

# A New Control Method Based on Artificial Immune Adaptive Strategy

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**Abstract**—An Artificial Immune Adaptive Strategy combining immune adaptive control and immune genetic optimization is proposed based on the simulation of the immune mechanisms such as the antibodies' bifunctional structure and the immune cells' regulation principle. This strategy is capable of optimizing online parameters of the immune controller with fixed framework, better simulating the behavior of the biological immune system. The proposed artificial immune adaptive strategy is applied to the control of Continuous Stirred Tank Reactor and its performance is compared with that of Cerebellar Model Articulation Controller. The results validate the effectiveness of artificial immune adaptive strategy.

**Index Terms**—Artificial immune adaptive strategy, cell regulation, bifunctional structure, optimization, intelligent control

## I. INTRODUCTION

It is challenging to control industrial plants due to their complicated nonlinear dynamics. In the past, many nonlinear control methods have been developed, such as adaptive control, robust control and variable structure control etc.. These methods rely heavily on the mathematical models of control plants. The (over)simplifications in deriving such models often lead to system performance degradation. Moreover, heavy demands on mathematical analysis can be too difficult to appreciate by many practitioners [1].

Intelligent control [2], [3] provides an alternative. The immune systems are a kind of complicated biologically motivated information processing system. They operate based on the ability of the immune response to identify the intrusion of antigens (As) and generate antibodies (Ab) to eliminate antigens so as to maintain the dynamically balanced health of human bodies. Therefore the immune system would be expected to provide a new methodology for dynamic problems and many researchers focused on the similarities between the behavior control system and the immune system, and have proposed many Artificial Immune Systems

(AIS's) [4]–[13].

However, till now many researches focus mainly on mimicking the capability on genetics and the optimization of immune system [6]–[13]. Meanwhile, a little of research paper have been publish on the immune control method or algorithm which proposed for the imitation prototype only by the T cell and the B cell special immune mechanism. By mimicking T-cell feedback adjusting mechanism, Takahashi [14] proposed an immune P controller consisting of the activation term (to control the response speed) and the suppression term (to stabilize the organism), but this kind of immune controller cannot compensate for the errors from noise and nonlinear disturbance. Takahashi [15] then proposed an improved immune PID controller. However, their method is not necessarily suitable for complex objects. Ding [16] proposed another adaptive immune controller. The main difference was the function depicting the action between killer T-cell and antigen is fuzzy mapping. The later immune controllers are mostly variants of the previous immune controllers [17]–[19].

The previous immune controller was designed mainly based on the interactions of suppressor T-cell, helper T-cell and killer T-cell with many simplifications and assumptions. These simplifications and assumptions were stopgap at that time. However, its drawbacks become more evident by today's standards.

In fact, the living system can maintain stability because of both genetic optimization and the ability to adapt, i.e., the double peculiarities of the immune action--adjusting control and genetic optimization. This can also be seen from the bifunctional structure of the antibody. Besides the control and elimination of antigen, the immune system has other behaviors such as genetic optimization [20]–[24].

*Analysis 1.* Antibody is essentially a bifunctional molecule with a variable region (V-region) and a constant region (C-region). The variation in C-region is limited. The variation in V-region is significant, by which different antigens can be bound. It is mostly the variation in the V-region that provides the adaptive capability and the fast response for the immune system [5], [21], [25]. Many earlier Immune Optimization Algorithms were designed by simulating V-region, while C-region is overlooked [5], [25]. On the other hand, the immune controller has a fixed form and the characteristics of

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C-region are more exhibited, while the simulation of C-region is insufficient.

*Analysis 2.* There may be innate chaos in the exact time system according to the researches on the creation model of leukocyte [24]. Pool [23] pointed out that the healthy variability is a certain well-heeled type of disorder called chaos. Goldberger concluded that chaos provides the body with the flexibility to respond to various stimuli. What stated above enlighten us that chaos may be an important factor for the quick immune response to eliminate the foreign antigens and keep healthy and normal body.

This paper will focus attention on the antibodies' structure, the immune cells' regulation and chaotic proliferation, as well as both adaptive control and genetic optimization of the immune system. Then a novel Artificial Immune Adaptive Strategy(AIAS) featuring many immune characteristics is proposed and applied to control a complex object -- Continuous Stirred Tank Reactor (CSTR). Along with the better experiment results, this paper thus sheds new light on the control of complex systems.

## II. ARTIFICIAL IMMUNE FEEDBACK OPTIMIZATION CONTROLLER

### A. Adaptation principle of biological immune cells

The immune system protects bodies from the invading substances such as viruses, bacteria and other parasites by immune response and a large number of lymphocytes. There are mainly two kinds of lymphocytes: B and T cells originate in the bone marrow, T-cells mature in the thymus and B cells in the marrow. T-cells act as regulators and effectors [26]–[28]. The regulators regulate the immune response but cannot kill antigens. There are also two types of regulators: helper T-cells that assist the immune response and suppressor T-cells that inhibit the immune response. The effectors eliminate antigens directly and is therefore named killer T-cells—this type of immune response is cellular immunity; after the immune system is stimulated by antigens, a fraction of B-cells undergo terminal differentiation to produce antibodies to react with antigens idiotypically—this is called humoral response, which requires the assistance of helper T-cells.

This mechanism which is called the T-cell regulated immune circuit is key during the immune response [29]. If the concentration of invading substances exceeds some level, Antigen Presenting Cells (APCs) begin to digest the large molecules of certain antigens and transfer the information to the helper T-cells, which then stimulate B-cells, killer T-cells and suppressor T-cells. This is the main feedback mechanism of the immune system. After being stimulated, suppressor T-cells inhibit the activity of all other cells, e.g., helper T-cells, killer T-cells and B-cells, and eventually tranquilize the reaction of immune system—this is another feedback mechanism of the immune system. The cooperation between the feedback mechanisms guarantees the immune system to rapidly respond. In [29] a simple feedback mechanism on T-cell regulated circuit can be obtained.

### B. Immune feedback controller

In this subsection we will give an outline on Immune

Feedback Controller according to [15], [28], [29]. Define the amount of antigens at the  $k$  th generation as  $\varepsilon(k)$ , the output of the helper T-cells stimulated by the antigens as  $T_h(k)$ , and the effect of suppressor T-cells on helper T-cells as  $T_s(k)$ . Thus the total stimulation received by the killer T-cells is

$$S(k) = T_h(k) - T_s(k), \quad (1)$$

where

$$T_h(k) = K_1 \varepsilon(k), \quad (K_1 > 0), \quad (2)$$

$$T_s(k) = K_2 f[\Delta S(k)] \varepsilon(k), \quad (K_2 > 0). \quad (3)$$

In (2) and(3),  $K_1$  is the stimulation factor,  $K_2$  is the suppression factor,  $f(\cdot)$  is the nonlinear function depicting the effect of the reaction of the killer T-cells for the antigens.  $f(\cdot)$  and  $\Delta S(k)$  can be defined as:

$$\Delta S(k) = S(k) - S(k-1), \quad (4)$$

$$f(x) = 1.0 - \exp(-x^2 / a). \quad (5)$$

The parameter  $a$  changes the function shape.

By treating  $\varepsilon(k)$  as the control error  $e(k)$  of a control system, and  $S(k)$  as the control output  $u(k)$ , the immune feedback law can be described as

$$u(k) = K \{1 - \eta f[\Delta u(k)]\} e(k). \quad (6)$$

In (5) parameter  $K = K_1$  controls the response speed, and parameter  $\eta = K_2 / K_1$  controls the stabilization effect. It's obvious that the performance of immune feedback law greatly depends on the selection of  $K$ ,  $\eta$  and the nonlinear function  $f$ . Note that the control algorithm of conventional P controller is

$$u(k) = K_p e(k). \quad (7)$$

We know that the controller (6) based on the immune feedback law is merely a nonlinear P controller comparing (6) with (7), whose proportional gain

$$\overline{K_p} = K \{1 - \eta f[\Delta u(k)]\} \quad (8)$$

is tuned by its own output [14]:  $\Delta u(k)$ .

The P-type immune controller in (8) is not very effective for second- and higher order systems and could not compensate for the control error caused by noise and/or nonlinear disturbances. To overcome this drawback, an improved PID-type immune controller was proposed in  $z$ -transform as follows [15], [16], [18], [19]

$$u_{PID}(k) = K_p \{1 - \eta f[\Delta u(k)]\} \left(1 + \frac{K_I}{z-1} + K_D \frac{z-1}{z}\right) e(k). \quad (9)$$

### C. Artificial immune adaptive strategy

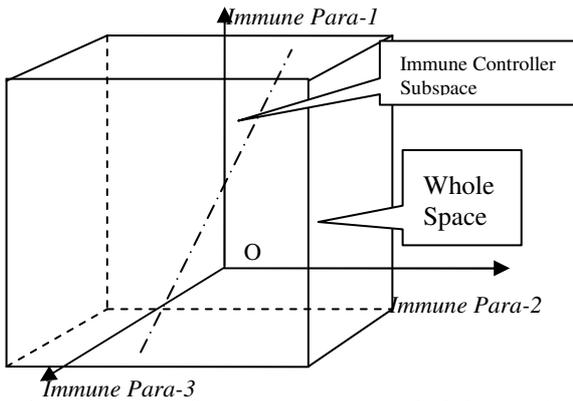


Fig. 1. Immune controller parameters' space and whole space for control parameters.

The improved immune controller in (9) can essentially be treated as a kind of nonlinear PID controller which conducts searching in the whole space for control parameters, as depicted in Fig. 1.

In Fig. 1 each axis represents different Immune Parameters, respectively. From Fig. 1 it can be seen that the previous immune controller makes the whole space for control parameters degenerate to a line or a curve, which may miss the optimal or suitable parameters. This key weakness in fact exists in most immune controllers [15], [16], [18], [19]. Furthermore, how the immune system can maintain the stability of living system in dynamic environments so fast?

As stated in Introduction, chaos maybe an important factor for the quick immune response Therefore we turn to research the logistic equation  $x_{n+1}(t) = \mu x_n(t) (1 - x_n(t))$  ( $n=0, 1, 2, \dots$ ) in genetics. Logistic equation depicts a chaotic sequence describing the changing rule of insects' number over generations, with better randomness than usual RANDOM function..

Furthermore, as is well-known, chaos exhibits ergodicity in the whole space, as shown in Fig. 2. Fig. 2 is an illustration for logistic equation.

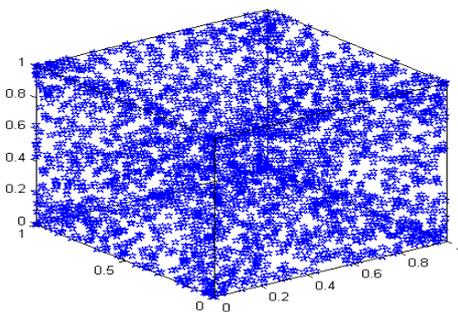


Fig. 2. Ergodicity of chaos.

Utilizing the chaotic proliferation, global search in the whole immune parameters' space can be realized. As a result, suitable parameters can be found, as described in our previous works [5]–[7] on Immune Genetic Algorithm(IGA). IGA is outlined as below.

Considering an optimisation problem  $\max f(X)$ ,  $X \in R^n$ ,  $f(X) > 0$ , perform the steps below:

I. Initialization with  $n:=0$ :

- Specify the population size  $N$ . Generate  $N$  binary strings

of length  $l$  in uniform probability to form the initial population  $X(0) = \{X_1(0), X_2(0), \dots, X_N(0)\}$ ;

- Specify the evolution termination criteria;
- Divide the initial population into  $K$  small sub-populations.

II. Calculate the fitness of each individual in the population  $X(n)$ . If  $X(n)$  meets the given termination criteria, the calculation is stopped and the individual with the best fitness is selected as the solution, otherwise go to step iii with  $n:=n+1$ .

III. The following operations are in the  $i$ -th sub-population:

- Immune Recombination: exchange  $q$  pairs of genes.
- Immune Mutation: toggle each position in a string with a probability  $p_m$ .
- Concentration control and stimulation value control:

$$A_i(n+1) = A_i(n) + \left( \alpha \frac{\sum_{j=1}^N \gamma_{ij} a_j(n)}{N} + \beta g_i - k_i \right) a_i(n), \quad (10)$$

$$a_i(n) = \frac{1}{1 + \exp(0.5 - A_i(n))}. \quad (11)$$

Through the iteration of the above equations, the corresponding antibody's concentration  $a_i(n)$  can be obtained. Then calculate the selection probability  $SGAslct\_pro_i(n)$  in the way of SGA, calculate  $\min(a_i(n), SGAslct\_pro_i(n))$  as the immune selection probability.

- Immune Reproduction: process the copy in the children string according to step (iii.3).

- Immune Metabolism: 5% of the least stimulated individuals are selected to be eliminated; then new individuals with high affinity are created using logistic equation and added.

IV. After calculations to each sub-population, get the maximal fitness value. If maximal fitness value is larger than that provided by existed antibodies in immune network, the corresponding individual is added to the table of memorized antibody, the corresponding maximal fitness value is added to the table describing antibody fitness value. Otherwise, the immune memory mechanism is started up to search better antibody with higher affinity.

V. Terminate check. After the operations above, the offspring  $X(n+1)$  can be got. If  $X(n+1)$  meets the given evolution termination criteria, the calculation is stopped, otherwise go to step iii with  $n:=n+1$ .

During the immune reaction there exist both adaptation of lymph cells and immune optimization. The two aspects coordinate with each other, resulting in fast response and stabilization.

Based on the discussions above, a new Artificial Immune Adaptive Strategy (AIAS) embodying immune control with fixed structure and immune genetic optimization with variable behavior is proposed as below.

In Fig. 3 the output of Immune Feedback Controller is

$$u_{IFOC}(k) = \{K_p \cdot [1 - \eta f[\Delta u(k)]]\} + \frac{K_I}{z-1} + K_D \frac{z-1}{z} e(k), \quad (12),$$

which depicts the lymph cells' adaption.

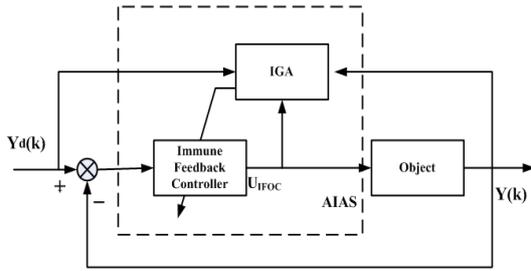


Fig. 3. Artificial immune adaptive strategy.

In Fig. 3,  $\eta$  is immune suppression factor,  $u_{IFOC}$  is the output of immune feedback/optimization controller with a fixed schema,  $K_P$ ,  $K_I$ ,  $K_D$  are immune control parameters which can be viewed as general PID parameters from the pure classic automatic control point of view. The immune parameters i.e.  $\eta$  and PID parameters are determined by immune optimization subsystem-IGA, which stands for the immune genetic optimization, and the immune cells' chaotic proliferation. By immune optimization with IGA, suitable immune parameters can be obtained and transferred to immune feedback controller. To guarantee negative feedback control, set  $0 < \eta f[\Delta u(k)] \leq 1$ .

From (12) it can be seen that AIAS is composed of 2 modules---immune optimization module and immune control module. The former depicts immune genetic optimization and the variation of V-region in antibody, while the latter has a fixed structure, which mimicks the lymph cells' adaption/control and also depicts the stability of antibody. After the steps above we completed the simulation of the biological immune system and its actual behaviors in an artificial immune model.

From (12) it can also be seen that  $K_P$ ,  $K_I$  and  $K_D$  are independent of each other. This is different from the previous immune controllers. Through such independency not only the original immune cell feedback adaption model is inherited but also the immune control parameters can be adapted online asynchronously in the whole space.

Table 1 gives an explicit illustration with comparisons between real immune system and AIAS.

TABLE 1. COMPARISONS BETWEEN BIOLOGICAL IMMUNE SYSTEM AND AIAS.

Biological Immune System	AIAS	
	Immune Control	Immune Optimization
Organism gets hurt or infected by foreign material	Control result is dissatisfactory or the plant(system) is unstable or disturbed	Control result is dissatisfactory or the plant(system) is unstable or disturbed
Foreign antigens attack the organism	The output of the plant(system) does not reach the desired value	The associated fitness function is proposed to be optimized.
Antibodies protect the organism and eliminate the antigens	Calculate $u_{IFOC}$ as the controller's output to control the plant(system).	Give out the corresponding values of $K_P$ , $K_I$ , $K_D$ , $\eta$ related to global optimum.
Organism gets recovered	The error of the system is 0 or realize satisfactory control	In optimization, the termination criteria is met.

### III. CSTR CONTROL ON AIAS

In this section we will control CSTR with AIAS. The diagram is shown as Fig. 4. Acetic acid anhydride hydrolyzation is chosen here. Interactants are controlled by

pumps to flow across CSTR with a fixed speed. The thermal energy released is brought out by looped coolant in pack of CSTR. The model of CSTR [2] is:

$$\frac{dx_1}{dt} = -x_1 u_F + D_a (1 - x_1) \exp\left(\frac{x_2}{1 + x_2 / \gamma}\right), \quad (13)$$

$$\frac{dx_2}{dt} = -x_2 (u_F + \beta) + H D_a (1 - x_1) \exp\left(\frac{x_2}{1 + x_2 / \gamma}\right) + u_c \beta, \quad (14)$$

$$y = x_1, \quad (15)$$

where  $D_a = 3.0$ ,  $\beta = 1.5$ ,  $\gamma = 229$ ,  $H = 2.55$ ,  $u_F = 1.0$ ,  $x_1$  is reaction conversion,  $x_2$  is reaction temperature,  $y$  is output.

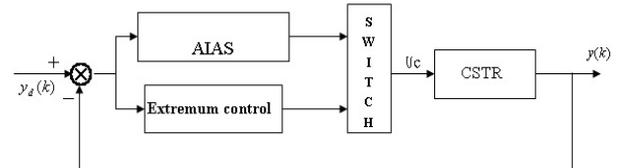


Fig. 4. CSTR control system on AIAS.

The time constant of CSTR is much bigger than sample period, therefore online optimization can be realized [3].

According to Table 1, antigen corresponds to  $e(k) = y_d(k) - y(k)$  in immune control at time point  $k$ , while in IGA, antigen corresponds to the fitness function. The immune parameters  $K_P$ ,  $K_D$ ,  $K_I$ ,  $\eta$  are encoded in one chromosome as the individual  $IM_i(ck)$  in IGA, then optimization proceeds to obtain antibody (control parameters). The parameter  $ck$  denotes immune generation number at each  $k$ .

At time  $k$  the output of CSTR is  $y(k) = f_1(k, IM_i(ck))$ . Taking global consideration of the system performance, ITSE (integral of time weighted squared errors) is introduced to calculate fitness function to suppress overshoot. Thus in AIAS the fitness function should be

$$Fitness(ck) = 1 / \sum_{k=0}^{\infty} k \cdot [y_d(k) - y(k)]^2. \quad (16)$$

Considering the errors in earlier stages have less influence, we design a new criterion named as windowed integral of time weighted squared errors (WITSE), by carrying out windowing transformation with the window size being 3. Thus the revised fitness is written as

$$Fitness(ck) = 1 / \sum_{i=0}^2 \{(k-i) \cdot [y_d(k-i) - y(k-i)]^2\}. \quad (17)$$

Also considering that as the control proceeds the system is stabilized, the historical errors are treated by adaptive dampening, which can be formulated as

$$Fitness(ck) = 1 / (k \cdot [y_d(k) - y(k)]^2 + \frac{1}{k} \sum_{i=1}^2 \{(k-i) \cdot [y_d(k-i) - y(k-i)]^2\} + \varepsilon) = \varphi(k, IM_i(ck)). \quad (18)$$

Eq. (18) shows that as time  $k$  grows larger, the historical errors are reduced. At later stages the major impact on system

performance should be the current error. After the previous modifications, not only can this system inhibit early overshoot, the tracing accuracy in later period is also guaranteed. The item  $\mathcal{E}$  is introduced to prevent the divisor from being 0.

By making (18) as the antigen and optimizing the appropriate immune parameters with IGA, we can execute the control on CSTR.

In Fig. 4 extrema control is used to adjust the system rapidly when error is very large. Expert adjustor is adopted to switch the extrema control and AIAS. The algorithm is

$$u_c = \begin{cases} u_{ex+}, & e \geq e_+, \\ u_{ex-}, & e \leq -e_+, \\ u_{IFOC}, & |e| < e_+, \end{cases} \quad (19)$$

where (19)  $u_c$  denotes the actual control from the expert adjustor,  $u_{ex\pm}$  is the output of extrema controller,  $u_{IFOC}$  is the output of AIAS,  $e_+$  is the upper bound of error which should be empirically determined.

#### IV. EXPERIMENTS AND DISCUSSIONS

In this section simulations are proceeded to test the performance of AIAS by comparisons with other methods.

In AIAS  $K_p, K_D, K_I$  are constrained within the range from 0 to 400,  $N$  is 30 and  $K$  is 5 in IGA. According to [16],  $f(x) = 1 - \exp(-x^2/a)$ ,  $a=0.01$  and  $\eta \in [0,0.8]$  [16]. The initial conditions are  $y_d(0)=0$ ,  $y(0)=0$ ,  $x_1(0)=0$ ,  $x_2(0)=0$ ,  $e_+=0.01$ ,  $u_{ex+}=4.87$ ,  $u_{ex-}=0$ . The size of immune population is 30 and the length of binary individual is 10. Two bits are selected stochastically for mutation. The generation number in IGA is set to 3. The control results using AIAS is shown as Fig. 5. The average time for each control point is  $1985.5s/2000=0.99275s$  with our MATLAB implementation, hence the control can be achieved in real time.

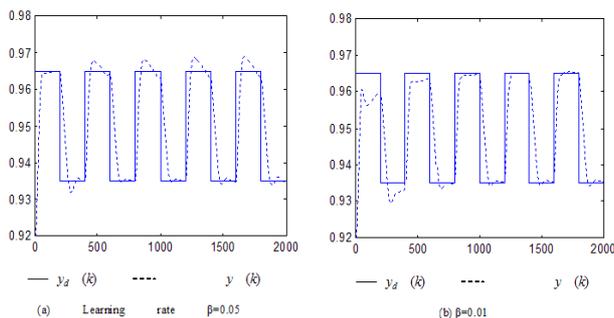


Fig. 5. CSTR tracing results with CMAC [2].

In all of the subsequent figures, x-axis denotes the time. We also show the control results with other methods, to compare with our AIAS.

Fig. 6 shows the control results by regular immune controller using (9) and (5). This controller only simulates the C-region of antibody so the overshoot is large and the adjust time is also longer. Although we adjust parameters many times, the control effect is still inferior to that of AIAS.

Fig. 5 is the results by CMAC inverse control. Although the learning rate is changed, the overshoot is still large and the adjust time is long.

Comparing the figures it is clear that the method in this paper is superior.

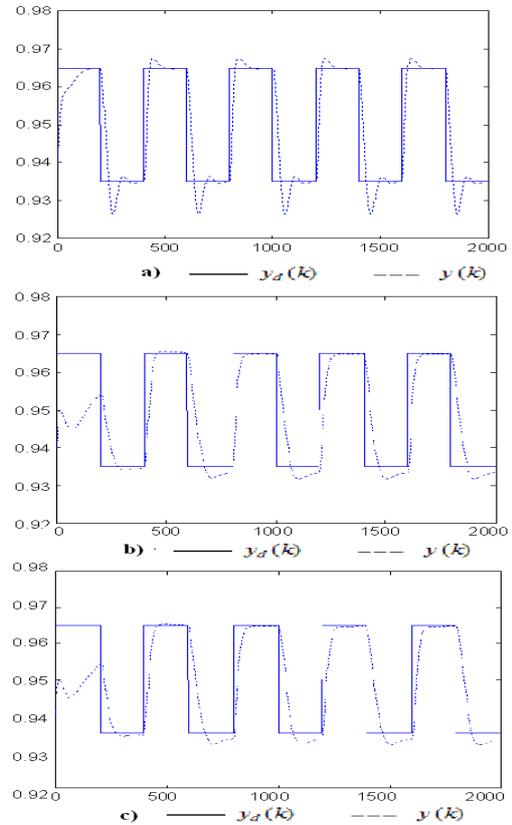


Fig. 6. CSTR control with Immune Controller[16]. (a)  $k_p=200$ ,  $k_i=5$ ,  $k_d=0.5$ ,  $\eta=0.3$ ,  $a=1$ ; (b)  $k_p=120$ ,  $k_i=1$ ,  $k_d=1$ ,  $\eta=0.54$ ,  $a=1$ ; (c)  $k_p=120$ ,  $k_i=1$ ,  $k_d=1$ ,  $\eta=0.8$ ,  $a=1.5$ .

#### V. CONCLUSIONS

Through further study on the immune characteristics such as the bifunctional structure of immune molecular, adaptation and optimization functions, cells' chaotic proliferation etc., a new AIS's---Artificial Immune Adaptive Strategy (AIAS) is proposed with its feasibility analyzed and it is applied to control CSTR, which is a typical complex nonlinear system. As the result of this new control strategy, it will bring about two benefits and progresses as following:

In process control, when error between the desired value and the real output occurs, the controller will start up optimization process to search suitable parameters in the global space quickly to carry out control, which can simulating real immune actions more comprehensively. Therefore the idea that both immune adaptive control and immune genetic optimization can be combined in one structure will shed new light on how to design more effective AIS.

IGA is used in optimization. As proved in our previous works [26], although there are 4 parameters needed to be optimized, IGA can maintain diversity and prevent premature convergence effectively, which leads the appropriate parameters can be obtained quickly so that the system can be controlled better with temperature and humidity tracking

precisions being raised obviously, settling time and overshoot decreasing obviously.

To sum up, the results indicate that the proposed method performs better than its peers and other traditional intelligent control methods such as CMAC and suggest an optimistic prospect in real world applications.

Last but not least, for non-slow-changed complex objects, there are drawbacks of adopting the proposed method. Thus further study on Artificial Immune Systems is necessary if we aim to achieve better results and widen its applications.

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