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Stationary Distribution and Extinction of Stochastic HTLV-I Infection Model with CTL Immune Response under Regime Switching*

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Abstract In this paper, the stochastic HTLV-I infection model with CTL immune response is investigated. Firstly, we show that the stochastic system exists unique positive global solution originating from the positive initial value. Secondly, we obtain that the existence of ergodic stationary distribution of the model by stochastic *Lyapunov* functions. Thirdly, we establish sufficient conditions for extinction of the infected cells. Finally, numerical simulations are carried out to illustrate the theoretical results.

Keywords Stochastic HTLV-I infection model, Ergodic stationary distribution, Extinction, Markov switching.

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1. Introduction

Human T-cell leukemia virus includes type-I (HTLV-I) and type-II (HTLV-II), it is pathogen that causes T-cell leukemia and lymphoma in adults. HTLV-I can be transmitted by blood transfusions, injection or sexual contact, or by placenta, birth canal as well as breast-feeding which harms public health, human society and world economy seriously [1–3]. To fight against HTLV-I, which is a kind of infectious disease, we need to pay enough attention to inventing effective drugs and updating treatment methods. What's more, it is known that the dynamic nature of virus spread also has practical significance for disease prevention and control [4, 5].

The Cytotoxic T lymphocyte (CTL) play an important role in antiviral mechanism and they are the main immune factor inhibiting cell replication [6]. In certain infectious diseases, specific CTL kills infected cells, not viruses, such as hepatitis B. Therefore, the dynamics of virus infection model with CTL response has attracted lots of researchers attention [7, 8], which is essential for identifying risk factors of the HAM/TSP development and taking therapeutic measures [9].

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The CTL immune response to a single pool of infected cells has been considered in the HTLV-I infection. This interaction can be described by the following system [10].

$$\begin{cases} \dot{x}(t) = \lambda - \beta x(t)y(t) - d_1 x(t), \\ \dot{y}(t) = \beta x(t)y(t) - ay(t)z(t) - d_2 y(t), \\ \dot{z}(t) = py(t) - d_3 z(t), \end{cases} \tag{1.1}$$

where $x(t), y(t)$ and $z(t)$ are numbers of uninfected of cells, infected cells, and CTL immune cells, respectively. Wang etc obtained the global dynamics of differential system (1.1) which is determined by one important threshold parameter $R_0^* = \lambda\beta/(d_1 d_2)$. If $R_0^* > 1$, then the system (1.1) has two steady states, the infection free steady state and the endemic steady state. It is well-known that if a basic reproductive number $R_0^* < 1$, the infection free steady state is locally asymptotically stable and the endemic steady state does not exist [10].

The meaning of the parameters in the model (1.1) is given in the following list:

λ: the production of healthy $CD4^+T$ cells rate;

β: a constant means the infection rate;

a: the rate of CTL elimination;

$py(t)$: the proliferation rate of CTL cells by contacting the infected cells;

d_1: the natural mortality rate of $x(t)$;

d_2: the mortality rate of infected cells caused by virus;

d_3: the natural mortality rate of $z(t)$;

Those magnificent works provide a great perspective of the epidemic model. But in the real world, the virus dynamics model will inevitably be affected by random fluctuations. Aimed to make the virus dynamic model (1.1) better reflect the actual situation, it is essential to take into account the real random interference in the disease dynamics model.

The pathogenesis of different stages of HTLV-I infection is different, chemotherapy treatment, drug, and cell transplantation have the diverse effects. The external environment will influence people's body and mind. As a result, the system possibly changed from one environmental regime to another.

Note that the epidemic models may be perturbed by telegraph noise which can cause the system to switch from one environmental regime to another [11]. Almost the switching between environmental regimes is usually memoryless and the waiting time for the next switching follows the exponential distribution [12]. Therefore the regime switching can be described by a continuous time Markov chain $r(t)_{t>0}$ with values in a finite state space.

In [13], Jiang and Qi thought that the deterministic model (1.1) disturbed by the telegraph noises and white noises. Furthermore, they consider the standard incidence $\beta xy/(x+y)$ instead of the bilinear incidence βxy. Then model (1.1) under regime switching reduces to

$$\begin{cases} dx = [\lambda(r(t)) - \frac{\beta(r(t))x(t)y(t)}{x(t)+y(t)} - d_1(r(t))x(t)]dt + \sigma_1(r(t))x(t)dB_1, \\ dy = [\frac{\beta(r(t))x(t)y(t)}{x(t)+y(t)} - a(r(t))y(t)z(t) - d_2(r(t))y(t)]dt + \sigma_2(r(t))y(t)dB_2, \\ dz = [p(r(t))y(t) - d_3(r(t))z(t)]dt + \sigma_3(r(t))z(t)dB_3, \end{cases} \tag{1.2}$$

where $B_i (i = 1, 2, 3)$ is independent standard Brownian motions and $\sigma_i(r(t))(i = 1, 2, 3)$ is the intensity of $B_i (i = 1, 2, 3)$.