Parameter Estimation Using an Adaptive Immune Clone Selection Algorithm

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Abstract

A novel Artificial Immune Algorithm, namely Adaptive Immune Clone Selection Algorithm is proposed in this paper for parameter estimation which can be formulated as a multi-modal optimization problem with high dimension. In this method the secondary response, adaptive mutation regulation and vaccination operator are introduced in the generic Clone Selection Algorithm to improve the convergence speed and global optimum searching ability. Simulation results for identifying the parameters of a dynamic system are presented to demonstrate the effectiveness of the proposed method.

1. Introduction

System identification plays a key role in health monitoring, non-destructive evaluation, and active control of civil infrastructures. Because of its wide applicability, considerable efforts have been devoted to develop methods for identification of system models and their parameters.

Currently, a wide range of analytical techniques exists for linear and non-linear systems, such as the recursive least square methods [1]-[3], extended Kalman filter [4], unscented Kalman [5][6] and Monte Carlo filter [7][8]. However, for civil engineering system these methods' applicability and success are limited for complexity and incomplete prior information. Instead, some successes have been achieved with various intelligent optimization algorithms. Evolution strategy algorithms have been presented for the identification of multiple degree of freedom (DOF) systems [9]. Perry *et al*.[10] have presented a modified Genetic Algorithm (GA) to identify structural systems. GAs have been used to solve the global system identification problem in shear-type building structures [11]-[13]. Tang *et al*. [14][15] and Ye and Wang [16] introduced the PSO to the structural systems identification.

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A recently developed computational intelligence technique, inspired by biology, has emerged: the Artificial Immune Algorithm (IA) [17], which can be used for solving computational problems. Although still relatively young, the IA is emerging as an active and attractive field involving models, techniques and applications of greater diversity [18]. Over the last years, there has been increasing interest in the area of IA and their applications for solving complex optimization problems, such as IIR Filter design [19], truss structure optimal design [20], anti-spam filter design [21]. Compared with GA, IA has affinity calculation function, which could explain the relationship not only between the antigen and the antibody but also between antibodies. That makes IA has the unique characteristic to guarantee the survival of the variant offspring that could match the antigen better. Related papers [19] show that the algorithms based on IA have much better performance than conventional probabilistic optimization algorithms.

Nevertheless, when solving complex multi-model problems the simple IAs are also hard to get out of the local optimum and the convergence speed is slow. Besides, the simple IAs just simulate a part of the immune system's mechanism, such as the memory, oblivion, and self-adaptive mechanisms are still need to further explore. For improving the IAs' effectiveness to solve complex problems, in this paper by introducing the secondary response, adaptive mutation regulation and vaccination operator these three strategies into the generic Clone Selection Algorithm (CSA) [22], we proposed a novel Artificial Immune Algorithm, namely Adaptive Immune Clone Selection Algorithm (AICSA). Some numerical examples are presented from which the effectiveness and efficiency of the AICSA are investigated.

2. Problem Formulation

The basic idea in system identification is to compare the time dependent response of the system and a parameterized model by a norm or some performance criterion giving a measure to how well the model response fits the system response. Hence, the objective is to find a set of parameters that minimize the prediction error between system output $y(t)$, i.e., the measured data, and model output $\hat{y}(\theta, t)$ at each time-step *t* .

Therefore, our interest lies in minimization the predefined error norm of the outputs, e.g., the following mean square error function.

$$
f(\theta) = \frac{1}{T} \sum_{t=1}^{T} ||y(t) - \hat{y}(t)||^2
$$
 (1)

where $\left\| \cdot \right\|$ represents the Euclidean norm of vectors. Formally, the optimization problem requires finding a vector $\theta^* \in R^n$, so that a certain quality criterion is satisfied, namely that the error norm $f(*)$ is minimized. The function $f(*)$ is commonly called a fitness function or objective function. In IA, typically an objective function is used which reflects the goodness of solution. The identification problem thus is treated as a linearly constrained multi-dimensional optimization problem, namely

minimize
$$
f(\theta)
$$
, $\theta = (\theta_1, \theta_2, \dots, \theta_n)^T$

$$
st.\theta \in S, S = \left\{\theta : \theta_{\text{mix}} \leq \theta_i \leq \theta_{\text{mix}}, \forall i = 1, 2, \dots, n\right\} \qquad (2)
$$

where $f(\theta)$ = objective function which maps decision variable θ into objective space $f = R^n \rightarrow R$, S is the n-dimensional feasible search space, θ_{max} and θ_{min} denote the upper bounds and the lower bounds of the n parameters respectively.

3. Algorithm

3.1. Improved Clone Selection Operator

Just as the Evolutionary Algorithms (EAs)[23], the Artificial Immune Algorithms work on the encoding of the parameter set rather than the parameter set itself (except where the real-valued individuals are used). For an optimization task we consider minimizing the objective function $f(\theta), \theta = (\theta_1, \theta_2, \dots, \theta_n)^T$. For the binary code antibody $A \in H^1$, $H^1 = \{0,1\}^1$ denotes all binary cluster set with same length *l* . $\overline{A} = \{A_1, A_2, \dots, A_n\}$ is the antibody population, and $A_i = \{a_{i1}, a_{i2}, \dots, a_{i} \}$. The binary cluster is divided into n segments with the

length l_i , $l = \sum_{i=1}^{n}$ 1 $l = \sum l_i$, where each segment is expressed as *i* =

 $\theta_i \in [\theta_{\text{max}_i}, \theta_{\text{min}_i}]$ $i = 1, 2, \dots, n$ respectively.

Set the estimation parameters as antigen. The antibody-antigen affinity function is chosen as $f(\theta)^{-1}$. The antibody-antibody affinity function is defined as the following equation:

$$
D_{ij} = \|X_i - X_j\| \quad i, j = 1, 2, \cdots, m \tag{3}
$$

where $\|\cdot\|$ is an arbitrary norm, generally taking Euclidean Distance for real-valued coding and Hamming Distance for binary coding. $D = (D_{ij})_{m \times m}$ *i*, $j = 1, 2, \cdots, m$ is the affinity matrix of antibody-antibody. *D* is a symmetrical matrix, which indicates the diversity of the antibody population.

A vaccination operator is introduced in the generic clone operator [22]. It simulates vaccine injection and adaptively extracts antigen's pre-knowledge from the antibody population. It can inhibit the antibody generation's retrogression and enhance the propagation speed of good gene in the antibody population.

The improved clone operator can be described as follows:

Clone Θ : The clone operator Θ is defined as:

$$
\Theta(\overline{A}) = [\Theta(A_1), \Theta(A_2), \cdots, \Theta(A_n)]^T
$$
 (4)

where $\Theta(A_i) = I_i \times A_i$, $i = 1, 2 \cdots, m$, and I_i is q_i dimension row vectors.

$$
q_i = \text{Int}\left[M_c \cdot \frac{aff(A_i)}{\sum_{j=1}^{m} aff(A_j)} \cdot \Omega_i\right] i = 1, 2, \cdots, m \quad (5)
$$

where $M_c > m$ is the expected clone scale, and Int(\cdot) is the integral function up. Besides, Ω is given by:

$$
\Omega_i = \min\{\exp(D_{ij})\} \quad i \neq j; \quad i, j = 1, 2, \cdots, m \tag{6}
$$

After cloning the antibody population is like this:

$$
\overline{B} = \left\{ \overline{B}_{1}, \overline{B}_{2}, \cdots, \overline{B}_{m} \right\} \tag{7}
$$

where:

$$
\overline{B}_i = \left\{ A_{i1}, A_{i2}, \cdots, A_{im} \right\} \quad A_{ij} = A_i \quad j = 1, 2, \cdots, q_i \quad (8)
$$

Clone Mutation T_m^C :

$$
p(A_{ij} \to A'_{ij}) = p_m^{\mathrm{d}(A_{ij}, A'_{ij})} (1 - p_m)^{1 - \mathrm{d}(A_{ij}, A'_{ij})}
$$
(9)

Where, $d()$ is the Hamming Distance, p_m is the mutation possibility.

Vaccination Operator T_v^c : Randomly select a segment of the best antibody's gene as a vaccine. Then randomly select *m* antibodies and embed the vaccine into their genes, so they will have the same gene as the best antibody at this segment. Perform this operator when the global antibody-antigen affinity improves every *v* times.

Clone Selection T_s^c :

 $\forall i = 1, 2, \cdots, m \text{ if } b = \max\left\{aff(A'_i), j = 1, 2, \cdots, q_i\right\}$

and $aff(A_i) < b$ then A'_i replaces the antibody A_i in the aboriginal population.

3.2. Adaptive Immune Clone Selection Algorithm

 Although compared to GAs, CSA represents many advantages, however it is still difficult to solve complex problems. For satisfying the requirement of solving complex problems, in AICSA we design three strategies which are secondary response, adaptive mutation regulation and vaccination operator to improve the generic CSA's convergence speed and global optimum searching ability.

The AICSA is summarized as follows:

Step1: Initiate the antibody population $\overline{A}(0)$, enact algorithm parameters.

Step2: Calculate the antibody-antigen affinity $aff(A(k))$.

Step3: Record each *K* generations' best searching results, and compute these *K* results' deviation to the *K*-th result, the equation is like this:

$$
std_i = \sqrt{\frac{1}{K} \sum_{j=1}^{K} (\hat{\theta}_{ij} - \hat{\theta}_{ik})^2} \quad i = 1, 2, \cdots, n \quad (10)
$$

where $\hat{\theta}_y$ is the *i*-th parameter's best searching result of

the *j*-th generation in these *K* generations. The algorithm only "remembers" the last *K* generations' information while "forgets" the former generations' information, since the last generations' information already contains the former generations' information theoretically. Generally *K* should be selected such that it is sufficient to get a good estimation of the searching direction but not so large that it includes very old results that will slow the convergence. In this paper we choose *K* to contain *h* iterations in which the global antibody-antigen affinity improves.

Step4: Regulate the mutation probability: according to the following equation, the corresponding mutation probability of each antibody can be calculated.

$$
t = \exp\left(\max(l_i) \times \frac{aff(A_i(k))}{\sum_{j=1}^m aff(A_j(k))}\right) \tag{11}
$$

$$
p_m^i(k) = \frac{1}{l} + [c + k' + t]^{-1} \quad i = 1, 2, \cdots, m \qquad (12)
$$

where k' is the number of iterations in which the global optimum antibody-antigen affinity improves or improves a certain percentage, and $c \geq 0$ is a constant to control the beginning mutation probability, generally $c = 1$. The item 1 $(A_{i}(k))$ $(A_{.}(k))$ *i m* $\sum_{j=1}^{\infty}$ α_{jj} $\left(\frac{1}{j}\right)$ *aff A k aff A k* $\sum_{j=1}$ is used to

ensure that the antibody who has higher antibodyantigen affinity will have smaller mutation probability, and *k*′ is used to make the mutation probability become smaller and smaller along the searching process but isn't change when the searching process get trapped in a local optimum therefore keep the algorithm's ability to get out of the region, and $\frac{1}{1}$ *l* is used to ensure at least there will be one gene to mutate in the latter searching process. **Step5:** Adapt the antibody population.

If there are $D_{ii}(k) \leq c_i$, $i, j = 1, 2, \dots, n, i \neq j$, where *c*, is the threshed of the antibody-antibody affinity and generally $0 \leq c_1 \leq \frac{1}{l}$ *l* $\leq c_{1} \leq -1$, then randomly select two different antibodies to perform one-point crossover according to a probability p_c set before, and use the generated new antibodies to replace A_i or A_j equally, but don't change the antibody-antigen affinity .

Step6: According to the affinity and the clone scale, perform the improved clone operator and get the new antibody population $\overline{C}(k)$;

Step7: Secondary immune response

Kill the last *R* bad antibodies whose antibodyantigen affinities are smaller than the others, and regenerate *R* new antibodies randomly in a reduced search space according to the former information extracted from the searching process, if $k > K$, then get the next generation $\overline{A}(k+1)$. The reduced searching space is defined as:

$$
\begin{cases}\n\theta'_{\text{max }i} = \hat{\theta}_{ik} + std_i \\
\theta'_{\text{min }i} = \hat{\theta}_{ik} - std_i\n\end{cases}
$$
\n(13)

and

$$
\begin{cases} \theta'_{\max i} = \theta_{\max i} & if \quad \theta'_{\max i} > \theta_{\max i} \\ \theta'_{\min i} = \theta_{\min i} & if \quad \theta'_{\min i} < \theta_{\min i} \end{cases} \quad i = 1, 2, \cdots, n \quad (14)
$$

where θ_{max} and θ_{min} is the up and low bound of the original search space, and θ'_{max} and θ'_{min} is the up and low bound of the new reduced search space, but note that the whole generation's search space is not change. **Step8:** $k = k+1$; if satisfied the halt condition, end, or else return to step2.

By using clone selection, the algorithm can integrate the global searching and local searching, and by using the vaccination operator AICSA enhances the propagation speed of good genes. In AICSA the mutation probability is regulated adaptively according to antibody-antigen affinity and along with the searching process to make sure that the good antibody's mutation probability is smaller and the bad antibody's mutation probability is bigger, and the mutation probability is bigger in the former searching phase for a large-scale search and smaller in the latter searching phase for a fine search. By simulating the memory and oblivion mechanism of immune system, AICSA uses the secondary response strategy to extract knowledge from the antigen generations through the last searching process, and using this knowledge to direct the new antibodies' generation for enhancing the convergence speed. Therefore AICSA is a selfadaptive learning system which can accumulate knowledge from the searching process and regulate its population adaptively, and furthermore it is easy to stable.

4. Simulation Results

In order to assess the effectiveness of the parameter estimation technique with the AICSA presented above, numerical simulations of a five DOF dynamic system is carried out. The properties of the dynamic system are given in Table 1. It is assumed that the system is excited by known forces and that the response of the system, in terms of accelerations, is recorded at all points. Table 1**. System parameters**

The dynamic equation of motion of a dynamic system can be written as

$$
M\ddot{x}(t) + C\dot{x} + Kx(t) = u(t)
$$
 (15)

where M, C and K are the mass (m_1, \dots, m_5) , damping (c_1, \dots, c_s) and stiffness (k_1, \dots, k_s) matrices, x is the displacement vector and u is the input force vector. The damping matrix C is given by:

$$
C = \alpha M + \beta K, \quad \xi_r = \frac{\alpha}{2\omega_r} + \frac{\beta \omega_r}{2} \tag{16}
$$

In simulation test it is assume that $\alpha = 0.7510$ and β = 0.0026 are known.

Therefore, the system is fully described by the set of parameters

$$
\theta = (m_{1}, \cdots, m_{5}, k_{1}, \cdots, k_{5})
$$
 (17)

In simulation test, parameters are set as follows:

For AICSA and CSA, the searching range is half to twice of the parameters' true value, and the code length of each parameter is 25, therefore the resolution of each estimated parameter is $\frac{1}{2^{25} - 1} \times 1.5 = 4.47 \times 10^{-8}$ $\frac{1}{1}$ × 1.5 = 4.47 × 10 $2^{25} - 1$ $- x 1.5 = 4.47 \times 10^{-8}$.

The number of population $m = 60$, the maximum evolutionary generation is 500, and the expected clone scale $M_c = 5m$. Otherwise:

• AICSA: $c_1 = 0$, $p_c = 1$, $R = 20$, $m_v = 0.7m$, $v=5$, and for K , $h=15$.

• CSA:
$$
p_m = 0.07
$$
.

The statistical simulation results of 20 independent runs for the example with the usage of the AICSA and generic CSA methods are shown in Tables 2-3. In addition, we present typical simulation results (including the convergent processes of objective value and all parameters) for the example with Figs 1-5.

Table 2. **Estimated results of AICSA**

Par.	True value	Estimation Results		Estimation Errors	
		Mean	Var.	Mean	Var.
k1	5e6	5.000e6	1.783	3.058e-7	2.302e-7
k2	4e ₆	4.000e6	0.952	2.157e-7	1.841e-7
$k3-k5$	3.5e6	3.500e6	0.619	1.613e-7	1.268e-7
$m1-m2$	4e ₃	4.000e3	7.519e-4	1.688e-7	1.124e-7
$m3-m5$	3e ₃	3.000e3	4.305e-4	1.448e-7	1.223e-7
		Note: Par.=parameter, Var.=variance			

Table 3. **Estimated results of CSA**

Note: Par.=parameter, Var.=variance

Fig 1. **One typical simulation result of m (kg) by AICSA**

Fig 3. **One typical simulation result of m (kg) by CSA**

Fig 5. **One typical convergence estimation characteristics**

From Tables 2-3, it can be seen that the results obtained by AICSA are very close to the true values.

The average and variance results obtained by AICSA greatly outperform those obtained by the CSA. The AICSA seems to be more powerful in escaping local optimum and in searching for the global optimum.

From Figs 1-4, we observe that CSA seemed to have more difficulty locating the solution than AICSA. It can be seen in Fig 5 that the objective function value reached in AICSA is very low, whereas in CSA a somewhat higher value has been reached (further away from the global optimum). This implies that the AICSA is more effective. And in the latter searching phase CSA improves the estimation results hardly, it needs more time to get out of the local optimum, but AICSA can get out of the local optimum easily and improves the results almost through the whole searching process until close to the resolution according to the length of the binary code. This property is due to AICSA reduces the mutation probability in the latter searching phase to make a fine searching while keeps the bad antibodies' mutation probability bigger to help get out of the local optimum. However, CSA keeps the same mutation through the whole searching process and depend on the high frequent mutation to aimlessly search better antibodies, hence it will waste much time. Instead, AICSA regenerates antibodies whose affinities are low in a reduced space closing to the estimation objective, and because these new antibodies are generated randomly in the reduced searching space so their distribution is even, hence the diversity of the antibody population doesn't decrease in fact. Therefore this strategy will greatly increase the chance to find new better antibodies while avoid the premature of the antibody population. Besides, AICSA also utilizes the vaccination operator to enhance good genes' propagation speed. Because of these, the AICSA performs significantly better than original CSA on estimation dynamic system's parameters in this paper.

5. Conclusion

This paper presents an AICSA strategy for parameter estimation of dynamic system. This novel strategy ensures that in the former searching phase the algorithm can search a relative large-scale space while undertakes a fine search in the latter searching phase through adaptively regulating mutation probability, therefore enhance the algorithm's ability to locate the global optimum. It also greatly improves the searching effectiveness through using the vaccination operator which simulates vaccine injection and the secondary response strategy which simulates the memory and oblivion mechanism of immune system. Consequently, AICSA can direct the searching process using the

former extracted information, thereby avoids an aimless and ineffective search such as CSA. Comparative studies have been investigated to assess the applicability of the AICSA for parameter estimation of dynamic system. From the analysis results, we observe that the AICSA significantly improves the estimation results compared to CSA .

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