\omega$$

$$= \frac{1}{2\pi} \sum_{j=1}^{2} \int_{-\infty}^{\infty} |\kappa_{ij}|^2 S_{\xi_i}(\omega)\,d\omega$$

$$= \frac{1}{2\pi} \sum_{j=1}^{2} \int_{-\infty}^{\infty} |\kappa_{ij}|^2\,d\omega, \quad i = 1, 2, \qquad (3.37)$$

because $S_{\xi_i}(\omega) = 1$.

Proceeding as above, after some tedious calculations, the population fluctuation intensity (variance) in η_i is given by

$$\sigma_{\eta_i}^2 = \frac{1}{2\pi} \int_{-\infty}^{\infty} \frac{P_i(\omega)}{M(\omega)}\,d\omega, i = 1, 2, \qquad (3.38)$$

where

$$P_1(\omega) = \omega^2 + D^2 + \{-B + \gamma_1 \exp(x_1^* + x_2^*)(\cos\omega\tau_2 - 1)\}^2$$
$$+ \{\gamma_1 \exp(x_1^* + x_2^*)\sin\omega\tau_2\}^2,$$

$$P_2(\omega) = \omega^2 + A^2 + \{-C + \gamma_2 \exp(x_1^* + x_2^*)(\cos\omega\tau_1 - 1)\}^2$$
$$+ \{\gamma_2 \exp(x_1^* + x_2^*)\sin\omega\tau_1\}^2,$$

$$M(\omega) = [-\omega^2 + (AD - BC) + \gamma_1 \exp(x_1^* + x_2^*)C(\cos\omega\tau_2 - 1)$$
$$+ \gamma_2 \exp(x_1^* + x_2^*) B(\cos\omega\tau_1 - 1) + \gamma_1\gamma_2 e^{2(x_1^* + x_2^*)}$$
$$\times \{\cos\omega(\tau_1 + \tau_2) - \cos\omega\tau_1 - \cos\omega\tau_2 + 1\}]^2$$
$$+ [\omega(A + D) + \gamma_1 \exp(x_1^* + x_2^*)C\sin\omega\tau_2$$
$$+ \gamma_2 \exp(x_1^* + x_2^*)B \sin\omega\tau_1 + \gamma_1\gamma_2 e^{2(x_1^* + x_2^*)}$$
$$\times \{\sin\omega(\tau_1 + \tau_2) - \sin\omega\tau_1 - \sin\omega\tau_2\}]^2. \qquad (3.39)$$

When $\tau_1 = \tau_2 = 0$, we have

$$P_1(\omega) = \omega^2 + B^2 + D^2,$$

$$P_2(\omega) = \omega^2 + A^2 + C^2,$$

$$M(\omega) = [-\omega^2 + (AD - BC)]^2 + (A + D)^2\omega^2$$
$$= [-\omega^2 + i(A + D)\omega + (AD - BC)]$$
$$\times [-\omega^2 - i(A + D)\omega + (AD - BC)]. \qquad (3.40)$$

Following Gradshteyn and Ryzhik (1980), the general integral encountered in calculations of fluctuation is of the type

$$I_n = \int_{-\infty}^{\infty} \frac{g_n(\omega)d\omega}{h_n(\omega)\, h_n(-\omega)}\,, \tag{3.41}$$

where

$$g_n(\omega) = b_0\omega^{2n-2} + b_1\omega^{2n-4} + \cdots + b_{n-1},$$
$$h_n(\omega) = a_0\omega^n + a_1\omega^{n-1} + \cdots + a_n. \tag{3.42}$$

When $n = 2$ the integral is given by

$$I_2 = \frac{\pi i(a_0 b_1 - a_2 b_0)}{a_0 a_1 a_2}. \tag{3.43}$$

Thus we identify $g_2(\omega)$ with $P_i(\omega), (i = 1, 2)$ and $h_2(\omega)\, h_2(-\omega)$ with $M(\omega)$ to obtain

$$a_0 = -1, \qquad a_1 = A + D, \qquad a_2 = AD - BC,$$
$$b_0(1) = 1, \qquad b_1(1) = B^2 + D^2, \tag{3.44}$$
$$b_0(2) = 1, \qquad b_1(2) = A^2 + C^2.$$

Hence,

$$\sigma_{\eta_1}^2 = -\left[\frac{B^2 + D^2 + (AD - BC)}{2(A + D)(AD - BC)}\right], \tag{3.45a}$$

$$\sigma_{\eta_2}^2 = -\left[\frac{A^2 + C^2 + (AD - BC)}{2(A + D)(AD - BC)}\right]. \tag{3.45b}$$

This result agrees with the previous one obtained by elementary means in Sect. 3.1. Explicit values of the spectral densities of the populations when $\tau_i > 0$ are difficult to obtain, as evaluation of the integrals (3.38) is a formidable task. But we can show numerically that increasing the time delays $\tau_i, i = 1, 2$, in the system increases the intensity of fluctuation of the populations (Fig. 1(a)–(b)).

4. Artificial realization

To gain a broad understanding of the dynamic behaviour of the allelopathic system we construct a number of individual realizations of the birth and death type process described by equation (3.1), using the following procedure (Nisbet and Gurney, 1982).
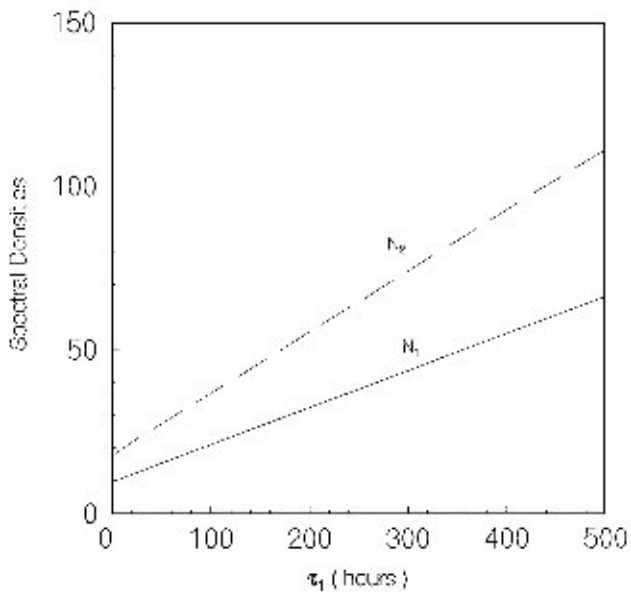
(i) An initial population size $N_i(0)$ is chosen;

Fig. 1a. Variation of spectral densities of the populations with τ_1 when $\tau_2 = 0$. Parameter values are $K_1 = 0.2$, $K_2 = 0.1$, $\alpha_1 = 0.007$, $\alpha_2 = 0.008$, $\beta_{12} = 0.005$, $\beta_{21} = 0.0015$, $\gamma_1 = 0.008$, $\gamma_2 = 0.005$ which satisfy the criteria for the existence of unique nonzero equilibrium.
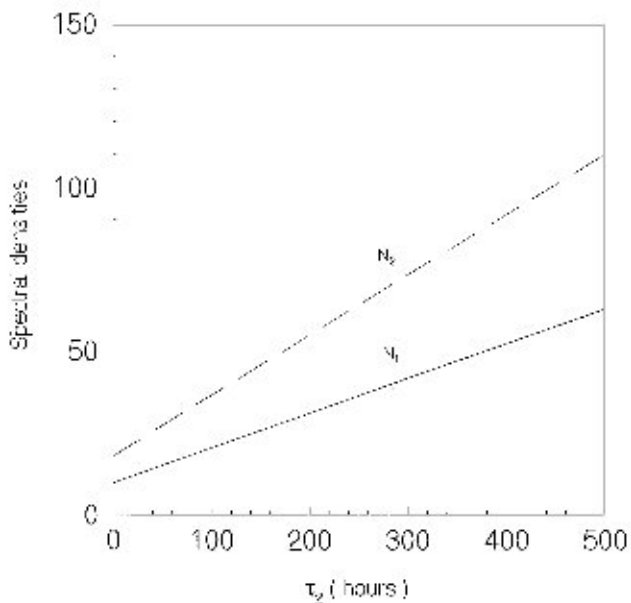

Fig. 1b. Variation of spectral densities of the populations with τ_2 when $\tau_1 = 0$. Parameter values are same as in Fig. 1a.

(ii) A time increment dt is chosen such that the birth rate $B_i(N_i)\, dt \ll 1$ and the death rate $D_i(N_1, N_2)\, dt \ll 1$ over the entire range of population;

(iii) A random number generator is used to generate two sets of a sequence of random numbers from a normal distribution with zero mean and variance equal to $\frac{1}{dt}$ (representing a white noise of unit spectral density);

(iv) At each time, obtain the next random number (R_i) from the sequence and perform the following operations:

(a) If $R_i < B_i(N_i)\, dt$, change $N_i \to N_i + 1$,

(b) If $R_i > 1 - D_i(N_1, N_2)\, dt$, change $N_i \to N_i - 1$,

(c) If $B_i(N_i)\, dt \leqq R_i \leqq 1 - D_i(N_1, N_2)$ dt, do not change N_i;

(v) Proceed from t to $t + dt$ and repeat step (iv).

In our case,

$$B_i(N_i) = K_i N_i$$

and

$$D_i(N_1, N_2) = N_i\{\alpha_i N_i + \beta_{ij} N_j + \gamma_i N_i N_j(t - \tau_j)\} \quad i, j = 1, 2\,(i \neq j).$$

Then proceeding as above, we obtain the results of the realization as depicted in Figs. 2–4. These show the combined effects of
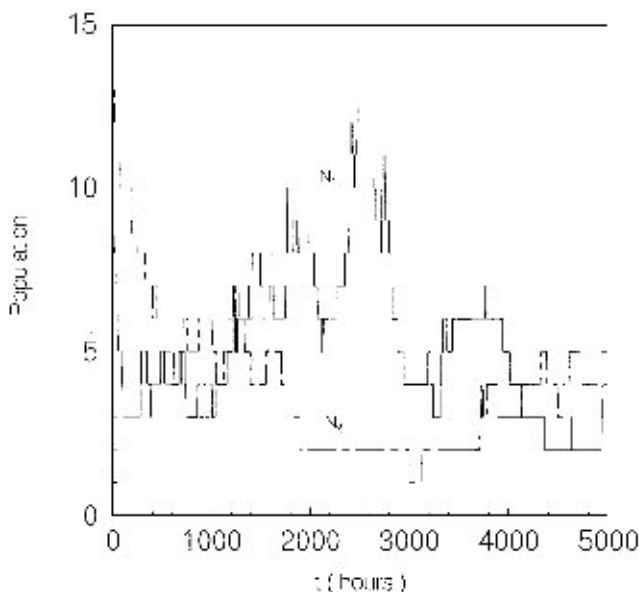
Fig. 2. Realization of the allelopathic inhibitory (AI) system exhibiting a statistically stationary state implying persistence of both the species for $\tau_1 = \tau_2 = 0$, with parameter values as in Fig. 1a.
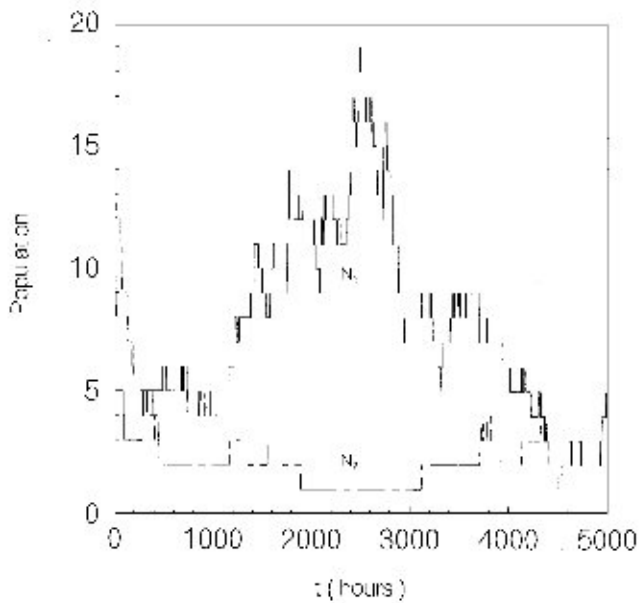
Fig. 3a. Stochastic realization of the AI model showing coexistence of the two species upto the maximum values of $\tau_1 (\tau_1 = 1344$ hours$)$ $\tau_2 = 0$. Parameter values are same as in the previous figures.
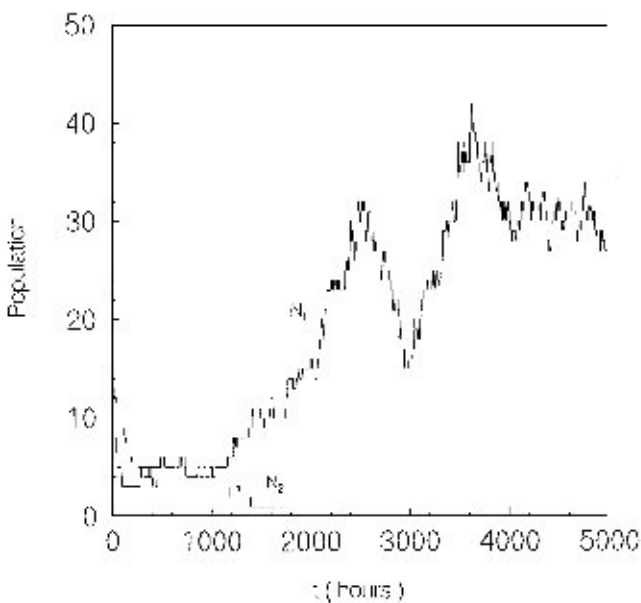

Fig. 3b. Stochastic realization of the AI model showing extinction of the second species (N_2) at the bifurcation value of τ_1 ($\tau_1 = 1345$ hours, i.e., approx. 57 days) when $\tau_2 = 0$. Parameter values are same as in the previous figures.
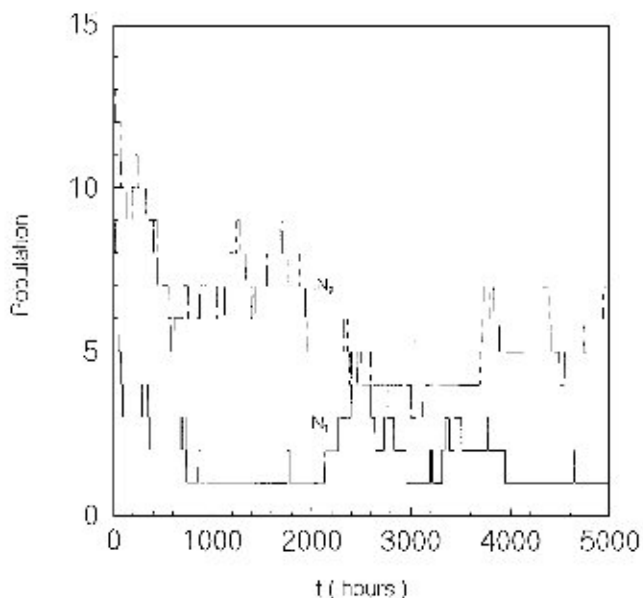
Fig. 4a. Stochastic realization of the AI model showing persistence of both the species (coexistence) upto the maximum value of τ_2 ($\tau_2 = 2451$ hours), when $\tau_1 = 0$. Parameter values are as in Fig. 1a.
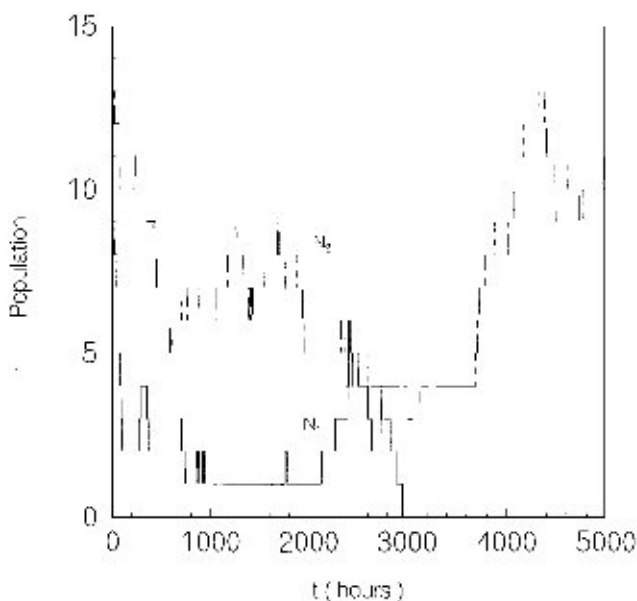

Fig. 4b. Solution (by realization) of the AI model bifurcating from stability to instability leading to extinction of the species N_1 at $\tau_2 = 2452$ hours (i.e., approx. 103 days) when $\tau_1 = 0$. Parameter values are as in previous figures.
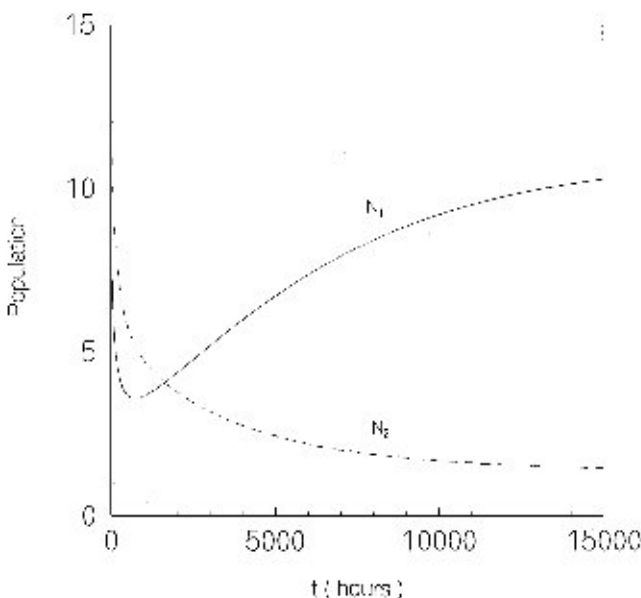
Fig. 5. Solution of the analogous deterministic AI model approaching a stable station-ary state for $\tau_1 = \tau_2 = 0$. Similar type of stable solutions for large t have been obtained for all $\tau_i > 0$. Parameter values are same as in Fig. 1a.

environmental fluctuation and of $\tau_i \geq 0$ on the persistence of the two species, for the parameter values $K_1 = 0.2$, $K_2 = 0.1$, $\alpha_1 = 0.007$, $\alpha_2 = 0.008$, $\beta_{12} = 0.005$, $\beta_{21} = 0.0015$, $\gamma_1 = 0.008$, and $\gamma_2 = 0.005$. A discussion of the appropriateness of parameter values in plankton allelopathy follows in Sect. 5. Figure 5 shows the dynamic behaviour of the analogous deterministic system (2.2), in which the coexistence of the species (i.e., the stability of the non-zero equilibrium) is shown to remain unaffected for all pairs of values of τ_i, as was also observed analytically in the local stability analysis.

When $\tau_i = 0$, environmental fluctuation contributes to fluctuation in the density of populations only, but cannot produce any drastic change in the steady-state dynamic properties of the allelopathic sys-tem as regards to the coexistence of the species. That is to say, the unique non-zero equilibrium, if it exists, is always asymptotically stable in the global sense as depicted by Fig. 2, as it was in the analogous deterministic system. On the other hand, it is observed (Figs. 3–4) that for each $0 \leq \tau_i \leq M_i$, there exists a bifurcation point $\tau_j \geq m_j$, $(i, j = 1, 2; i \neq j)$ at which the non-zero equilibrium becomes unstable, leading to extinction of the ith species in the stochastic system, contrary

to the analogous deterministic case. For example, from Figs. 3b, 4b, we see that $(\tau_1, \tau_2) = (1345, 0)$ and $(\tau_1, \tau_2) = (0, 2452)$ are two such pairs of bifurcation points (axial points on the (τ_1, τ_2)-plain) where one of the species becomes extinct.

5. Discussion

Deterministic models in ecology do not usually incorporate environmental fluctuation; they are often justified by the implicit assumption that in large populations, stochastic deviations are small enough to be ignored. Unfortunately one cannot ascertain, except by a parallel simulation experiments, the ultimate size of the population at or above which the deterministic approximations are reasonable. A stochastic model provides a more realistic picture of a natural system than its deterministic counterpart. Deterministic models will prove ecologically useful only if the dynamical patterns they reveal are still in evidence when stochastic effects are introduced.

Random variation will cause stochastic fluctuations in the solutions with constant variations. This superimposed noise will destroy the fine structure of the deterministic solutions. In reality, random fluctuations are always present, hence the use of deterministic models exhibiting substantially different dynamical behaviour from their stochastic counterparts is not appropriate.

Our study models the stochastic behaviour of a system with discrete time lags in its allelopathic effect in a random fluctuating environment characterized by white noise. The system in a random environment develops significant fluctuations, and the stochastic system persists with the coexistence of both the species when the allelopathic effect is instantaneous, that is $\tau_1 = \tau_2 = 0$ (Fig. 2); in this behaviour it follows its deterministic analogue (Fig. 5). The steady state of the stochastic system in this case is not absolutely fixed, but rather a "fuzzy" value around which the population fluctuates.

On the other hand, unlike its deterministic counterpart where a unique non-zero equilibrium is observed to be stable (implying persistence of both the species for all $\tau_i \geq 0$ (Fig. 5)), the stochastic system with environmental fluctuation shows instability of the unique non-zero equilibrium (implying extinction of the ith species at some $\tau_j \geq m_j$ corresponding to each $0 \leq \tau_i \leq M_i$, $(i = 1, 2)$).

Spectral density, denoting the intensity of fluctuation of each population as studied in this paper, has also been observed to be an increasing function of each time lag $\tau_i (i = 1, 2)$ (Fig. 1(a)–(b)). This shows that fluctuation in the density of each population increases

as the maturity time of the allelochemics increases, this increasing fluctuation drives the ith species to extinction at and above some threshold value of $\tau_j \geqq m_j$, called the bifurcation point, when there is an environmental fluctuation in the allelopathic inhibitory system (2.2).

For the numerical analysis, the values of the growth terms K_1 and K_2 are set to 0.2 and 0.1 respectively. This means that the growth term has been assumed to be respectively 2 and 1 cell division over a 10 hour period, for the carrying capacity $\frac{K_1}{\alpha_1} \approx 30$ and $\frac{K_2}{\alpha_2} = 12.5$ respectively. These values represent approximately 14,000 and 6,000 cells per litre, which are the respective theoretical maximum densities of the two species. Other parameters such as crowding (intra-species competition) coefficients, inter-species competition coefficients, allelopathic (inhibiting) coefficients $\alpha_i, \beta_{ij}, \gamma_i (i, j = 1, 2, i \neq j)$, respectively, have been chosen appropriately. The values of these parameters can be estimated from experimental observations and data collection of cell counts over a long period. The numerical analysis and artificial realization presented here are based on a purely hypothetical set of parameters. The aim of the study is to investigate the effect of environmental fluctuation introduced in a two-species allelopathically inhibiting system in the form of a white noise, characterized by a Gaussian distribution with zero mean and unit spectral density. It is observed that environmental fluctuation cannot change the dynamics of the analogous deterministic system appreciably in the absence of both the time delays $\tau_i, (i = 1, 2)$, to maturity of the allelochemics. Hence deterministic models are quite valid in this case for describing the approximate dynamic behaviour of the system.

On the other hand, for certain values of $\tau_i > 0$, the time-delay environmentally fluctuating stochastic allelopathic system exhibits an altogether different dynamical picture from its deterministic counterpart: the species becomes extinct as $t \to \infty$. Hence, deterministic models in the latter case are not quite appropriate, and may ultimately be misleading.

The model would be more realistic if seasonal variation of the fluctuating environment were considered by replacing K_i by $K_i[1 + K_i' \cos t/6 + \xi_i(t)]$, where K_i' regulates the amplitude of the annual oscillation, t measures time in months and $\xi_i(t)$ introduces the stochastic element as a white noise in the frequency term. Although introducing this modification in the allelopathic model (2.2) would make the analytic study of the model a formidable task, numerical studies using the method discussed in Sect. 4 are worth pursuing. However, the simple stochastic model (3.1) studied as an analogous counterpart of the deterministic model (2.2) already reveals the effect of environmental fluctuation on an allelopathic inhibiting system.

References

[1] Chattopadhyay, J., Effect of toxic substances on a two-species competitive system, Ecological Modelling, **84**: 287–289, 1996

[2] Coffey, W. T., Kalmykov, Yu. P. and Waldron, J. T., The Langevin Equation with Applications in Physics, Chemistry and Electrical Engineering. World Scientific, 1997

[3] Gradshteyn, I. S. and Ryzhik, I. M., Table of Integrals, Series and Products. Academic Press, New York, 1980

[4] Hellebust, J. A., Extracellular products in Algal Physiology and Biochemistry, W. D. P. Stewart, Ed., Univ. of California Press, Berkeley, 1974, pp. 838–863

[5] Maynard-Smith, J., Models in Ecology, Cambridge University Press, 1974, p. 146

[6] Mukhopadhyay, A., Chattopadhyay, J. and Tapaswi P. K., A delay differential equations model of plankton allelopathy. Mathematical Biosciences, **149**, 167–189, Elsevier

[7] Nisbet, R. M. and Gurney, W. S. C., Modelling Fluctuating Population. Wiley Interscience, New York, 1982

[8] Renshaw, E., Modelling Biological Population in Space and Time. Cambridge University Press, 1995

[9] Rice, E. L., Allelopathy (second edition). Academic Press, INC., 1984