

면역 알고리즘을 이용한 PID 제어기의 지능 튜닝

論 文

51D-1-2

Intelligent Tuning Of a PID Controller Using Immune Algorithm

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Abstracts - This paper suggests that the immune algorithm can effectively be used in tuning of a PID controller. The artificial immune network always has a new parallel decentralized processing mechanism for various situations, since antibodies communicate to each other among different species of antibodies/B-cells through the stimulation and suppression chains among antibodies that form a large-scaled network. In addition to that, the structure of the network is not fixed, but varies continuously. That is, the artificial immune network flexibly self-organizes according to dynamic changes of external environment (meta-dynamics function). However, up to the present time, models based on the conventional crisp approach have been used to describe dynamic model relationship between antibody and antigen. Therefore, there are some problems with a less flexible result to the external behavior.

On the other hand, a number of tuning technologies have been considered for the tuning of a PID controller. As a less common method, the fuzzy and neural network or its combined techniques are applied. However, in the case of the latter, yet, it is not applied in the practical field, in the former, a higher experience and technology is required during tuning procedure. In addition to that, tuning performance cannot be guaranteed with regards to a plant with non-linear characteristics or many kinds of disturbances.

Along with these, this paper used immune algorithm in order that a PID controller can be more adaptable controlled against the external condition, including noise or disturbance of plant. Parameters P, I, D encoded in antibody randomly are allocated during selection processes to obtain an optimal gain required for plant. The result of study shows the artificial immune can effectively be used to tune, since it can more fit modes or parameters of the PID controller than that of the conventional tuning methods.

Keyword: Immune Network, PID controller, Intelligent tuning.

1. Introduction

In recently years, a combined learning-based artificial intelligence (AI) such as neural network, genetic algorithm, and immune network structure have been interested in studying much attention for their robustness and flexibility against a dynamically changing system or complex system, since conventional artificial intelligent systems based on a functional decomposition, leading to a so-called sense-model-plan-action cycle have been criticized. Introduction

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systems based on a functional decomposition, leading to a so-called "Sense-model-plan-action" cycle have been criticized on many grounds over the last decade [1-3].

They are used extensively on industry in such diverse applications as fault prediction, fault diagnosis, supervisory control, energy management, production management, software engineering, and among others [4-5].

It is a challenge in control and computer communities to explore novel control strategies and philosophies for complex industrial processes. The application of intelligent system technologies in industrial control has been developing into an emerging technology, so-called "Industrial intelligent control" This technology is highly multi-disciplinary and rooted in systems control, operations research, artificial intelligence, information and signal processing, computer software and production background [2].

Each technique such as fuzzy, neural, and neuro-fuzzy is offering new possibilities and making intelligent system even more versatile and applicable in an ever-increasing range of industrial applications. Over the past decade or

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接受日字 : 2001年 8月 16日
最終完了 : 2001年 12月 18日

so, significant advances have been made in two distinct technological areas: fuzzy logic (FL) and neural networks (NNs) [1, 2].

There have been considerable interests for the past few years in exploring the application of fuzzy or neural network (FNN) systems, which combine the capability of fuzzy reasoning to handle uncertain information and the capability of artificial networks to learn from processes, to deal with nonlinearities and uncertainties of control systems.

On the other hand, biological information processing systems such as human beings have many interesting functions and are expected to provide various feasible ideas to engineering fields, especially intelligent control or robotics. Biological information in living organisms can be mainly classified into the following four systems: brain nervous, genetic system, endocrine system, and immune system. Among these systems, brain nervous and genetic systems have already been applied to engineering fields by modeling as neural network and genetic algorithms, they have been widely used in various fields. However, Only a little attention has been paid to application of the other systems, not to mention their important characteristics and model.

The purpose of the artificial immune system (AIS) is to implement a learning technique inspired by the human immune system which is a remarkable natural defense mechanism that learns about foreign substances. However, the immune system has not attracted the same kind of interest from the computing field as the neural operation of the brain or the evolutionary forces used in learning classifier systems [7].

The immune system is a rich source of theories and as such can act as an inspiration for computer-based solutions. The learning rule of the immune system is a distributed system with no central controller, since the immune system is distributed and consists of an enormous number and diversity of cells throughout our bodies.

The immune system has a naturally occurring event-response system which can quickly adapt to changing situations and shares the property with the central nervous system that a definite recognition can be made with a fuzzy stimulus. Since the immune system possesses a self organizing and distributed memory, it is thus adaptive to its external environment and allows a PDP (Parallel Distributed Processing) network to complete patterns against the environmental situation. The correct functioning of the immune system is to be insensitive to the fine details of the network connections, since a significant part of the immune system repertoire is

generate by somatic mutation processes.

In particular, immune system have various interesting features such as immunological memory, immunological tolerance, so on viewed from engineering. That is, it can play an important role to maintain own system dynamically changing environments. Therefore, immune system would be expected to provide a new paradigm suitable for dynamic problem including control problem dealing with unknown environments their rather than static system [7, 8].

Brooks, a pioneer of the approaches, has presented basic architecture for behavior arbitration of autonomous robots [1]. He has argued that intelligence should emerge from mutual interactions among competence modules (i.e. simple behavior/action), and interactions between a robot and its environment. However, the behavior based AI still has the following open questions: how do we construct an appropriate arbitration mechanism among multiple competence modules, how do we prepare appropriate competence modules. One of the promising approaches to tackle (target) the problem mentioned above is a biologically-inspired approach for AIS.

From the above facts, some researchers particularly focused on the similarities between the behavior arbitration system (or neural network) and the immune system, and have proposed a new decentralized consensus-making system inspired by the biological immune system in [3, 4], since both systems deal with various sensory inputs (antigens) through interactions among competence modules (lymphocytes and/or antibodies).

From this study, it would be expected there is an interesting AI technique suitable for dynamically changing environments by imitating the immune system in living organisms. However, the determination of the appropriate suggestion of competence modules (antibodies) or arbitrary affinity still remains an open question. It also need to try to incorporate an off-line meta-dynamics function into the previously proposed artificial immune network in order to autonomously construct appropriate immune networks [3-4]. However, the resulting stimulation signal is not delicate since they used the crisp mathematical approach.

This paper suggests the AIS based tuning technique for PID controller that can cope with a dynamic and complicated environment from the immunological standpoint inspired by dynamic equations.

2. Characteristic Of The Artificial Immune System

This paper suggests that the immune algorithm can

effectively be used in tuning of a PID controller. The artificial immune network always has a new parallel decentralized processing mechanism for various situations, since antibodies communicate to each other among different species of antibodies/B-cells through the stimulation and suppression chains among antibodies that form a large-scaled network. In addition to that, the structure of the network is not fixed, but varies continuously. That is, the artificial immune network flexibly self-organizes according to dynamic changes of external environment (meta-dynamics function). However, up to the present time, models based on the conventional crisp approach have been used to describe dynamic model relationship between antibody and antigen. Therefore, there are some problems with a less flexible result to the external behavior.

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Along with these, this paper used immune algorithm in order that a PID controller can be more adaptable controlled against the external condition, including noise or disturbance of plant. Parameters P, I, D encoded in antibody randomly are allocated during selection processes to obtain an optimal gain required for plant. The result of study shows the artificial immune can effectively be used to tune, since it can more fit modes or parameters of the PID controller than that of the conventional tuning methods.

The immune system protects our bodies from attack of foreign matters (antigens) which enter the bloodstream. One way in which immune system does this is by using antibodies which are proteins produced by B cells, which are a subpopulation of white blood cells.

The basic components of the biological immune system are macrophages, antibodies, and B-cell. B-cell is the cells maturing in bone marrow, which collectively form what is known as the immune network. Roughly EMBED Equation.3 distinct types of B-cell are contained in a human body, each of which has a distinct molecular structure and produces "Y" shaped antibodies from its surfaces [4]. When a B cell encounters an antigen, an artificial immune response is elicited, which causes the

antibody matches the antigen sufficiently well, its B cell becomes stimulated and can produce mutated clones which are incorporated into the immune network. That is, the antibody recognizes specific antigens which are the foreign substances that invade living creatures, and this reaction is often likened to a key and keyhole relationship. This network hypothesis is the concept that antibodies/B-cells are not just isolated, that is they are communicating to each other among different species of antibodies/B-cells. As illustrated in Fig.1, the stimulation and suppression chains among antibodies form a large-scaled network and works as a self and not-self recognizer. Therefore, the immune system is expected to provide new parallel decentralized processing for various situations. Furthermore, the structure of the network is not fixed, but varies continuously. It means that the artificial network flexibly self-organizes according to dynamic changes of external environment. This remarkable behavior, called metadynamics function, is mainly realized by incorporating newly-generated cells/antibodies and/or removing useless ones. The new cells are generated by both gene recombination in bone marrow and mutation in the proliferation process of activated cells. Although many new cells are generated every day, most of them have no effect on the existing network and soon die away without any stimulation [4-5].

3. Dynamic Characteristic In Immune System

3.1 Structure of Immune System

Jerne first point out the idea that there are some remarkable similarities between the nervous system and the immune system [4] and he also proposed that the immune response is regulated by a network of interactions in antibodies. That idea has been elaborated by Cohn, Edelman & Mountcastle, and Edelman & Reeke [9].

John E. Hunt & Denise E. Cooke described an artificial immune system which is based upon models of the natural immune system and Geoffrey W. Hoffman suggested a neural network model based on the analogy with the immune system.

In engineering field, robot, decentralized automation, data mining, memory, automatic control have been studied. To understand for model exactly, we need to figure out how they are constructed among the structures in immune system [5-7].

3.2 The Response of Immune System

The immune system has two types of response:

primary and secondary. If the immune system encounters the antigen for the first time, the primary reaction happens. At that point, the immune system learns about the antigen, thus the immune network prepares the body for any further invasion from that antigen. This learning mechanism creates the immune system memory [1].

The secondary response occurs when the same antigen encountered again. The characteristic of this response is to implement a more adaptive capability by a more rapid and more abundant production of antibody resulting from the priming of the B-cells in the primary response.

3.3 Antibodies in Immune System

Antibody is actually three-dimensional Y shaped molecules which consist of two types of protein chain: light and heavy. The function of antibody identifies the antigen they can bind by performing a complementary pattern match. It also possesses two paratopes which represents the pattern it will use to match the antigen. The regions on the molecules that the paratopes can attach are so-called epitopes [3, 7].

3.4 Interaction Between Antibodies

Describing the interaction among antibodies is important to understand dynamic characteristics of immune system. For the ease of understanding, consider the two antibodies that respond to the antigens A1 and A2, respectively. These antigens stimulate the antibodies, consequently the concentration of antibody A1 and A2 increases. However, if there is no interaction between antibody A1 and antibody A2, these antibodies will have the same concentrations [1, '3, 4]. Suppose that the idiotope of antibody A1 and the paratope of antibody A2 are the same. This means that antibody II is stimulated by antibody I, and oppositely antibody I is suppressed by antibody II as Fig. 1. In this case, unlike the previous case, antibody II will have higher concentration than antibody I. As a result, antibody II is more likely to be selected.

This means that antibody II has higher priority over antibody I in this situation. As we know from this description, the interaction among the antibodies acts based on the principle of a priority adjustment mechanism. The match algorithm is used for counting match level between the antigen and the antibody. The strength of the bind between two substances depends on how closely the two match, and the closer the match between antibody and antigen the stronger the molecular binding and the better the recognition.

3.5 Dynamics of Immune System

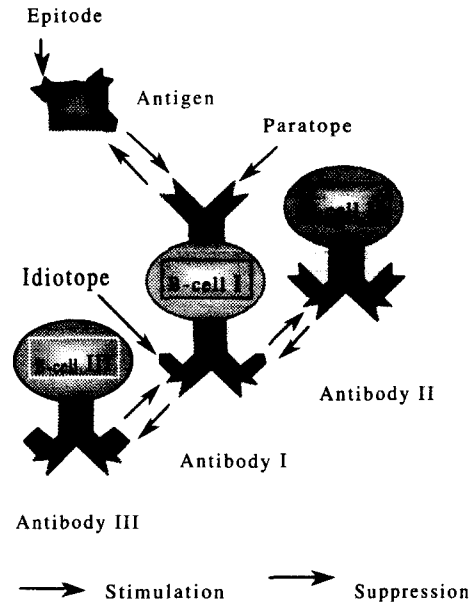


Fig. 1 Structure of idiotypic on Jerne network.

In the immune system, the level to which a B cell is stimulated relates partly to how well its antibody binds the antigen. We take into account both the strength of the match between the antibody and the antigen and the B cell objection affinity to the other B cells as well as its enmity. Therefore, generally the concentration of i -th antibody, which is denoted by δ_i , is calculated as follows [3]:

$$\frac{dS_i(t)}{dt} = \left(\begin{array}{l} \alpha \sum_{j=1}^N m_{ji} \delta_j(t) \\ -\alpha \sum_{k=1}^N m_{ik} \delta_k(t) + \beta m_i - \gamma_i \end{array} \right) \delta_i(t) \quad (1a)$$

$$\frac{d\delta_i(t)}{dt} = \frac{1}{1 + \exp\left(0.5 - \frac{dS_i(t)}{dt}\right)} \quad (1b)$$

where in equation (1), N is the number of antibodies, α and β are positive constants. m_{ji} denotes affinities between antibody j and antibody i (i.e. the degree of interaction), m_i represents affinities between the detected antigens and antibody i , respectively [3,4,7,8]. On the other hand, information obtained in lymphocyte population can be represented by [10]:

$$\Omega_j(N) = \sum_{i=1}^S -x_{ij} \log x_{ij} \quad (2)$$

where N is the size of the antibodies in a lymphocyte

population, S is the variety of allele and x_{ij} has the probability that locus j is allele i . Therefore, the means of information Ω_{ave} in a lymphocyte population is obtained as the following equation:

$$\begin{aligned} \Omega_{ave}(N) &= \frac{1}{M} \sum_{j=1}^M \Omega_j(N) \\ &= \frac{1}{M} \sum_{j=1}^M \left\{ \sum_{i=1}^S -x_{ij} \log x_{ij} \right\} \end{aligned} \quad (3)$$

where M is the size of the gene in an antibody.

The affinity $m_{\alpha\beta}$ between antibody α and β is given as follows:

$$m_{\alpha\beta} = \frac{1}{\{1 + \Omega(\alpha\beta)\}} \quad (4)$$

where $\Omega(\alpha\beta)$ is an information which obtained by antibody α and antibody β . If $\Omega(\alpha\beta)=0$, the antibody α and β match completely. Generally $m_{\alpha\beta}$ is given by range of 0-1.

3.6 Interaction Between Antigen and Antibody

The affinity $m_{\varphi\alpha}$ between antigen φ and antibody β is given by

$$m_{\varphi\alpha} = \frac{1}{1 + \delta_{\varphi\alpha}} \quad (5)$$

where $\delta_{\varphi\alpha}$ means the evaluated value to represent the connect strong between antigen φ and antibody α . Therefore, when $\delta_{\varphi\alpha}=0$, the antibody α and antigen φ is to be matched completely.

4. Tuning Of PID Controller By Immune Algorithm

4.1 Selection Mechanism for Control

If an antigen (objective function in this paper) is presented to the B cell object (PID controller), an immune response, that is, the learning is initiated. The level on B cell stimulation depends not only on how well it matches the antigen, but also how it matches other B cell objects in the immune network. The B cell object produces copies of itself, which turn on a mutation mechanism that generates mutations in the genes that code specially for the antibody molecule as shown in Fig. 2. That mirrors the mechanism called somatic hypermutation which occurs in the human immune system. Alternatively, if the

stimulation level falls below a given threshold, the B-cell object will die off and does not replicate. The stimulation of B cell object also depends on its affinity with other B cell objects in the immune system. The network is formed by B cell objects recognizing other B cell objects in the system. Survival of the new B cell objects depends on their affinity to the antigen and to the other B cell objects in the network. The new B-cell objects may have an improved match for the antigen and thus proliferate, and then it can survive longer than existing B cell objects. The immune network reinforces the B cell objects which are useful and have proliferated. By repeating this process of mutation and selection a number of encodes, the immune system learns to produce better matches for the antigen [7-8].

4.2 PID Controller Tuning By Immune Algorithm

The coding of an antibody in an immune network is very important because a well designed coding of antibody can give an increase of the efficiency of the controller. There are three type antibody in this paper: 1) antibody type 1 which is encoded to represent only P gain in the PID controller; 2) antibody type 2 which is encoded to represent I gain; 3) final antibody which is encoded to represent D gain as shown in Fig. 2. The value of the k -th locus of antibody type 1 shows P gain allocated to the route 1. That is, the value of the first locus of the antibody type 1-a means that P gain allocated to the route 1 is obtained by 20.

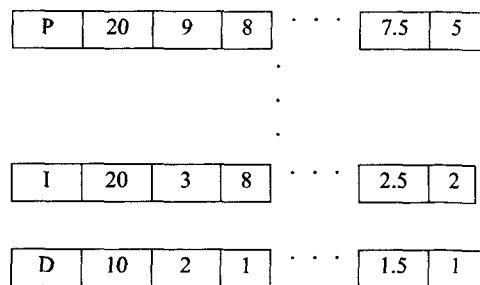


Fig. 2 Allocation structure of P, I, D gain in locus in antibody.

On the other hand, the k -th locus of antibody type 2 represents a I-gain for tuning of the PID controller. Here, the objective function can be written as the followings:

$$\delta_i = \sum_{n=1}^c \left\{ L_n - L_n^{object} \right\}^2 + \zeta_n \quad (6)$$

$$L_n = \sum_{i=1}^p (R_i I_{i,n})$$

$$f_n = \begin{cases} 0: L_n \leq L_n^{lim} \\ 1: Otherwise \end{cases}$$

- f_n : objective function
- z : the number of process for obtaining an optimal PID gain, respectively
- L_n : optimal level in process for selection of an optimal gain
- L_n^{object} : target optimal value in process in process for selection of an optimal gain
- ζ : penalty constant
- f_n : penalty function
- P : the number of route for selection of an optimal gain
- R_i : gain level in route i
- $I_{i,n}$: subsidiary function
- L_n^{lim} : limit speed in PID gain

This algorithm is implemented by the following procedures.

[step 1] *Initionalization and Recognition of antigen*: The immune system recognizes the invasion of an antigen, which corresponds to input data or disturbances in the optimization problem.

[step 2] *Product of antibody from memory cell*: The immune system produces the antibodies which were effective to kill the antigen in the past, from memory cells. This is implemented by recalling a past successful solution.

[step 3] *Calculation of affinity between antibodies*: The affinities $m_{\alpha\beta}$ obtained by equation (4) and the affinity $m_{\alpha\sigma}$ using equation (5) is calculated for searching the optimal solution.

[step 4] *Differentiation of lymphocyte*: The B-lymphocyte cell, the antibody which matched the antigen, is dispersed to the memory cells in order to respond to the next invasion quickly. This dispersed corresponds to strong the solution in a database.

[step 5] *Stimulation and suppression of antibody*: The expected value η_k of the stimulation of the antibody is given by:

$$\eta_k = \frac{m_{\alpha\sigma}}{\sigma_k}, \tag{6}$$

where σ_k is the concentration of the antibodies. The concentration is calculated by affinity based on phenotype

but not genotype because of the reduction of computing time. So, σ_k is represented by:

$$\sigma_k = \frac{\text{sum of antibodies with same affinity as } m_{\alpha\sigma}}{\text{sum of antibodies}} \tag{7}$$

Using by equation (7), a immune system can control the concentration and the variety of antibodies in the lymphocyte population. If antibody obtains a higher affinity against an antigen, the antibody stimulates. However, an excessive higher concentration of an antibody is suppressed. Through this function, an immune system can maintain the diversity of searching directions and a local minimum.

[step 6] *Stimulation of antibody*: To capture to the unknown antigen, new lymphocytes are produced in the bone marrow in place of the antibody eliminated in step 5. This procedure can generate a diversity of antibodies by a genetic reproduction operator such as mutation or crossover. These genetic operators are expected to be more efficient than generation of antibodies.

5. Simulation and Discussions

5.1 Tuning of PID Controller by Immune Algorithms

Fig. 3 shows the proposed control system in this paper and Fig. 4 represents results of PID controller by Ziegler-Nichols ($K_p=18.063$, $T_i=1.4$, $T_d=0.35$) and by

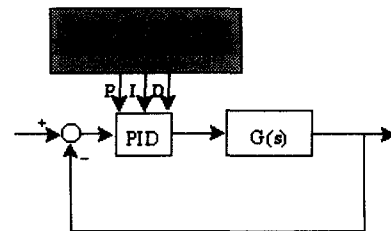


Fig. 3 shows the proposed control system

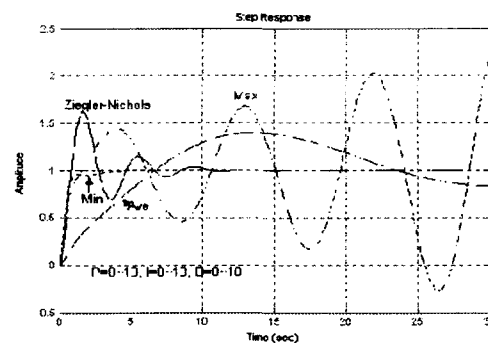


Fig. 4 Response to the range of parameter $P=0-10$, $I=0-10$, $D=0-10$ on parameter learning of immune network.

parameter of PID obtained by immune system, for the given plant EMBED Equation.3. There are some overshoot on response of PID controller tuned by Ziegler-Nichols.

However, Fig. 4 and Table 3 and 4 reveal that the variety of PID parameter can be obtained by immune system.

Table 1. Variation of the PID parameter by the learning value (P=0-10, I=0-10, D=0-10).

Iter	1(iter)	2	3	4	5	6	7	8	9	10(iter)
object value	572.4141	54.2722	100.9655	75.5645	59.4706	25.6183	27.2527	17.1854	10.2840	26.4712
P	1.56518	1.4963	1.4622	0.87322	1.4628	2.1945	2.1944	0.20014	3.7185	0.8375
I	0.11736	0.0958	0.2136	1.9192	2.2096	0.0942	6.0756	0.0954	0.00255	4.5776
D	0.03077	0.0726	0.0944	4.5596	0.7017	0.0178	0.0021	1.63181	3.7316	2.0400

Table 2. Variation of the PID parameter by the learning value (P=0-20, I=0-20, D=0-20).

Iter	1(iter)	2	3(iter)	4	5(iter)	6	7	8	9	10
object value	33.3666	12.00594	10.0435	11.0793	204.2822	15.4695	22.01618	45.12932	24.1704	15.7287
P	2.0909	4.56255	3.54625	4.9978	0.03400	3.20153	6.29459	0.0024	1.00400	0.1543
I	3.4669	13.18455	10.7000	10.5690	0.70133	10.0975	15.00325	12.0777	10.6512	0.66310
D	2.29350	13.17591	0.7654	10.4744	0.00610	14.2950	0.1624	0.76240	14.2421	2.15394

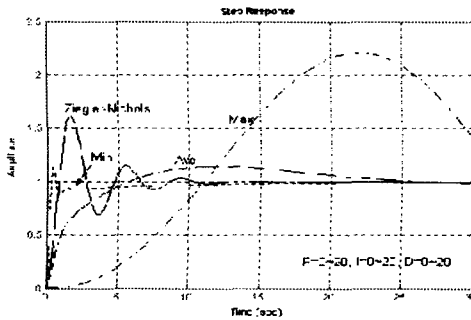


Fig. 5 Response to the range of parameter P=0-20, I=0-20, D=0-20 on parameter learning of immune network.

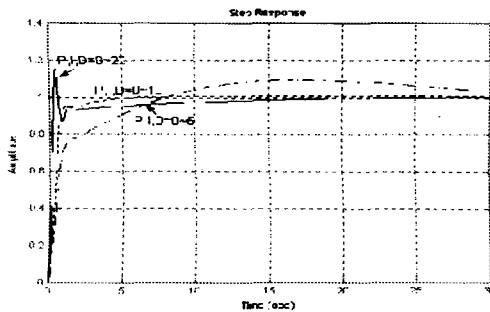


Fig. 6 Response to minimum values on parameter learning of immune network.

Fig. 6 represents comparative response to minimum values of parameters P, I, D after learning when learning range of parameters in immune network is 20 (=P=I=D), 10 (=P=I=D), and 5 (=P=I=D), respectively. Fig. 7 and 8 show that the case of average and maximum.

Fig. 9 is the response of minimum values when P is varied, I and D are fixed as 5. When P=10, the best response is given. Fig. 10 and 11 represent response of minimum and average when I and D are fixed as 10. On the other hand, Fig. 12 and 13 are response of minimum and average when P

is varied I, D are fixed as 20. The response of Fig. 12 has some more undershoot. Fig. 14 shows the response to minimum values when P and D are fixed as 5 and only parameter I varied for learning range on immune network. When I=10, the response has the biggest overshoot.

Fig. 15-16 represent when P, D=10 and only parameter I vary. Fig. 17-19 shows results when parameter P, I are fixed and only parameter D vary. Fig. 17 is in the case of 5 (P=I=5) and Fig. 18 is in the case of 10. (P=I=10) Fig. 19 shows response by the average. Fig. 20-21 shows the response in the fixed P, I (=20) and the varied D. Also, Fig. 22 shows response learned by different value on parameter P, I, D, respectively. Fig. 15-16 represent when P, D=10 and only parameter I vary. Fig. 17-19 shows results when parameter P, I are fixed and only parameter D vary. Fig. 17 is in the case of 5 (P=I=5) and Fig. 18 is in the case of 10. (P=I=10) Fig. 19 shows response by the average. Fig. 20-21 shows the response in the fixed P, I (=20) and the varied D.

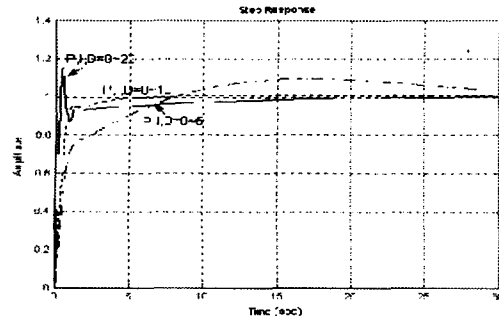


Fig. 7 Response to average values on parameter learning of immune network.

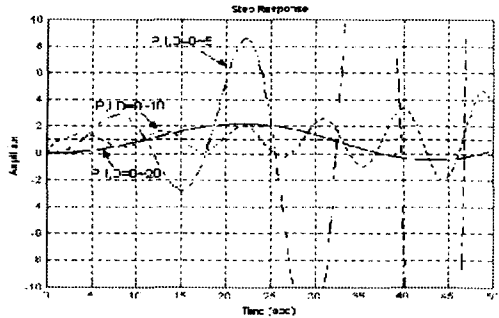


Fig. 8 Response to maximum values on parameter learning of immune network.

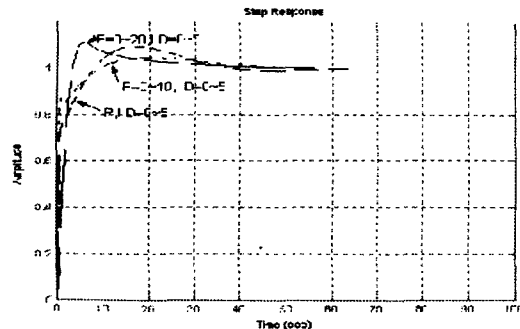


Fig. 9 Response to minimum values on parameter learning of immune network. (P; variation, I, D=5)

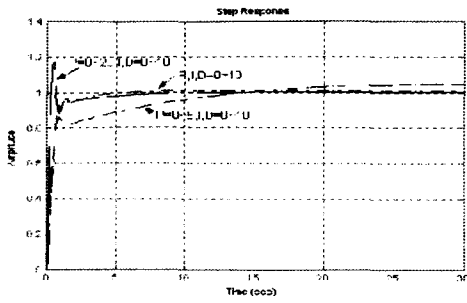


Fig. 10 Response to minimum values on parameter learning of immune network. (P=variation; I, D=10)

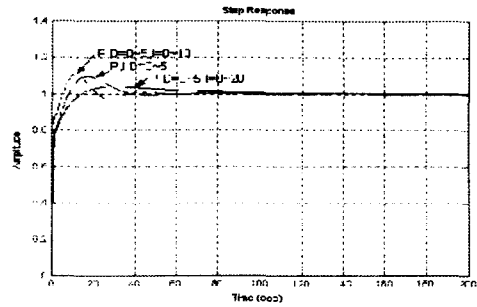


Fig. 14 Response to minimum values on parameter learning of immune network. (I=variation; P, D=5)

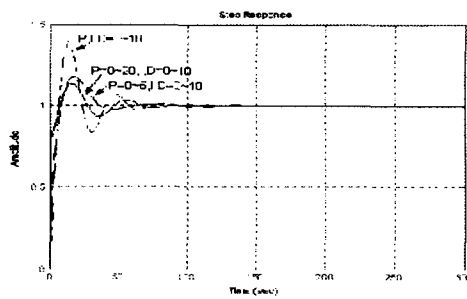


Fig. 11 Response to average values on parameter learning of immune network. (P=variation; I, D=10)

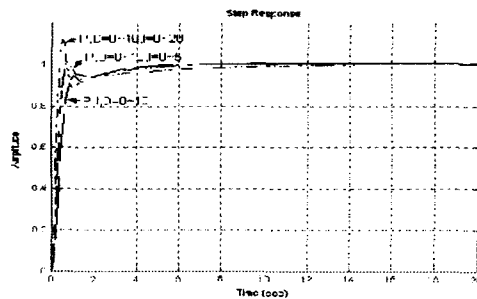


Fig. 15 Response to minimum values on parameter learning of immune network. (I=variation; P, D=10)

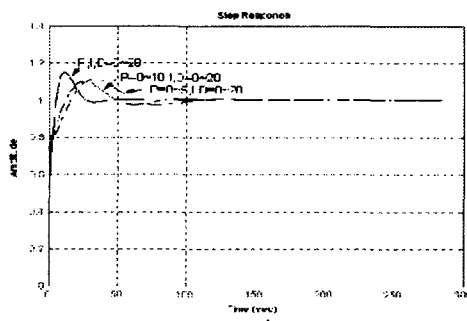


Fig. 12 Response to minimum values on parameter learning of immune network. (P=variation; I, D=20)

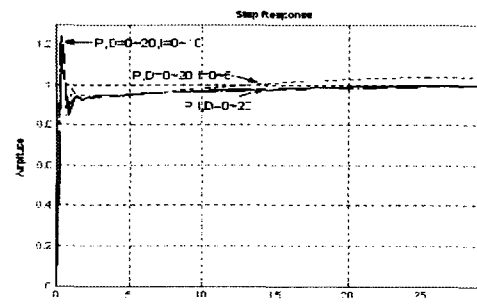


Fig. 16 Response to minimum values on parameter learning of immune network. (I=variation; P, D=20)

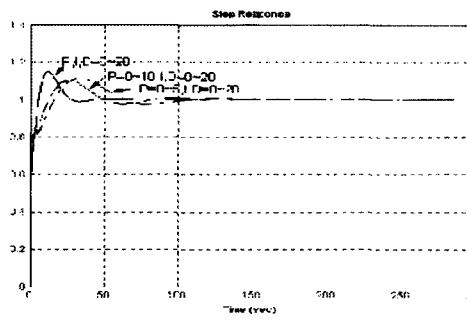


Fig. 13 Response to average values on parameter learning of immune network. (P=variation; I, D=20)

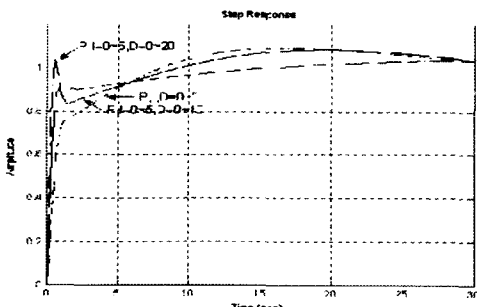


Fig. 17 Response to minimum values on parameter learning of immune network. (D=variation; P, I=5)

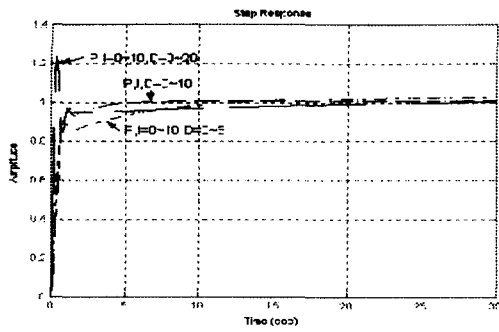


Fig. 18 Response to minimum values on parameter learning of immune network. (D=variation; P, I=10)

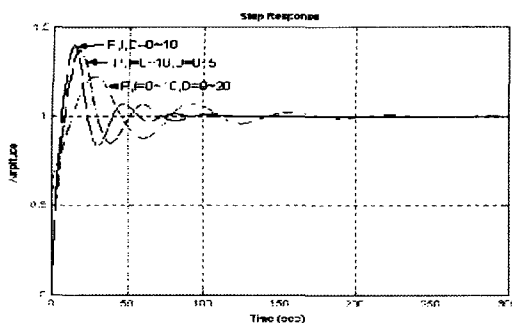


Fig. 19 Response to minimum values on parameter learning of immune network. (D=variation; P, I=0-10)

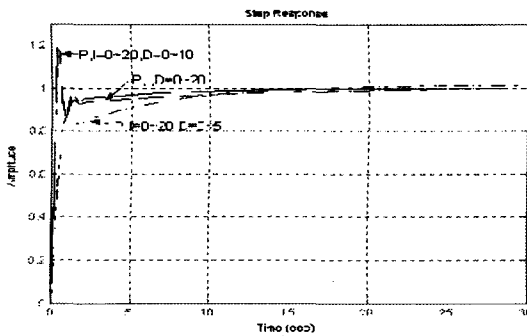


Fig. 20 Response to minimum values on parameter learning of immune network. (D=variation; P, I=20)

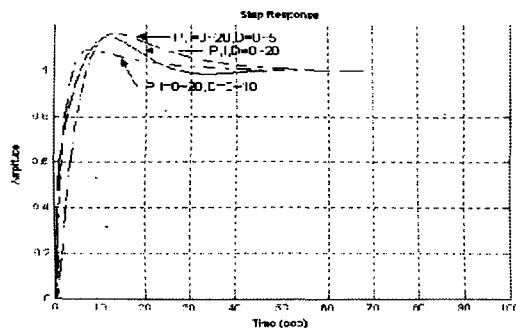


Fig. 21 Response to average values on parameter learning of immune network. (D=variation; P, I=20)

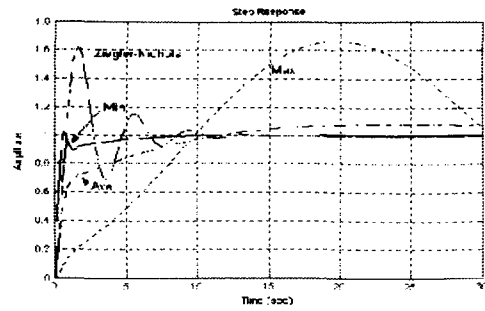


Fig. 22 Response to variation range on parameter learning of immune network. (P=0-20, I=0-10, D=0-5)

5.2 Efficiency of parameter variation

In this study, relationship between initial setting values for learning and P, I, D gain after learning by each cell in immune system is not revealed clearly as shown in Fig. 4-15 and Table 1-2. However, since there are variations in PID parameters to a different initial value of learning, there could be some relationship between both parameters. Therefore, it is necessary to study for this rule in the further study.

6. Conclusions

This paper suggests tuning method of the PID controller by immune network. Parameters P, I, D encoded in antibody randomly are allocated during selection processes to obtain an optimal gain for plant. The object function can be minimized by gain selection for control and the variety gain is obtained as shown in Table 1, 2, and 3. Through this table, an optimal gain required in the plant characteristic can be acquired. All results of simulation represent more satisfactory response than tuning in the Ziegler Nichols method. The parameter obtained by learning of immune system depends on initial range of parameter. Application of this immune system to the control system can be implemented through the result of this study. In the future study, experiments and more detailed discussion between the PID parameter and rule of immune network should be performed.

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This paper was performed by aids of the 2001 research fund of the Hanbat National University. Also, thanks for aids of related thing in University.



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