HIV/AIDS Model Based on Local Activity of CNN

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Abstract

A Cellular Neural Network (CNN) model of two strain (antiretroviral sensitive and resistant) Human Immunodeficiency Virus (HIV) is established, and then analyzed and simulated based on the local activity of CNN with five local state variables and one port. Numerical simulations exhibit that this CNN model may explain some complex phenomena during antiretroviral therapy which make it possible to judge the curative effect of long-time antiretroviral therapy and social influence. All these imply that the local activity of CNN provides a practical tool for the study of the complex dynamics of some coupled nonlinear systems, especially life systems.

Keywords: HIV/AIDS model, Cellular Neural Network (CNN), Local activity, Numerical simulation, Antiretroviral

1. Introduction

Coupled nonlinear dynamical systems have been widely studied in recent years. However the dynamical properties–the determination, prediction, and control of these systems are difficult to deal with. Nature abounds with complex patterns and structures emerging from homogeneous media, the local activity is the origin of these complexity (Chua, 1997; Chua 2005). The Cellular Neural Network (CNN), firstly introduced by Chua (Chua, 1988), has been widely studied for image processing, robotic, biological versions, higher brain functions and so on (Chua, 1988). It can model and study many coupled nonlinear systems (Chua, 1999). The local activity of CNN proposed by Chua (Chua, 1997) asserts that a wide spectrum of complex behaviors may exist if the cell parameters of the corresponding CNN are chosen in or nearby the edge of chaos (Chua, 1999; Chua, 2005). This theory has been successfully applied to the research on many complex systems with physical, biological, and chemical backgrounds.

Life systems consist of locally coupled homogeneous media. Virtually, dynamics of life systems are suitable to be described via locally connected CNNs. It may be expected that CNN will become a promising candidate for modeling life phenomena. This theory has been successfully applied to life systems, such as the biochemical model (Min, 2000), coupled excitable cell model (Min, 2002), tumor growth and immune model (Min, 2003), HBV infection model (Min, 2006), T cells (Ji, 2008), and so on.

HIV, the Human Immunodeficiency Virus, is the etiological agent of Acquired Immune Deficiency Syndrome (AIDS). Deaths due to AIDS are more than two million people per year recently, making it one of the destructive epidemics in history and the leading cause of death in the world (WHO, 2009). A number of theoretical studies have focused on the mathematical modeling of HIV/AIDS (Mukandavire, 2006; Martcheva, 2007; Bhunu, 2009). But these models were analyzed simply because of the complexity usually. In (Bhunu, 2009), a two strain (antiretroviral sensitive and resistant) HIV/AIDS model with treatment which allows AIDS patients with sensitive HIV-strain to undergo amelioration is presented as a system of nonlinear differential equations with five variables.

In this paper, the model in (Bhunu, 2009) is mapped into a CNN form and the equilibrium points are calculated and analyzed based on the local activity of CNN with five local state variables and one port (Dong, 2009). The bifurcation of this CNN has been calculated and analyzed. Numerical simulations show that this CNN model may explain some complex phenomena during HIV/AIDS treatments. The quantitative understanding of HIV/AIDS dynamics will make it possible to judge the curative effect of long-time antiretroviral therapy and social influence.

2. Analysis and Simulations Reaction-Diffusion CNN of HIV/AIDS Model

2.1 The HIV/AIDS Model and its CNN model

In (Bhunu, 2009), a two strain (antiretroviral sensitive and resistant) HIV/AIDS model with treatment which allows AIDS patients with sensitive HIV-strain to undergo amelioration is presented as a system of non-linear differential equations, which classifies people into five classes: susceptible (S), antiretroviral sensitive HIV infected (I_1), AIDS individuals with antiretroviral sensitive HIV (A_1), antiretroviral resistant HIV infected (I_2) and AIDS individuals with antiretroviral

resistant HIV (A_2). The formulation of HIV/AIDS model is

$$\begin{cases} \frac{dS}{dt} = a - \left(\frac{b_1(I_1+nA_1)+b_2(I_2+nA_2)}{S+I_1+A_1+I_2+A_2} + u\right)S \\ \frac{dI_1}{dt} = \frac{b_1(I_1+nA_1)S}{S+I_1+A_1+I_2+A_2} - (q_1+u)I_1 + yeA_1 \\ \frac{dA_1}{dt} = q_1 I_1 - (u+d_1+e)A_1 \\ \frac{dI_2}{dt} = \frac{b_2(I_2+nA_2)S}{S+I_1+A_1+I_2+A_2} - (q_2+u)I_2 \\ \frac{dA_2}{dt} = q_2 I_2 - (u+d_2)A_2 + (1-y)eA_1 \end{cases} \tag{1}$$

where, individuals are reproduced into the susceptible class at constant rate a, and u is a natural death rate in each human subgroup. The antiretroviral sensitive HIV infected becomes AIDS individuals with antiretroviral sensitive HIV ($I_1 \to A_1$) at rate q_1. Individuals in A_1 have an additional AIDS-induced death rate d_1, and are given antiretroviral therapy at rate e. A proportion y of individuals in A_1 given antiretroviral therapy respond well and move into I_1, and the complimentary proportion $1 - y$ develops resistance and enters A_2. The antiretroviral resistant HIV infected becomes AIDS individuals with antiretroviral resistant HIV ($I_2 \to A_2$) at rate q_2. Individuals in A_2 have an additional AIDS-induced death rate d_2. The force of infection y_i is given as

$$y_i = \frac{b_i(I_i + nA_i)S}{S + I_1 + A_1 + I_2 + A_2}, i = 1, 2$$

b_1, b_2 is the product of the effective contact rate and the transmission probability of antiretroviral sensitive and resistant HIV infection per contact, respectively. $n > 1$ is the modification parameter which models the fact that individuals in the AIDS stage are more infectious than the corresponding HIV infected not yet in the AIDS stage.

The HIV/AIDS CNN model has the form:

$$\begin{cases} \frac{dS_{ij}}{dt} = a - \left(\frac{b_1(I_{1ij}+nA_{1ij})+b_2(I_{2ij}+nA_{2ij})}{S_{ij}+I_{1ij}+A_{1ij}+I_{2ij}+A_{2ij}} + u\right)S_{ij} + D_1\nabla^2 S_{ij} \\ \frac{dI_{1ij}}{dt} = \frac{b_1(I_{1ij}+nA_{1ij})S_{ij}}{S_{ij}+I_{1ij}+A_{1ij}+I_{2ij}+A_{2ij}} - (q_1+u)I_{1ij} + yeA_{1ij} \\ \frac{dA_{1ij}}{dt} = q_1 I_{1ij} - (u+d_1+e)A_{1ij} \\ \frac{dI_{2ij}}{dt} = \frac{b_2(I_{2ij}+nA_{2ij})S_{ij}}{S_{ij}+I_{1ij}+A_{1ij}+I_{2ij}+A_{2ij}} - (q_2+u)I_{2ij} \\ \frac{dA_{2ij}}{dt} = q_2 I_{2ij} - (u+d_2)A_{2ij} + (1-y)eA_{1ij} \end{cases} \tag{2}$$

where

$$\nabla^2 S_{ij} = S_{i+1j} + S_{i-1j} + S_{ij+1} + S_{ij-1} - 4S_{ij}.$$

2.2 Analysis of Equilibrium Points

Let Eq.(2) be zero (where $D_1 = 0$) and solve it, we can get the three equilibrium points:

$$\begin{aligned} Q_1 &= \left(\frac{a}{u}, 0, 0, 0, 0\right), \\ Q_2 &= \left(\frac{a(d_2+q_2+u)}{A}, 0, 0, \frac{aB}{(q_2+u)A}, \frac{aq_2B}{(d_2+u)(q_2+u)A}\right), \\ Q_3 &= \left(\frac{Q_S}{Q}, \frac{Q_{I_1}}{Q}, \frac{Q_{A_1}}{Q}, \frac{Q_{I_2}}{Q}, \frac{Q_{A_2}}{Q}\right). \end{aligned}$$

where

$$A = b_2(d_2 + u + q_2n) - d_2q_2.$$
$$B = b_2(d_2 + u + q_2n) - (d_2 + u)(q_2 + u).$$
$$X = (d_1 + e + u)(q_1 + u) - eq_1y.$$
$$Y = b_1(d_1 + e + u + q_1n) - X.$$
$$Z = (b_1 - b_2)u^3 + ((b_1 - b_2)(d_1 + +d_2 + e) + n(b_1q_1 - b_2q_2) + b_1q_2 - b_2q_1)u^2 + ((d_1 + d_2 + e)(b_1q_2 - b_2q_1)$$
$$+(b_1 - b_2)(d_1d_2 + d_2e + q_1q_2n) + b_2e(q_1y - q_2n) + n(b_1d_2q_1 - b_2d_1q_2))u + q_1q_2n(b_1d_2 - b_2d_1)$$
$$+d_2(d_1 - e)(b_1q_2 - b_2q_1) + b_2q_1(d_2ey + eq_2ny - eq_2)y.$$
$$Q_{I_1} = a(d_1 + e + u)YZ/X.$$
$$Q_{A_1} = aq_1YZ/X.$$
$$Q_{I_2} = aq_1b_2en(1 - y)Y.$$
$$Q_{A_2} = aeq_1(1 - y)((d_1 + e + u)(b_1u + b_1q_2 - b_2u - b_2q_1) + q_1(b_2ey + b_1nu + b_1nq_2))Y/X.$$
$$Q_S = \frac{(q_2 + u)(Q_{I_1} + Q_{I_2} + Q_{A_1} + Q_{A_2})}{(b_2 - q_2 - u)Q_{I_2} + b_2nQ_{A_2}}.$$
$$Q = (b_1^2 - b_1b_2)u^4 + ((b_1 - b_2)(b_1(2d_1 + d_2 + 2e + q_1n) - d_1q_1) + b_1(b_1(q_1n + q_2) - b_2(q_1 + q_2n)))u^3$$
$$+(eq_1(b_1b_2(1 - n) + b_1d_2 - b_2d_2)y + b_1^2(q_1^2n^2 + d_1^2 + e^2 + 2(d_2 + q_2 + nq_1)(d_1 + e) + 2q_1n(d_2 + q_2)$$
$$-2d_1e) + q_1(b_2 - b_1)(e(d_1 + d_2) - d_1q_2) + d_1q_1q_2(b_2d_1n - b_1d_1) - b_1b_2(e^2 + q_1q_2n^2 - 2d_1e + q_1n(d_1$$
$$+q_2) - 2(d_1 + e)(q_2n + d_2q_1) - d_2q_1(1 + n)) + d_1^2(b - 2d_1e) + d_1^2(b_2q_1 - b_1b_2 - b_1q_1))u^2 + ((b_1b_2eq_1(1$$
$$-n)(e + q_1n) + d_2eq_1(b_1 - b_2)(d_1 + e) + b_2eq_1(d_1 + d_2)(b_1 - q_1) + b_1eq_1(d_2q_2 + d_1q_1n - b_2d_1n))y$$
$$+b_1^2((d_2 + q_2)((d_1 + nq_1)^2 + e(e + 2d_1 + 2q_1n)) + d_1^2(b_2(q_1 - b_1)(d_2 + q_1 + q_2n) + b_1q_1(d_2 + q_2))$$
$$-b_1b_2q_1q_2n^2(d_1 + e + q_1) + e^2(b_1b_2n(q_1 - q_2) - b_1b_2(q_1 + d_2) + d_2q_1(b_2 - b_1)) + q_1^2(b_2(d_1d_2 - d_1e$$
$$+d_2e - b_1n(d_2 - d_1 + e - en)) - b_1d_1n(d_2 + q_2) + n(b_2d - 1q_1 - b_1d_2e))) + b_1eq_2(b_1 + d_2)(b_2 + q_2)$$
$$-b_1(q_1 + e)(d_2 + nq_2) + 2b_1d - 2eq_1(b_2 - b_1) - q_1q_2d_1e(d_1 + d_2) + b_1b_2d_1q_1n(e - d_2) - b_1b_2enq_1(d_2$$
$$+q_1))u - b_1d_1^2d_2q_1q_2 + (d_2eq_1(d_1 + e)(b_1b_2 + b_1q_2 - 2b_2q_1) + q_1^2en(b_1d_2q_1 - b_2d_1q_2 + b_1d_2q_2))y + q_1(d_1$$
$$+e)(b_1d_2n(2b_1q_2 - d_1q_1 - eq_1) + b_2d_1q_1(q_2n + d_2)) - b_1d_2q_1(b_2 + q_2)(2d_1e + d_1^2 + e^2) + q_1^2e^2b_2d_2(1 + y^2)$$
$$+q_1^2n^2b_1q_2(b_1d_2 - b_2d_1) + b_1^2d_2eq_2(2d_1 + e) + b_2d_1eq_1(d_2q_1 - b_1q_2n).$$

The equilibrium Q_1, Q_2, Q_3 stand for the disease-free equilibrium, retroviral resistant HIV strain only equilibrium and both HIV strain coexist equilibrium, respectively. But we cannot find the antiretroviral sensitive HIV strain only equilibrium in chapter 3.2.1 in (Bhunu, 2009).

Obviously, the numbers of the equilibrium points could be greater than zero, then, we can get:

$$A > 0 \quad \Rightarrow \quad \frac{b_2(d_2 + q_2n + u)}{d_2q_2} > 1$$

$$B > 0 \quad \Rightarrow \quad R_2 = \frac{b_2(d_2 + q_2n + u)}{(d_2 + u)(q_2 + u)} > 1 \tag{3}$$

$$Y > 0 \quad \Rightarrow \quad R_1 = \frac{b_1(d_1 + q_n + e + u)}{(d_1 + e + u)(q_+u) - eq_1y} > 1 \tag{4}$$

where R_1, R_2 are equivalent to " R_1, R_2 " in chapter 3.2.1 in (Bhunu, 2009)(See Page 366).

Consequently, the Jacobian matrix at the equilibrium points $Q_i, i = 1, 2, 3$ is

$$J(Q_i) = [J_1, J_2, J_3, J_4, J_5],$$

where

$$
J_1 = \begin{bmatrix} \frac{b_1(I_1+nA_1)+b_2(I_2+nA_2)}{-(S+I_1+A_1+I_2+A_2)} + \frac{(b_1(I_1+nA_1)+b_2(I_2+nA_2))S}{(S+I_1+A_1+I_2+A_2)^2} - u \\ \frac{b_1(I_1+nA_1)}{S+I_1+A_1+I_2+A_2} - \frac{b_1(I_1+nA_1)S}{(S+I_1+A_1+I_2+A_2)^2} \\ 0 \\ \frac{b_2(I_2+nA_2)}{S+I_1+A_1+I_2+A_2} - \frac{b_2(I_2+nA_2)S}{(S+I_1+A_1+I_2+A_2)^2} \\ 0 \end{bmatrix}.
$$

$$
J_2 = \begin{bmatrix} \frac{-b_1 S}{S+I_1+A_1+I_2+A_2} + \frac{(b_1(I_1+nA_1)+b_2(I_2+nA_2))S}{(S+I_1+A_1+I_2+A_2)^2} \\ \frac{b_1 S}{S+I_1+A_1+I_2+A_2} - \frac{b_1(I_1+nA_1)S}{(S+I_1+A_1+I_2+A_2)^2} - q_1 - u \\ q_1 \\ \frac{-b_2(I_2+nA_2)S}{S+I_1+A_1+I_2+A_2} \\ 0 \end{bmatrix}.
$$

$$
J_3 = \begin{bmatrix} \frac{-b_1 nS}{S+I_1+A_1+I_2+A_2} + \frac{(b_1(I_1+nA_1)+b_2(I_2+nA_2))S}{(S+I_1+A_1+I_2+A_2)^2} \\ \frac{b_1 nS}{S+I_1+A_1+I_2+A_2} - \frac{b_1(I_1+nA_1)S}{(S+I_1+A_1+I_2+A_2)^2} + ye \\ -d_1 - e - u \\ \frac{-b_2(I_2+nA_2)S}{S+I_1+A_1+I_2+A_2} \\ (1-y)e \end{bmatrix}.
$$

$$
J_4 = \begin{bmatrix} \frac{-b_2 S}{S+I_1+A_1+I_2+A_2} + \frac{(b_1(I_1+nA_1)+b_2(I_2+nA_2))S}{(S+I_1+A_1+I_2+A_2)^2} \\ \frac{-b_1(I_1+nA_1)S}{S+I_1+A_1+I_2+A_2} \\ 0 \\ \frac{b_2 S}{S+I_1+A_1+I_2+A_2} - \frac{b_2(I_2+nA_2)S}{(S+I_1+A_1+I_2+A_2)^2} - q_2 - u \\ q_2 \end{bmatrix}.
$$

$$
J_5 = \begin{bmatrix} \frac{-b_2 nS}{S+I_1+A_1+I_2+A_2} + \frac{(b_1(I_1+nA_1)+b_2(I_2+nA_2))S}{(S+I_1+A_1+I_2+A_2)^2} \\ \frac{-b_1(I_1+nA_1)S}{S+I_1+A_1+I_2+A_2} \\ 0 \\ \frac{b_2 nS}{S+I_1+A_1+I_2+A_2} - \frac{b_2(I_2+nA_2)S}{(S+I_1+A_1+I_2+A_2)^2} \\ -d_2 - u \end{bmatrix}.
$$

2.3 The Bifurcation Diagram of Equilibrium Points

Take parameters from Table 1 and let $b_1, b_2 \in (0, 1)$ are variables, we can calculate the bifurcation of the CNN model Eq.(2) at the equilibrium points Q_1, Q_2 and Q_3 based on the local activity of CNN with five local state variables and one port (Dong, 2009), see Fig.2.

In Fig.2, the domains are coded as follows: edge of chaos (locally active and stable) domain (shown red), locally active and unstable domain (shown green) and locally passive domain (shown blue).

2.4 Simulations and Analysis

Take parameters from Table 1 and let b_1, b_2 be different numbers, we model the dynamic trajectories of Eq.(2) using MATLAB, see Table 2.

From EQ.(3) and Eq.(4) and take parameters list in Table 1, we can get

$$R_1 > 1 \quad \Rightarrow \quad b_1 > 0.0941 \tag{5}$$
$$R_2 > 1 \quad \Rightarrow \quad b_2 > 0.0969 \tag{6}$$

From Fig.2, Table 2, and Eq.(3)-Eq.(6), we can conclude:

1. When b_1, b_2 are both less than 0.0941, b_1, b_2 is located in the red domain of Fig.2(a)(the Edge of Chaos domain), $\max\{R_1, R_2\} < 1$, Eq.(2) converges to the equilibrium point Q_1 (disease-free) neglecting initial values which implies that there is no any HIV infected and AIDS individuals.

2. When b_1 or b_2 is greater than 0.969, Eq.(2) converges to the equilibrium point Q_2 or Q_3, which implies that there are HIV infected and AIDS individuals.

    (a) when $b_1 > 0.0969$ and b_2 is more less than b_1, b_1, b_2 are located in the blue domain of Fig.2(c) (the locally passive domain), Eq.(2) converges to the equilibrium point Q_3 (both HIV strain coexist equilibrium) and $I_2 < A_2$ neglecting initial values which implies that antiretroviral sensitive and resistant HIV infected and

AIDS individual coexist and the number of antiretroviral resistant AIDS individual is larger than the number of HIV infected. This suggests that the treatment intends to help the community by lengthening the lives of AIDS patients and reducing HIV/AIDS mortality and hence reducing the number of orphans in affected communities.

(b) when $b_1 > 0.0969$, and $b_2 < b_1$ but near b_1, b_1, b_2 are located in the red domain of Fig.2(c) (the Edge of Chaos domain), Eq.(2) converges to the equilibrium point Q_3 and $I_2 > A_2$ neglecting initial values which implies that antiretroviral sensitive and resistant HIV infected and AIDS individual coexist and the number of antiretroviral resistant AIDS individual is less than the number of HIV infected. This suggests that the treatment intends to prolong the incubation period only, but does not reduce infectiousness and take more HIV infections, thus may not benefit the community.

(c) When $b_1 > 0.0969$ and $b_2 \geq b_1$, b_1, b_2 are located in the red or blue but near red domain of Fig.2(b), Eq.(2) converges to the equilibrium point Q_2 (retroviral resistant HIV strain only equilibrium) and $I_2 > A_2$ neglecting initial values which implies that there are no antiretroviral sensitive HIV infected and AIDS individual, but antiretroviral resistant HIV infected and AIDS individual still exist. This suggests that the treatment is very effective to antiretroviral sensitive HIV infected and AIDS individual, but the antiretroviral sensitive HIV infected and AIDS individual are more difficult to be treated, which may not benefit the community.

In the following discussions, we select some parameters list in Table 2, the simulation results are shown in Fig.3 and Fig.4. Fig.3 model the convergence to equilibrium point Q_1, and Fig.4 model the situation when $b_1 = 0.6$ and b_2 varies from 0.011 to 0.095.

3. Conclusion and Future Works

A CNN model of two strain (antiretroviral sensitive and resistant) HIV is established, and then analyzed and simulated based on the local activity of CNN with five local state variables and one port (Dong, 2009). Numerical simulations exhibit that this CNN model may explain some complex phenomena during antiretroviral therapy which make it possible to judge the curative effect of long-time antiretroviral therapy and social influence.

Practically, the dynamic behaviors of HIV/AIDS infection and therapy are very complex and puzzling. Then, more and accurate experimental data are needed for modeling the dynamics of HIV/AIDS. Further research for HIV/AIDS dynamics is promising. More complex the model is, more approximate to the reality, but more difficult to be analyzed. The local activity of CNN provides a possible method for some of them.

All these imply that the local activity of CNN provides a practical tool for the study of the complex dynamics of some coupled nonlinear systems. Especially, it may be expected that CNN will become a promising candidate for modeling life phenomena.

Acknowledgement

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Table l. Model parameters and their interpretations

Parameter	Symbol	Value(/year)
Recruitment rate	a	0.029
Product of effectively rate and probability	b_1, b_2	0.011-0.95
Modification parameter	n	1.02
Natural mortality rate	u	0.02
Natural rate of progression of HIV	q_1, q_2	0.1
Proportion of effectively treated	y	0.2
Treatment rate for AIDS cases	e	0.33
AIDS related death rate	d_1, d_2	0.333-0.4

Table 2. Cell parameters and corresponding dynamic properties of the CNN of HIV/AIDS model

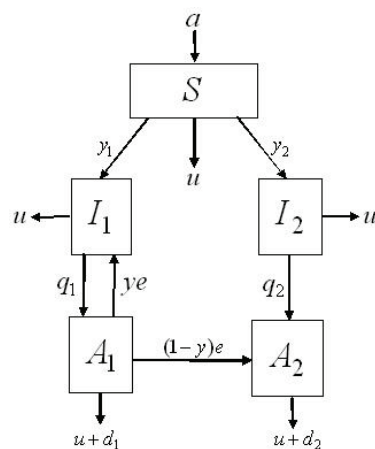| No.	b_1	b_2	Domain in Fig.1	Equilibrium point	Relation of I_2 and A_2	R_1	R_2
1	0.011	0.011	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.1140	0.1169
2	0.011	0.05	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.1140	0.5315
3	0.011	0.094	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.1140	0.9993
4	0.05	0.011	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.5181	0.1169
5	0.05	0.05	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.5181	0.5315
6	0.05	0.094	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.5181	0.9993
7	0.094	0.011	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.9740	0.1169
8	0.094	0.05	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.9740	0.5315
9	0.094	0.094	red in Fig.2(a)	Q_1	$I_2 = A_2$	0.9740	0.9993
10	0.011	0.097	red in Fig.2(b)	Q_2	$I_2 > A_2$	0.1140	1.0312
11	0.097	0.097	red in Fig.2(b)	Q_2	$I_2 > A_2$	1.0051	1.0312
12	0.097	0.011	blue in Fig.2(c)	Q_3	$I_2 < A_2$	1.0051	0.1169
13	0.2	0.011	blue in Fig.2(c)	Q_3	$I_2 < A_2$	0.2072	0.1169
14	0.2	0.08	blue in Fig.2(c)	Q_3	$I_2 < A_2$	0.2072	0.8505
15	0.2	0.094	blue in Fig.2(c)	Q_3	$I_2 < A_2$	0.2072	0.9993
16	0.2	0.095	red in Fig.2(c)	Q_3	$I_2 > A_2$	0.2072	1.0099
17	0.2	0.19	red in Fig.2(c)	Q_3	$I_2 > A_2$	0.2072	2.0198
18	0.2	0.2	red in Fig.2(b)	Q_2	$I_2 > A_2$	0.2072	2.1261
19	0.2	0.5	blue in Fig.2(b)	Q_2	$I_2 > A_2$	0.2072	5.3153
20	0.2	0.95	blue in Fig.2(b)	Q_2	$I_2 > A_2$	0.2072	10.099
21	0.6	0.011	blue in Fig.2(c)	Q_3	$I_2 < A_2$	6.2171	0.1169
22	0.6	0.36	blue in Fig.2(c)	Q_3	$I_2 < A_2$	6.2171	3.8270
23	0.6	0.4	red in Fig.2(c)	Q_3	$I_2 > A_2$	6.2171	4.2523
24	0.6	0.56	red in Fig.2(c)	Q_3	$I_2 > A_2$	6.2171	5.9532
25	0.6	0.6	blue in Fig.2(b)	Q_2	$I_2 > A_2$	6.2171	6.3784
26	0.6	0.95	blue in Fig.2(b)	Q_2	$I_2 > A_2$	6.2171	10.099
27	0.9	0.011	blue in Fig.2(c)	Q_3	$I_2 < A_2$	9.3256	0.1169
28	0.9	0.56	blue in Fig.2(c)	Q_3	$I_2 < A_2$	9.3256	5.9532
29	0.9	0.58	blue in Fig.2(c)	Q_3	$I_2 > A_2$	9.3256	6.1658
30	0.9	0.87	blue in Fig.2(c)	Q_3	$I_2 > A_2$	9.3256	9.2486
31	0.9	0.9	red in Fig.2(b)	Q_2	$I_2 > A_2$	9.3256	9.5676
32	0.9	0.95	red in Fig.2(b)	Q_2	$I_2 > A_2$	9.3256	10.099

Figure 1. Structure of the HIV/AIDS model

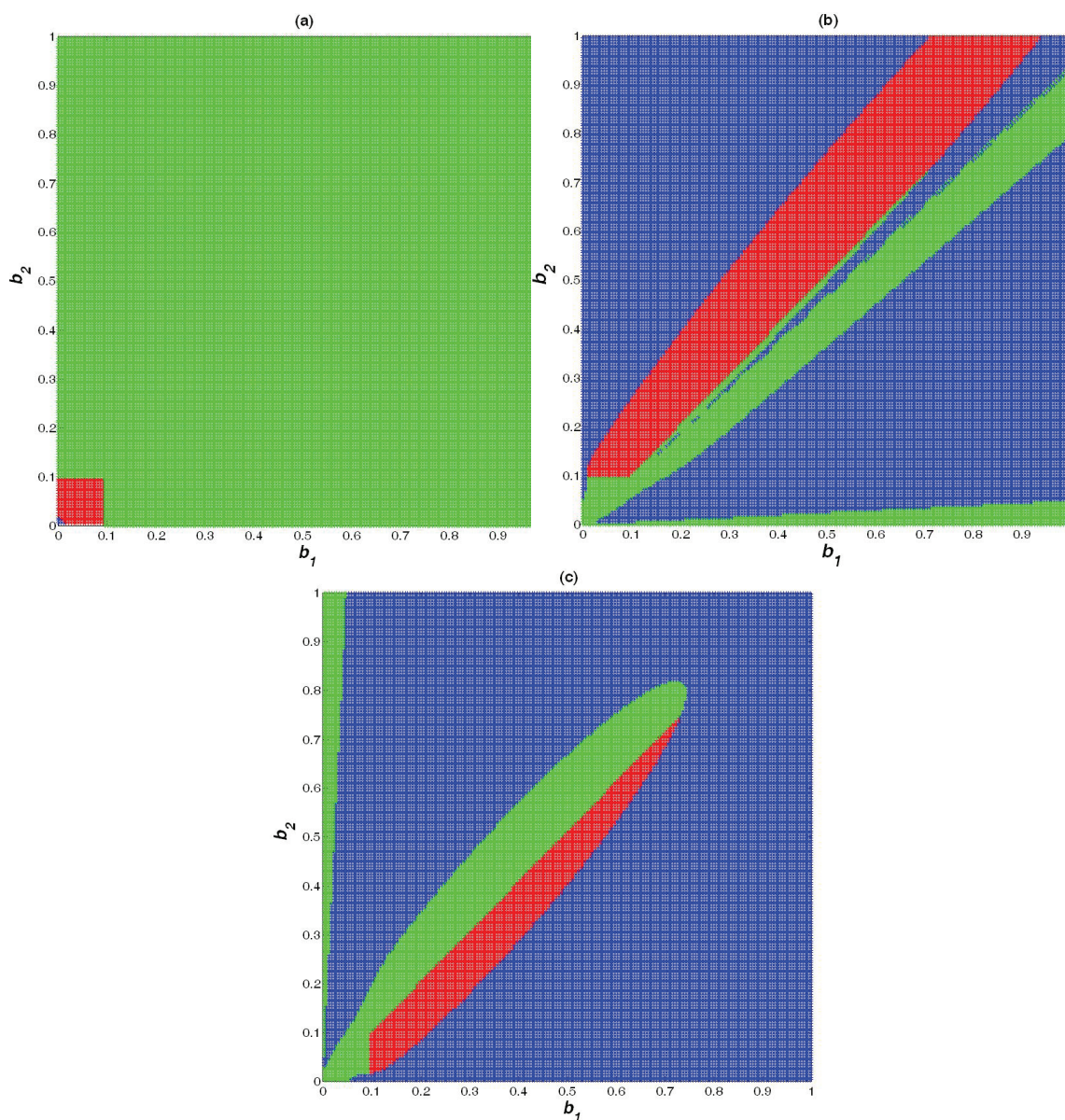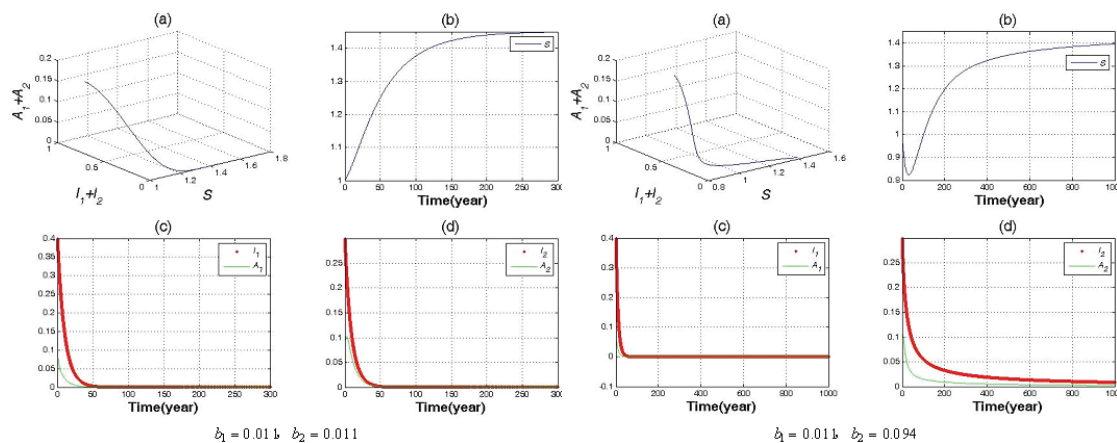Figure 2. Bifurcation diagrams of Eq.(3), when $b_1, b_2 \in (0, 1)$, at the equilibrium point: (a) Q_1,(b)Q_1,(c) Q_1
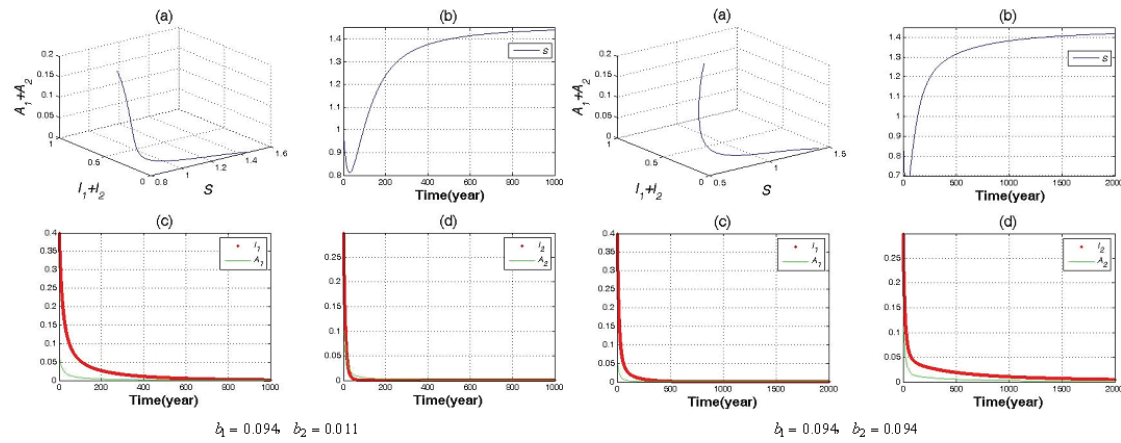
$b_1 = 0.094, \quad b_2 = 0.011$ $b_1 = 0.094, \quad b_2 = 0.094$

Figure 3. The trajectories of Eq.(2) converging to Q_1

$b_1 = 0.6, \quad b_2 = 0.011$ $b_1 = 0.6, \quad b_2 = 0.36$

$b_1 = 0.6, \quad b_2 = 0.4$ $b_1 = 0.6, \quad b_2 = 0.56$

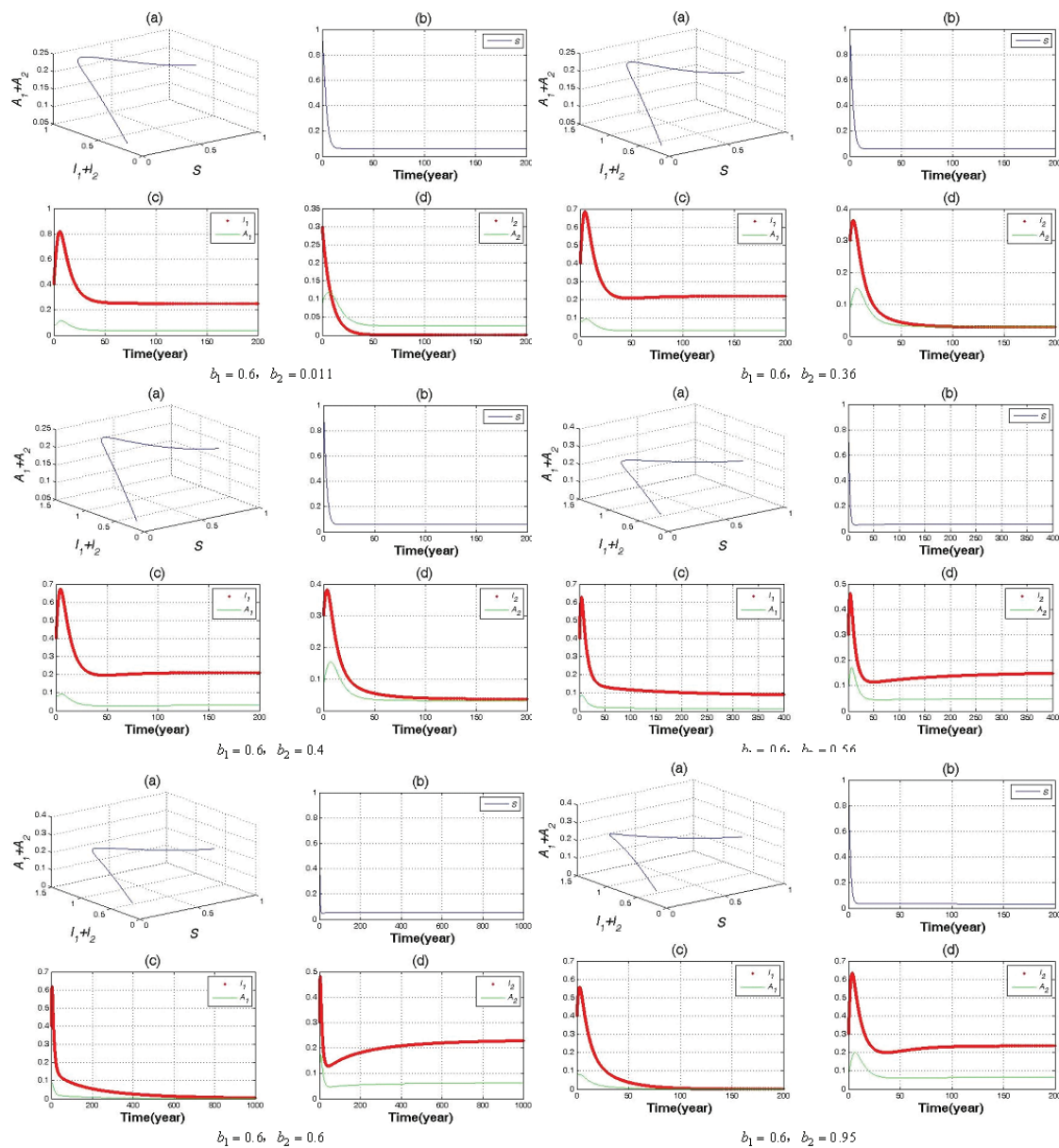$b_1 = 0.6, \quad b_2 = 0.6$ $b_1 = 0.6, \quad b_2 = 0.95$

Figure 4. The trajectories of Eq.(2) when $b_1 = 0.6$