Influences of noise correlation and time delay on stochastic resonance induced by multiplicative signal in a cancer growth system*

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This paper investigates the stochastic resonance (SR) phenomenon induced by the multiplicative periodic signal in a cancer growth system with the cross-correlated noises and time delay. To describe the periodic change of the birth rate due to the periodic treatment, a multiplicative periodic signal is added to the system. Under the condition of small delay time, the analytical expression of the signal-to-noise ratio $R_{\rm SNR}$ is derived in the adiabatic limit. By numerical calculation, the effects of the cross-correlation strength λ and the delay time τ on $R_{\rm SNR}$ are respectively discussed. The existence of a peak in the curves of $R_{\rm SNR}$ as a function of the noise intensities indicates the occurrence of the SR phenomenon. It is found that λ and τ play opposite role on the SR phenomenon, i.e., the SR is suppressed by increasing λ whereas it is enhanced with the increase of τ, which is different from the case where the periodic signal is additive.

Keywords: time delay, cross-correlated noise, stochastic resonance, cancer growth system

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1. Introduction

In recent years, much attention has been directed towards nonlinear physics and its application to uncover biological complexities. Studies have confirmed the important role of noise in the nonlinear stochastic systems.[1] In particular, many studies have focused on the growth law of tumor cells via dynamics, especially stochastic dynamics.[2−8] Many noise-induced effects, such as noise-induced transition,[2,3] stochastic resonance,[4−6] noise-enhanced stability and resonant activation,[7] have been observed and studied in the tumor cell growth system. These studies further deepen the understanding of the growth law of tumor cells. The cancer research is now an interdisciplinary field, and so far the investigation of the growth law of tumor cells is still a challenging subject.

Since the stochastic resonance (SR) phenomenon was first found in the study of the periodic changes of the ancient climate,[9] it has been extensively investigated both theoretically and experimentally in many scientific fields including biophysics due to its potential applications.[1] For example, the SR phenomenon has been investigated in the bistable system,[10] the bias linear system,[11] the single mode laser system,[12] the biological system,[4−6,13] and so on. The SR is a typical example for the construction role of noise. The adiabatic elimination theory,[14] linear response theory[15] and perturbation theory[16] were employed to characterize the SR phenomenon. The typical signature of the SR is the existence of a maximum of the output signal and of the signal-to-noise ratio (SNR) as a function of noise intensity. The residence-time distribution offers another possibility to characterize the SR.[17] It is noteworthy that many previous studies on the SR are just for the case of additive signal. However, in some situations, the signal should be multiplicative in many physical systems, such as in a membrane-protein system.[18] Hence, the study of the SR should be extended to the case of the multiplicative signal. Collins *et al.* introduced a multiplicative aperiodic signal into a symmetric bistable potential with the presence of additive noise, and found aperiodic SR.[19] Recently, C. Nicolis and G. Nicolis studied the SR in the presence of slowly varying control parameters and a multiplicative periodic signal in a bistable system.[20] Their results may give rise to a new method for the control of the transition rates.[21] On the other hand, due to the finite transmission times of the signals, or other key quantities (such as information, matter, energy, etc.), the time delay

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is ubiquitous in nature.[22] Accordingly, the inclusion of the time delay is more natural in many physical systems. Many studies have been devoted to the effects of noise on the time-delayed systems, and have shown that the combination of noise and time delay fundamentally changes the statistical properties of the systems.[23,24] Particularly, the effect of time delay on the SR has been investigated in different time-delayed stochastic systems, which revealed that the time delay has important influence on the SR.[25,26] The analytical approximate method for time-delayed stochastic systems has been developed for the case of small delay time.[22] When the delay time is large, the numerical simulation method can be used.[24] More recently, Du and Mei[27] studied the effects of noise correlation and time delay on the SR induced by the multiplicative signal in a bistable system driven by cross-correlated noises. They revealed that both noise correlation and delay time can induce the critical behaviour on the SR phenomenon, which is distinctly different from the case of the additive signal. Thus, it is of interest to study the effect of time delay on the SR induced by multiplicative signal in various systems.

There are many examples of processes in the biological world which involve significant delays.[28] The most obvious ones include the time between initiation of cellular division and effective division in the case of mitosis, and the time required for digestion in the case of consumption of nutrient and its conversion to viable biomass. Accordingly, the inclusion of the time delay is more natural in the cancer growth dynamics. Moreover, when we take simultaneously the effects of the periodic treatment and the environmental fluctuations on the cancer growth dynamics into account, it is physically reasonable to introduce the multiplicative signal and the cross-correlated noises into the system. However, so far, how these factors influence the dynamics of the cancer growth has not been fully understood yet. Thus, the SR phenomenon induced by the multiplicative signal, in the cancer growth system with the cross-correlated noises and time delay, needs to be investigated. The effects of the noise correlation and delay time on the SR phenomenon deserve further discussion in the cancer growth dynamics.

In this paper, based on the stochastic logistic model proposed by Ai *et al.*[2] a time-delayed stochastic cancer growth model is put forward with consideration of the time delay and periodic treatment. To describe the effect of the periodic treatment on the tumor cell growth, a multiplicative periodic signal is introduced into the system. The effects of the noise correlation and delay time on the SR induced by the multiplicative signal are studied in terms of the SNR technique. This study aims to contribute to a better understanding of the behaviour of the tumor cells under the influences of the delay time, cross-correlated noises and periodic treatment. In the next section, the approximate analytical expression of the SNR of the system is derived, and the effects of the noise correlation and time delay on the SNR of the system are analysed. A brief conclusion and some discussions are given in the final section.

2. The effects of the noise correlation and time delay on the SNR of the system

The logistic growth model is[29]

$$\dot{x} = rx\left(1 - \frac{x}{K}\right), \tag{1}$$

where r is the linear per capita birth rate, K is the carrying capacity of the environment and x is the population. This model can be used as a basic model of the tumor cell growth. However, it is an ideal equation without any fluctuation and time delay. In fact, the tumor cells are always influenced by some external factors, such as temperature, drugs, radiotherapy, and so on. Taking the stochastic properties of these factors into consideration, physically we are reasonable to introduce a multiplicative noise and an additive noise into Eq. (1) to describe the effects of these external factors on the tumor cell growth. The fluctuation of some external factors affects the linear per capita birth rate r, and generates a multiplicative noise $\xi(t)$. Some factors such as drugs, radiotherapy, and emigration or immigration of tumors change the number of tumor cells in a local area, and give rise to a negative additive noise $-\eta(t)$. Thus, the Langevin equation of the tumor cell growth system is[2,3]

$$\dot{x} = rx\left(1 - \frac{x}{K}\right) + x\xi(t) - \eta(t), \tag{2}$$

where $\xi(t)$ and $\eta(t)$ are the Gaussian white noises with zero mean, and

$$\langle \xi(t)\xi(t') \rangle = 2\alpha\delta(t - t'), \tag{3}$$

$$\langle \eta(t)\eta(t') \rangle = 2D\delta(t - t'), \tag{4}$$

$$\langle \xi(t)\eta(t') \rangle = 2\lambda\sqrt{\alpha D}\delta(t - t'), \tag{5}$$

where α and D are the intensities of the noises, and λ ($0 \leq \lambda \leq 1$) denotes the strength of the cross-correlation between $\xi(t)$ and $\eta(t)$. The deterministic potential corresponding to Eq. (2) is

$$V(x) = -\frac{r}{2}x^2 + \frac{r}{3K}x^3, \tag{6}$$

which has two stable states $x_1 = K$, $x_2 = 0$.

On the other hand, when the tumor cells are under a periodic chemotherapeutic treatment, the linear per capita birth rate should have a periodic form, for instance, a cosinoidal form.[4] Moreover, the time delay is also an important factor in the tumor cell growth system. If we further take these two factors into account, based on Eq. (2), the stochastic delay differential equation for the cancer growth process can be obtained as

$$\dot{x}(t) = rx(t)\left[1 - \frac{x(t-\tau)}{K}\right] + x(t)\xi(t) - \eta(t) + x(t)\left[1 - \frac{x(t)}{K}\right]A\cos(\Omega t), \tag{7}$$

where τ is the delay time, and A is the amplitude of the input periodic signal and Ω is its frequency.

The delay Fokker–Planck equation corresponding to Eq. (7) is given by[2,22]

$$\frac{\partial P(x,t)}{\partial t} = -\frac{\partial}{\partial x}\int\left[rx\left(1 - \frac{x_\tau}{K}\right) + \alpha x - \lambda\sqrt{\alpha D} + x\left(1 - \frac{x}{K}\right)A\cos(\Omega t)\right]P(x,t;x_\tau,t-\tau)\mathrm{d}x_\tau + \int\frac{\partial^2}{\partial x^2}[\alpha x^2 - 2\lambda\sqrt{\alpha D}x + D]P(x,t;x_\tau,t-\tau)\mathrm{d}x_\tau, \tag{8}$$

where $x_\tau = x(t-\tau)$, and $P(x,t;x_\tau,t-\tau)$ is the joint probability distribution. Since the frequency Ω is very small, there is enough time for the system to reach local equilibrium during the period of $1/\Omega$. Thus, in the condition of the small delay time and the first order approximation,[22] the quasi-steady-state distribution function can be derived from Eq. (8) in the adiabatic limit[14]

$$P_{\mathrm{qst}}(x,t) = \frac{N}{\alpha x^2 - 2\lambda\sqrt{\alpha D}x + D}\exp\left[\frac{-\Phi(x,t)}{\alpha}\right], \tag{9}$$

where N is the normalization constant, and the generalized potential $\Phi(x,t)$ reads

$$\Phi(x,t) = -\frac{1}{2}\beta_3 x^2 - \left(2\lambda\sqrt{\frac{D}{\alpha}}\beta_3 + \beta_2\right)x - \left[\lambda\beta_2\sqrt{\frac{D}{\alpha}} + \frac{1}{2}\beta_1 + \left(2\lambda^2 - \frac{1}{2}\right)\beta_3\frac{D}{\alpha}\right] \times \ln(|\alpha x^2 - 2\lambda\sqrt{\alpha D}x + D|) + \frac{1}{\sqrt{(1-\lambda^2)D\alpha}}\left[(1-2\lambda^2)D\beta_2 - \lambda\sqrt{D\alpha}(\beta_1 - \alpha) + (4\lambda^3 + 3\lambda)D\beta_3\sqrt{\frac{D}{\alpha}}\right]\arctan\left(\frac{\alpha x - \lambda\sqrt{D\alpha}}{\sqrt{(1-\lambda^2)D\alpha}}\right), \tag{10}$$

in which

$$\beta_1 = r\left(1 + \frac{\lambda\tau\sqrt{\alpha D}}{K}\right) + A\cos(\Omega t) + \alpha, \tag{11}$$

$$\beta_2 = -\left[\frac{r\tau(1+r+\alpha)}{K} + \frac{1+r\tau}{K}A\cos(\Omega t)\right], \tag{12}$$

and

$$\beta_3 = \frac{r\tau[r + A\cos(\Omega t)]}{K^2}. \tag{13}$$

Firstly, we consider the mean first-passage time (MFPT) of the system from the initial state $x_1 = K$ to the final state $x_2 = 0$. For the case of small D and α, by using the steepest-descent approximation,[30] we can obtain the explicit expression of the modified MFPT is

$$T(x_1 \to x_2) = \frac{2\pi}{\sqrt{|V''(x_1)V''(x_2)|}}\exp\left[\frac{\Phi(x_2) - \Phi(x_1)}{\alpha}\right]. \tag{14}$$

Here, the prime of $V(x)$ denotes the differentiation with respect to the variable x. Then the state transition rate is obtained as[14,31]

$$W = \frac{1}{T(x_1 \to x_2)} = \frac{r}{2\pi} \exp\left[\frac{\Phi(x_1) - \Phi(x_2)}{\alpha}\right]. \tag{15}$$

By virtue of the theory of SR proposed by McNamara and Wiesenfeld,[14] we can obtain the expression of the SNR of the system in adiabatic limit,

$$R_{\rm SNR} = \frac{\pi W_1^2 A^2}{4W_0}\left[1 - \frac{W_1^2 A^2}{2(W_0^2 + \Omega^2)}\right]^{-1}, \tag{16}$$

where

$$\begin{aligned} W_0 = 2W|_{A\cos(\Omega t)=0} &= \frac{r}{\pi}\exp\left\{\alpha^{-1}\left[-\frac{1}{2}\tau r^2 + 2\lambda\sqrt{\frac{D}{\alpha}}\frac{\tau r^2}{K} + r\tau(1+\alpha+r)\right]\right. \\ 4 \quad &+ \alpha^{-1}\left[\lambda\sqrt{\frac{D}{\alpha}}\frac{r\tau(1+r+\alpha)}{K} - \left(2\lambda^2 - \frac{1}{2}\right)\frac{D\tau r^2}{\alpha K^2} - \frac{1}{2}\alpha - \frac{1}{2}r\left(1 + \frac{\lambda\tau}{K}\sqrt{D\alpha}\right)\right] \\ &\times \ln\left|\frac{\alpha}{D}K^2 - 2\lambda\sqrt{\frac{\alpha}{D}}K + 1\right| + \frac{1}{\alpha\sqrt{(1-\lambda^2)D\alpha}}\left[\frac{(2\lambda^2-1)Dr\tau}{K}(1+r+\alpha) - \lambda r\sqrt{D\alpha} - \frac{\lambda^2 r\tau D\alpha}{K}\right) \\ 4 \quad &\left. + \frac{(4\lambda^3+3\lambda)D\tau r^2}{K^2}\sqrt{\frac{D}{\alpha}}\right]\left[\arctan\left(\frac{\alpha K - \lambda\sqrt{D\alpha}}{\sqrt{(1-\lambda^2)D\alpha}}\right) + \arctan\left(\frac{\lambda}{\sqrt{1-\lambda^2}}\right)\right]\right\}, \end{aligned} \tag{17}$$

and

$$\begin{aligned} W_1 &= 2\frac{{\rm d}W}{{\rm d}[A\cos(\Omega t)]}\bigg|_{A\cos(\Omega t)=0} \\ &= \frac{W_0 r}{\pi\alpha}\left\{-\frac{r\tau}{2} - \frac{2\lambda r\tau}{K}\sqrt{\frac{D}{\alpha}} + r\tau + 1 + \left[\frac{(1-4\lambda^2)Dr\tau}{2\alpha K^2} - \lambda\sqrt{\frac{D}{\alpha}}\frac{r\tau+1}{K} - \frac{1}{2}\right]\right. \\ &\times \ln\left|\frac{\alpha}{D}K^2 - 2\lambda\sqrt{\frac{\alpha}{D}}K + 1\right| + \frac{1}{\sqrt{(1-\lambda^2)D\alpha}}\left[(2\lambda^2-1)\frac{D(r\tau+1)}{K}\right. \\ &\left.\left. - \lambda\sqrt{D\alpha} + \frac{Dr\tau(4\lambda^3+3\lambda)}{K^2}\sqrt{\frac{D}{\alpha}}\right]\left[\arctan\left(\frac{\alpha K - \lambda\sqrt{D\alpha}}{\sqrt{(1-\lambda^2)D\alpha}}\right) + \arctan\left(\frac{\lambda}{\sqrt{1-\lambda^2}}\right)\right]\right\}. \end{aligned} \tag{18}$$

By virtue of the expression (16) for the SNR, we can investigate the effects of correlated intensity λ and delay time τ on the SNR $R_{\rm SNR}$ as a function of the multiplicative noise intensity α and additive noise intensity D respectively by numerical calculation. The results of the numerical calculations are plotted in the following figures.

In Figs. 1 and 2, the SNR $R_{\rm SNR}$ as a function of the multiplicative noise intensity α is presented for different values of the cross-correlated strength λ and the delay time τ, respectively. From Figs. 1 and 2, one can see that there exhibits a maximum of the SNR as a function of α, which is the identifying characteristic of the SR phenomenon. It is shown that when the delay time τ and the additive noise intensity D are fixed, with the increasing cross-correlation strength λ, the maximum of $R_{\rm SNR}$ decreases and the position of the maximum of $R_{\rm SNR}$ shifts towards larger value of α. As λ further increases, the maximum of $R_{\rm SNR}$ will eventually disappear. That is to say that in the presence of the multiplicative signal, the SR phenomenon induced by the multiplicative noise can be restrained in this cancer growth system by increasing λ (see Fig. 1). However, when λ and D are fixed, the maximum of $R_{\rm SNR}$ increases as τ increases, which implies that the SR phenomenon induced by the multiplicative noise can be enhanced by increasing τ for the case of the multiplicative signal (see Fig. 2). The position of the maximum of $R_{\rm SNR}$ keeps unchanged with increasing τ. One should note that the effect of the delay time on the SR is different from the case of the additive signal, in which the SR is weakened by increasing the delay time.[25]

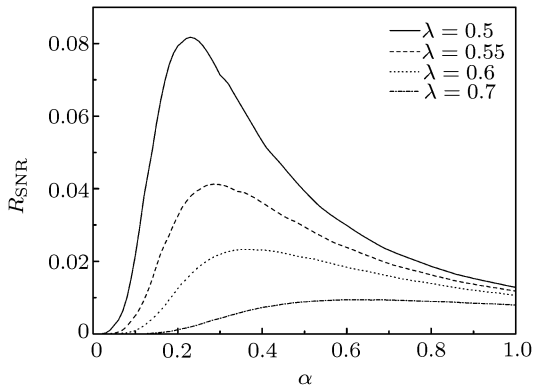


Fig. 1. Plot of the SNR ($R_{\rm SNR}$) as a function of the multiplicative noise intensity (α) when $r = 1.0$, $K = 2.0$, $A = 0.05$, $\Omega = 0.001$, $D = 0.01$, and $\tau = 0.3$ are fixed. The cross-correlation strength λ takes various values.

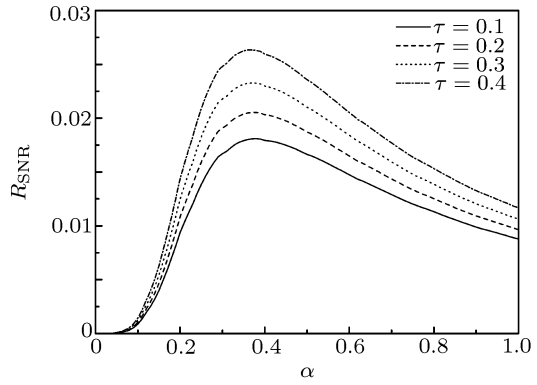


Fig. 2. Plot of the SNR ($R_{\rm SNR}$) as a function of the multiplicative noise intensity (α) when $r = 1.0$, $K = 2.0$, $A = 0.05$, $\Omega = 0.001$, $D = 0.01$, and $\lambda = 0.6$ are fixed. The delay time τ takes various values.

The SNR $R_{\rm SNR}$ as a function of the additive noise intensity D is respectively plotted in Figs. 3 and 4, for different values of the cross-correlation strength λ and the delay time τ. From Figs. 3 and 4, for optimally selected values of the additive noise intensity D, $R_{\rm SNR}$ as a function of D also shows a maximum, which indicates that the SR phenomenon induced by the additive noise occurs. Figure 3 shows that the SR phenomenon induced by the additive noise is sensitively influenced by increasing λ. With increasing λ, the maximum of $R_{\rm SNR}$ decreases expeditiously and the position of the maximum shifts towards smaller value of D, which indicates that the SR phenomenon induced by the additive noise can be weakened by increasing λ. However, as τ increases, the maximum of $R_{\rm SNR}$ increases and the position of the maximum shifts towards larger value of D (see Fig. 4). That is to say that the SR induced by the additive noise can be enhanced by increasing τ in the presence of the multiplicative signal.

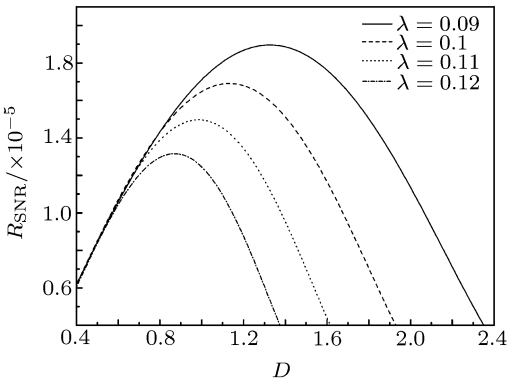


Fig. 3. Plot of the SNR ($R_{\rm SNR}$) as a function of the additive noise intensity (D) when $r = 1.0$, $K = 2.0$, $A = 0.05$, $\Omega = 0.001$, $\alpha = 0.02$, and $\tau = 0.2$ are fixed. The cross-correlation strength λ takes various values.

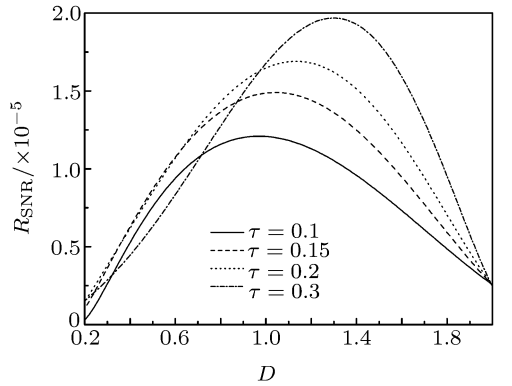


Fig. 4. Plot of the SNR ($R_{\rm SNR}$) as a function of the additive noise intensity (D) when $r = 1.0$, $K = 2.0$, $A = 0.05$, $\Omega = 0.001$, $\alpha = 0.02$, and $\lambda = 0.1$ are fixed. The delay time τ takes various values.

3. Discussion and conclusion

In summary, we have investigated the SR phenomenon induced by a multiplicative periodic signal in the cancer growth system with the cross-correlated noises and time delay by using the theory of SNR proposed by McNamara and Wiesenfeld[14] and the small delay approximation.[22] The approximate expression for SNR is obtained. By virtue of the expression for SNR and through numerical computation, we investigated the effects of the noise correlation and delay time of the SR phenomenon. The results indicate that the existence of a maximum in the SNR is the identifying characteristic of the SR phenomenon. The SR phenomenon induced by both multiplicative noise and additive noise can be suppressed by increasing the cross-correlation strength whereas it is enhanced by increasing the delay time, which is different from the case of the additive signal studied by Cai and Mei.[25] As the

cross-correlation strength increases, the position of the maximum of the SNR shifts towards a larger value of multiplicative noise intensity and towards a smaller value of additive noise intensity. However, with the increasing delay time, the position of the maximum SNR remains the same in the $R_{\rm SNR}$–α plot and shifts towards a larger value of additive noise intensity in the $R_{\rm SNR}$–D plot. The present study reveals that the introduction of a multiplicative periodic signal can provide new features of SR in a cancer growth system. It will be helpful in deepening the understanding of tumor cell growth behaviour under the influences of periodic treatment, time delay and cross-correlated noises.

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