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Optimization of Distributed Autonomous Robotic Systems Based on Artificial Immune Systems

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Abstract **- In this paper, we optimize distributed autonomous robotic system based on artificial immune system. Immune system has B-cell and T-cell that are two major types of lymphocytes. B-cells take part in humoral responses that secrete antibodies and T-cells take part in cellular responses that stimulate or suppress cells connected to the immune system. They have communicating network equation, which have many parameters. The distributed autonomous robotics system based on this artificial immune system is modeled on the B-cells and T-cells system. So performance of system is influenced by parameters of immune network equation. We can improve performance of Distributed autonomous robotics system based on artificial immune system.**

I. INTRODUCTION

Distributed Autonomous Robotic System (DARS) is that each robot perceives its environments such as an object and the other robot's behavior etc., and they determine their behavior independently, and cooperate with the other robots in order to perform the given tasks very well. DARS has no function to integrate information of environment. But a robot individually understands surrounding and behavior of other robots, and decides its behavior autonomously to cooperate with other robots. The whole system is composed with individual robots' behavior. Biological immune system is also a distributed autonomous system. It is not a centralized control system. But it can cope with varying environment autonomously. So immune system is applied by a distributed autonomous robotic system [1].

Artificial immune system is the model of a biological immune system. The basic components of artificial immune system are B-cell and T-cell, which are two major types of lymphocytes. B-cell takes part in humoral responses that secrete antibodies and T-cells take part in cellular responses that stimulate or suppress cells connected to the immune system. Operation of B-cell and T-cell can express artificial immune network equations [2]. These equations renew a stimulated value of antibody. Also antibody is stimulated or suppressed from antigen, other antibodies and T-cell. Ratios of these are controllable and affect performance of system.

In this paper we find an optimal parameter of the composed system by genetic algorithm. The basic operators of genetic algorithm are crossover and mutation. Crossover selects two individuals randomly and changes genes. Mixing the elite individuals' gene makes new better elites. Mutation overcomes the limit of crossover. It changes a gene into opposition gene. Transformation of gene gives variety to population. As features of genetic algorithm, there are a coded parameter, parallel searching, blind searching, and probable operator. These things help us to find optimal solution of the system. Also genetic algorithm can find optimal solution better than process of trial and error. So we find optimal solution at this problem by using genetic algorithm.

II. CONCEPT OF IMMUNE SYSTEM

A. Clonal Selection

Each lymphocyte (whether B cell or T cell) is genetically programmed to be capable of recognizing essentially only one particular antigen. The immune system as a whole can specifically recognize many thousands of antigens, so the lymphocyte recognizing any particular antigen must represent only a minute proportion of total. How then is an adequate immune response to an infectious agent generated? The answer is that when an antigen binds to the few cells that can recognize it, they are induced to proliferate rapidly. Within a few days there are a sufficient number to mount an adequate immune response. In other words, the antigen selects for and generates the specific clones of its own antigen- binding cells, which is called a clonal selection. This operates for both B cells and T cells [2].

Lymphocytes that have been stimulated by binding to their specific antigen take the first steps towards cell division. They express new receptors that allow them to respond to cytokines from other cells, which is signal proliferation. The lymphocyte may also start to secrete cytokines themselves. They will usually go through a number of cycles of division, before differentiating into mature cells, again under the influence of cytokines. For example, proliferating B cells eventually mature into antibody-producing plasma cells. Even when the infection has been overcome, some of the newly

produced lymphocytes remain, available for re-stimulation if the antigen is encountered once more. These cells are called memory cells, since they retain the immunological memory of particular antigen. It is memory cells that confer the lasting immunity to a particular pathogen.

Fig. 1 shows operation of B cell clonal selection that is basic response of immune system.

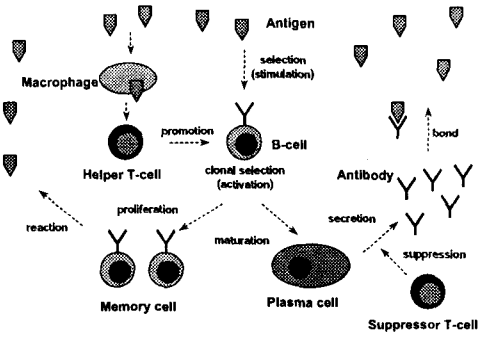

Fig. 1. Operation of T-cell and B-cell clonal selection.

B. Idiotopic Network

Tolerance to self-antigens is established during onto gene. However, during the neonatal period the unique binding regions of antigen-specific receptors on B-cells (antibody) and T-cells are present at levels that are too low to generate tolerance. This antigen-specific site is called an idiotope. Thus antibodies (B-cells) are stimulated not by antigens but also by other antibodies (B-cells). Jerne who is an immunologist proposed idiotopic network hypothesis (immune network hypothesis) based on mutual stimulus and suppression between antibodies. According to this hypothesis, antibodies interact with each other through idiotope and paratope. Such a network relationship plays an important role that keeps up the concentration of antibody in the immune system. Therefore, immune system is parallel-distributed system that operates on not a unit level but system level.

C. Immune Learning and Immune Memory

It is clear that immune response is not merely a matter of checking a pre-determined set of possible paratopes against the epitope. Somehow, the immune system has to actually solve the optimization problem of finding an antibody with a paratope that very nearly "matches" a given epitope. It has to recognize a given epitope.

The immune system generates antibodies that can recognize an enormous range of antigens even before it encounters them. Many of these will never be called upon to protect individually against infection. However, the tremendous numbers of infectious organisms and their capacity to change their antigens through mutation make it necessary for all these different antibodies to be available, just in case they are ever needed.

Regarding the nature of the randomized replacement which gives rise to high degrees of mutation. There are several different hypothesis. For instance, it is not inconceivable that positions are filled at random by errors introduced in the process of repair. In the process of repair or mutation, the chromosome of B cell changes. This leads to change of variable region (see Fig. 1). By clonal selection and network hypothesis, if B cells (antibodies) that have close to antigen-binding site are proliferation, finally B cell that has exact antigen-binding site is produced. This mechanism is immune learning. As a result of this, newly lymphocytes are remaining. This lymphocyte is called memory cell that immunity can hold the same antigen.

III. Immune Network Model of B-cell and T-cell

Equation (1)-(3) are the modified immune network equations that are modeled relationship of antigen, B-cell (antibody), and T-cell of immune system.

$$S_i(t) = S_i(t-1) + \left(\left(\alpha \frac{\sum_{j=1}^{N} (m_{ij} s_i(t))}{N} - \alpha \frac{\sum_{j=1}^{N} (m_{ki} s_k(t))}{N} + \beta g_i - c_i(t-1) - k_i \right) s_i(t) \right) \quad (1)$$

$$s_i(t) = \frac{1}{1+\exp(0.5 - S_i(t))} \quad (2)$$

$$c_i(t) = \eta(1 - g_i(t)) S_i(t) \quad (3)$$

Where $i, j = 0, N\text{-}1$, N is a number of antibody types, $S_i(t)$ is stimulus value of antibody i, $s_i(t)$ is concentration of antibody i, $s_j(t)$ is not concentration of self-antibody but concentration of other robot's antibody getting by communication, $c_i(t)$ is concentration of T-cell which control concentration of antibody, m_{ij} is mutual stimulus coefficient of antibody i and j, g_i is affinity of antibody i and antigen, α,β,η are constants. Table 1 is role of T-cell when in different states.

Table 1. The role of T-cell in different states.

$g_i(t)$	$S_i(t)$	$c_i(t)$	state	Role of T-cell
big	small	very small	antigen invading	helper T-cell
big	Big	small	eliminating	-
small	Big	big	eliminated	suppressor T-cell
small	small	small	stable	-

IV. Modeling Of Group Behavior

The objective of the system is for robots to find and eat foods; the foods spread out in the environment[1-2]. The density of the food is classified into four levels that are high, medium, low, and nothing. A robot includes several strategies such as aggregation, random search, dispersion, and homing. Also the robot eats the food when distance between agent and food is less than a predefined constant distance. Aggregation is the ability to gather a group of agents in order to establish and maintain some maximum inter-agent distance. Random search is a finding food by moving random direction. Dispersion is to spread out a group of agent in order to establish and maintain some minimum inter-agent distance. Homing is a finding and going a particular region or location. Stimulus value of antigen to antibody is defined as Fig 2, according to the number of food detection during past given times.

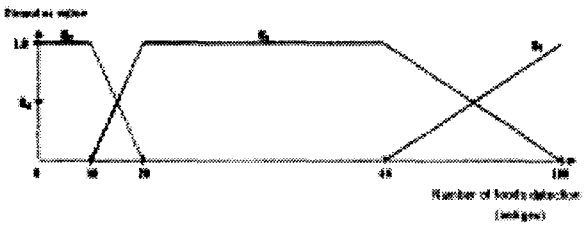

Fig. 2. Stimulus function of antigen to antibody (g_i).

IV. Genetic Algorithms

Genetic algorithms (GA) are derivative-free stochastic optimization methods based loosely on the concepts of natural selection and evolutionary processes. They were proposed and investigated by John Holland. Characters of GA are parallel-search, applicable to both continuous and discrete problems, flexibility facilitates both structure and parameter identification and stochastic and less likely to get trapped in local minima

Major components of GA include encoding schemes, fitness evaluations, parent selection, crossover operators, and mutation operators. Encoding schemes is transform points in parameter space into bit string representations. Fitness evaluation is calculating the fitness value of each member in the population when the first step after creating a generation. Selection operation determines which parents participate in producing offspring for the next generation. Crossover is usually applied to selected pairs of parents with a probability equal to a given crossover rate. Mutation operator generates new chromosomes [3-5].

V. Optimization Of Immune System

We use GA to optimizing immune network. The immune network equation has a mutual stimulus coefficient and 4 parameters. Mutual stimulus is determined by a trial and error. Since parameters are very sensitivity, we determine those by GA operation. GA's parameters are established like table 2. Also we use a roulette selection, and one point crossover. Evaluation of system is how much whole system is similar the stimulus function of antigen that we proposed. High fitness has small error between calculated system and simulated system.

Table 1. Parameter

Population Size	20
Resolution	8(bits)
Crossover Rate	0.6
Mutation Rate	0.03

Fig. 3 is a fitness landscape, which included maximum and average fitness of each generation. Fig 4 is percentage graph of antibody in the aiming system, and Fig 5 is measurement graph of antibody in the simulated system.

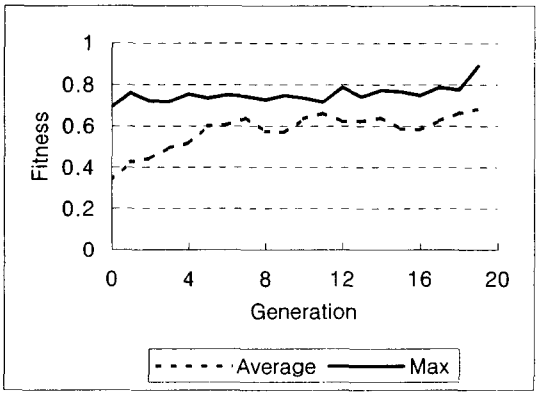

Fig. 3. Fitness landscape

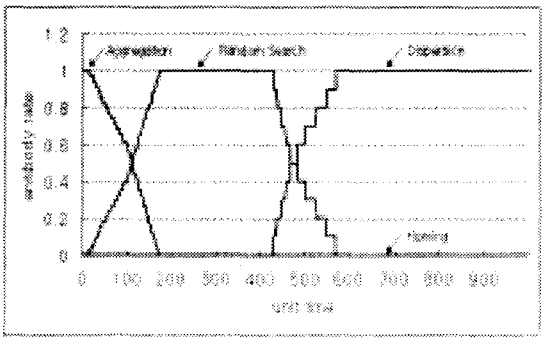

Fig. 4. ratio of aiming system.

The fitness landscape (Fig. 3) shows that parameters are influencing performance of system. Also Fig 4 and Fig 5 show that parameters are influencing a process between immune network and environment. Fig. 4 is a ratio of aiming system by stimulus function of antigen to antibody and Fig. 5 is a measured number of antibodies of simulated system. We can see that the graph of antibody follow ratio of aiming system.

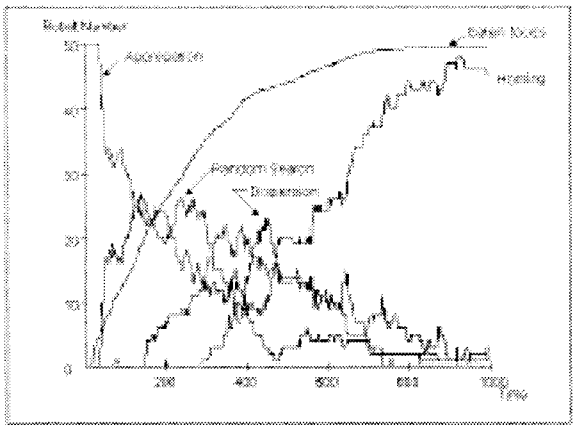

Fig. 5. Measurement of antibody of simulated system.

VI. Conclusion

This paper shows that the performance of DARS based on AIS can be improved by adjusting parameter of immune network. Also we find the optimal parameter of immune network in the proposed system. Optimized DARS almost satisfy requirement of the whole system. However, the optimization of parameter is not perfect, because immune network has other components, which influence the system. The mutual coefficients are constant but they can control the operation of the system. Therefore we must study on the optimization of coefficient.

Acknowledgments

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