Catastrophic chaos theory: predicting the edge between health and death

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Abstract: Recently, we proposed a macroscopic model for explaining the reason why organs such as a blast cyst, ectodermal endoderm, mesoderm, heart, and hand generate at about sevenfold cell divisions during the morphogenetic process and the biochemical standard clock such as circadian one. (Naitoh, AROBJ 2008, 2012, JJAIM 2011) The present standard clock model derived logically with experimental observations is described by a nonlinear differential equation predicting time-evolutions of six macroscopic molecular groups: three gene groups and three enzyme groups, which include acceleration and depression factors. In our previous reports (Naitoh, Proc. JSST, 2011, J. of Physics, 2012), we also find that the fundamental network pattern of neurons, which has been a mystery for a long time, will be dominated by the equation. Here, the macroscopic model extended for describing also aging processes shows various types of cycles and reveals the physical condition for determining whether or not living beings such as the human beings can survive after getting ill. It is stressed that, after getting ill, living systems with too fast generation of information molecules such as various genes result in the death, whereas too fast production of enzymes leads to regeneration. This may also explain an essential feature underlying cancerous process.

Keywords: Health, Death, Catastrophe, chaos.

1. INTRODUCTION

Our previous reports [1,2] clarified the minimum excitatory network of chemical reactions necessary for biological self-replication, which is the origin of life such as primitive bacteria and archaea. The minimum system as a closed loop has four types of macroscopic molecular groups: two information groups x11 and x12 and two functional molecular groups x21 and x22. (Fig. 1) It should be stressed that only three molecular groups are necessary for realizing the self-organizing replication, i.e., x11, x12, and x22, because x21 can be automatically generated by these three groups. As collision of two molecules generally occurs with a high probability in nonliving molecular pool, simultaneous collision of three molecules or impact of third molecules on two connecting molecules are possible with a certain probability, although simultaneous collision of four molecules is relatively difficult. Only three molecules of x11, x12, and x22 led to the emergence of living beings.

We can topologically see the symmetric and asymmetric circles of reaction networks in Fig. 1. [1,2] Fusion of asymmetric and symmetric size ratios of molecules (bipolarity of sizes of 1:1 and about 2:3) [1,4,5,7] will naturally result in the fusion of asymmetrical and symmetrical network patterns (bipolarity of topology).

Complementary pairs of RNA such as double-strand RNA (dsRNA), i.e., only one molecular group, may form the simplest excitatory cycle. However, information and a catalytic function are undetached in this type of system, because each strand of dsRNA has both of them. This leads to the fact that the dsRNA and DNA suitable for stabilizing information is not conducive to the production of various functions for inducing multi-cellar systems having complex geometries. Thus, living beings select the detachment of information and function. [13]

2.MORPHOGENETIC PROCESS

The main temporal mystery is the standard clock, i.e., the basic molecular instrument regulating the biological rhythm common to the cell cycle, proliferation and differentiation induced by the stem cell cycle, neural pulse, neural network, and circadian clock. In order to generate the standard clock, at least two more inhibitory molecules (molecular groups) of information and function should be added to depress a monotonous increase in DNA. [2-5, 10,11,12,13].

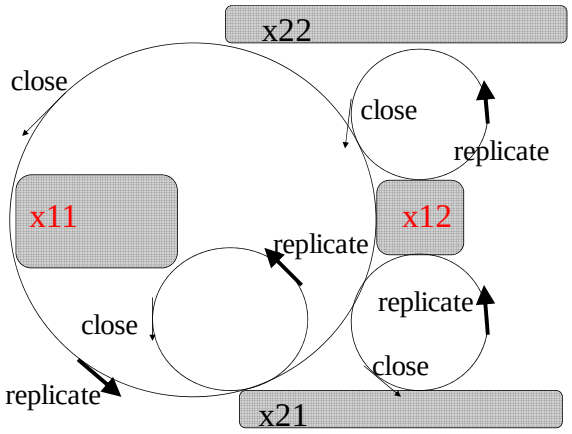

Fig. 1 Four-stroke molecular engine for living systems as minimum biological cycle

Here, we define x13 and x23 as the other molecular groups for inhibitory factors repressing reactions. These two groups are incorporated in the four groups of x11, x12, x21, and x22, because today's cells, the morphogenetic processes of multi-cellar systems, and neural systems use negative controllers such as Oct-4 and SOX2 for producing tissues and organs [6]. This leads to a macroscopic model having six types of molecular groups, or in other words, a six-stroke engine (Fig. 2). We can describe the densities of the six molecular groups x_{ij} at generation N after the mother cell generation in the morphogenetic process, by the following equations.

$$x_{1i}^{N+1} - x_{1i}^{N} = \alpha_{i1} x_{1i}^{N} \otimes x_{21}^{N} \quad ,(i = 1,2,3)$$
$$x_{2i}^{N+1} - x_{2i}^{N} = \alpha_{i2} \delta(x_{1i}^{N} - \xi_i x_{23}^{N}) \otimes x_{22}^{N} \quad ,(i = 1,2,3) \tag{1}$$

where $x_{ij} \otimes x_{km}$ denotes the smaller value among x_{ij} and x_{km} and also where $\delta(x)$ and α_{ij} denote the larger value of x or 0, i.e., max (x, 0) and constant of reaction probability, respectively.[2-5] Statistical mechanics inevitably leads to the mathematical form on the right-hand side in Eq. 1, because of collision probability.

Numerical solutions for Eq. 1 show about a sevenfold beat cycle of densities for molecular groups on average, while varying the parameters in Eq. 1 results in four- to ten- fold beat cycles.

An important point is that the actual morphogenetic processes show about seven-beat cycles of molecular densities [2-5].

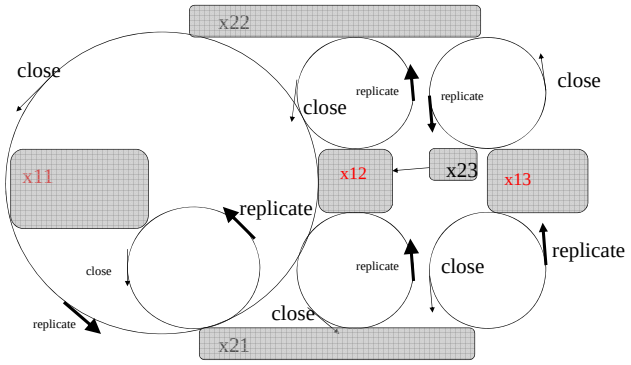

Fig. 2 Six-stroke molecular engine including depression effect

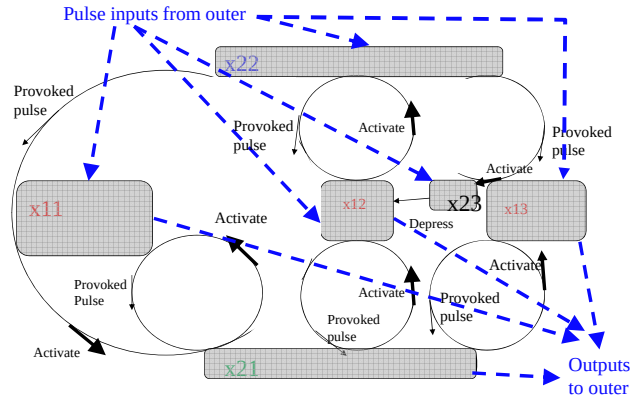

Fig. 3 Six cellar network for the brain system

3. NEURAL TOTWORK

Equation 1 will also reveal the standard topology of the cortical neural circuit (network), the integration mechanism of brain functions, the neural system for muscle control, and the chemical reaction network inside a neuron [3,5,7].

Figure 3 shows the standard pattern for neural networks, which includes inputs and outputs. For the network, six variables of x_{ij} (i=1-2, j=1-3) are redefined as the activation level of neurons, related to the density of molecules and amount of total energy inside the neurons.

There are two sides in Fig. 3, one for inputs and the other for outputs, which correspond to information and functional molecules in Eq. 1 and Fig. 3. The upside-down topology of inputs and outputs in Fig. 3 will also be possible.

The most important point is that the present equation (Eq. 1) and Fig. 3 describe the essential physics underlying the network of neural cells and the molecular network inside a neuron, whereas the Hodgkin-Huxley (H-H) model [8] describes only the outer electrical quantities such as electron flow and voltage for a single neuron. [It should be added that some variations modified from the network pattern in Fig. 3 and Eq. 1 are also possible, by varying the arbitrary constants and also by adding more molecular types except for x13 and x23.]

It should be stressed that the circadian clock of about 24-25 hours are also seven times the fundamental temperature oscillation of about 3.5 hours. [9]

Equation 1 is an ordinary differential one that eliminates spatial variations of quantities, because the spatial diffusion of molecules and cells is relatively fast in comparison with temporal oscillations and also because a lot of molecules move between cells.

Moreover, emphasis is placed on the fact that the cycles of boom and bust appearing in economic and social systems are also the seven-beat on average, because economic systems are produced by human brains. Flux and reflux of companies and capital can also be clarified by the present analysis. [2]

4 MODEL EXTENDED WITH TOTAL ENERGY LIMIT

The model (Eq. 1) in the previous sections was derived under the assumption of an infinite energy supply. However, energy supplied for molecular networks, cell colonies, organs, neural networks, or economic systems will be limited, because the surface-to-volume ratio of each system decreases according to an increase in the number of molecules, cells, neurons, or populations, leading to a condition of insufficient energy. Thus, a new energy restriction term should be added to Eq. 1, which results in Eq. 2 [3, 7].

$$x_{ij}^{N+1} - x_{ij}^{N} = \alpha_{ij}(x_{1j}^{N} - \beta_{ij} x_{23}^{N}) \otimes x_{2i}^{N} - \varepsilon_{ij} [x_{ij}^{N}]^{q},$$
$$x_{ij} \geq 0, \quad x_{1j}^{N} - \beta_{ij} x_{23}^{N} \geq 0. \ (i = 1-2, j = 1,-3) \tag{2}$$

where $q > 2$ is set in case that the symbol $\otimes$ is defined as product, whereas $q > 1$ if the symbol $\otimes$ means smaller value among two.

Let us solve the time-dependent process including morphogenesis and aging processes by using Eq. 2. Numerical solutions for the equation extended with total energy limit show a transition to sick situation such as cancer in the aging process of the human beings including the brain, i.e., a mysterious transition from chaotic oscillation at 2nd stage (from 80 to 200 generation) to periodic one at 3rd stage (after about 250), while the

vibration amplitude keeps a constant level. (Fig. 4)

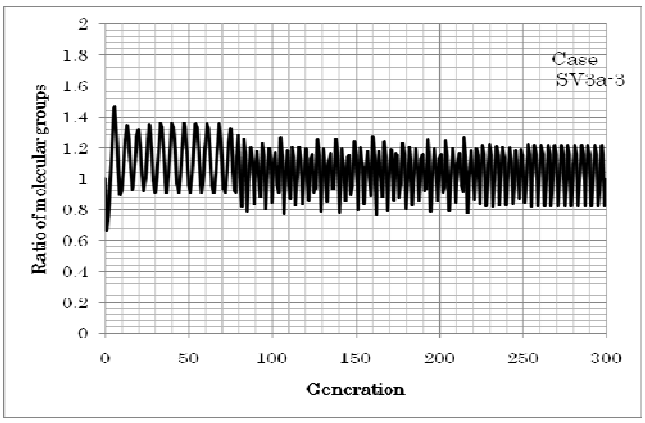

Fig. 4.Transition to sick condition as the 3rd stage in the aging process of the human being including the brain.

5. DEATH OR REGENERATION

Here, we further examine the mysterious equation (Eq. 2) by varying the parameters in detail. Increasing values of α_{11} and α_{21} result in very rapid decreases of densities for some molecular groups: biological catastrophe. An important point is that there are two essential patterns of the catastrophic processes: death and regeneration from asphyxia. (See Figs. 5 and 6) Figure 5 demonstrates the regeneration from asphyxia, whereas Fig. 6 shows the death, because densities of molecular groups cannot increase again. The critical condition for determining whether or not the living system comes back is related to the densities D1, D2, and D3 of information groups x11, x12, and x13 just after the crash occurred. If the densities of D1, D2, and D3 are zero, the death comes.

6. CONCLUSION

The present model clarifies the edge between the death and regeneration after becoming ill. Excessive replication of information molecules leads to the death of information system, which results in the death of the whole system.

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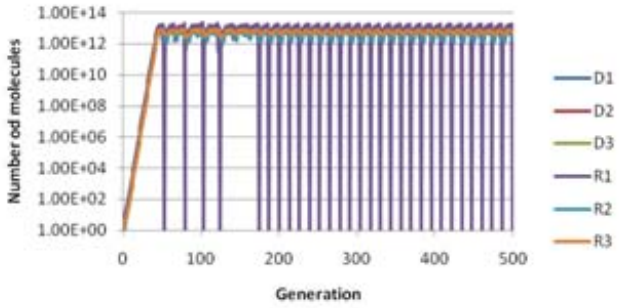

Fig. 5 Recoverable catastrophes appearing in the solution for Eq. 2.
$\alpha_{11}=1.0, \alpha_{12}=1.0, \alpha_{13}=1.0, \alpha_{21}=3.2, \alpha_{22}=1.0, \alpha_{23}=1.0, q=2.0, \varepsilon_{ij}=1.0\times10^{-13}$

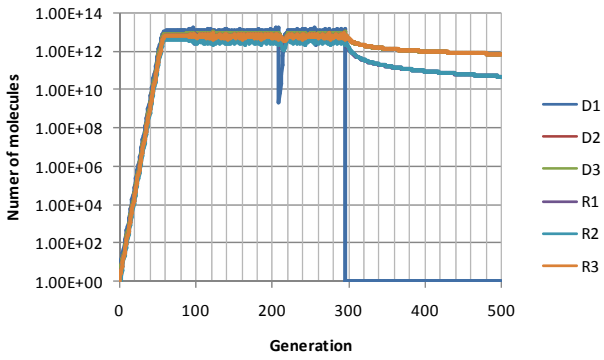

Fig. 6 Unrecoverable catastrophe seeing in the solution of Eq. 2 in case of very high increase of information proteins.
$\alpha_{11}=2.0, \alpha_{12}=1.0, \alpha_{13}=1.0, \alpha_{21}=1.0, \alpha_{22}=1.0, \alpha_{23}=1.0, q=2.0, \varepsilon_{ij}=1.0\times10^{-13}$