Emergent Construction of Artificial Immune Networks for Autonomous Mobile Robots

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Abstract

In the behavior-based artificial intelligence(AI) approach, there are the following problems that have to be resolved: how do we construct an appropriate arbitration mechanism, and how do we prepare appropriate competence modules. Therefore, we have been investigating a new behavior arbitration mechanism based on the biological immune system. However, we have not show a solution to the latter problem. In this paper, we try to incorporate the metadynamics function and selection mechanism into our previously proposed method in order to construct an appropriate immune network without human intervention.

1 Introduction

In recent years much attention has been focused on *behavior-based AI*, which has already demonstrated its robustness and flexibility against dynamically changing world. In this approach, intelligence is expected to result from both mutual interactions among *competence modules* (*i.e.* simple behavior/action) and interaction between the robot and environment. However, there are still open questions: 1) how do we construct a mechanism that realizes appropriate arbitration among multiple competence modules, and 2) how do we prepare appropriate competence modules.

Brooks has showed a solution to the former problem with the use of *subsumption architecture* [1, 2]. Although this method demonstrates highly robustness, it should be noted that this architecture arbitrates the prepared competence modules *on a fixed priority basis*. It would be quite natural to vary the priorities of the prepared competence modules according to the external and/or internal situation. *Maes* proposed an another flexible mechanism called *behavior network system* [3, 4]. In this method, agents (*i.e.* competence modules) form the network using cause-effect relationship, and an agent suitable for the current situation and the given goals emerges as the result of activation propagation among agents. This method, however, is difficult to apply to a problem where it is hard to find the cause-effect relationship among agents.

One of the promising approaches to tackle the above mentioned problems is a biologically-inspired approach. Among biological systems, we particularly focus on the immune system, since it has various interesting features such as *immunological memory, immunological tolerance, pattern recognition*, and so on viewed from the engineering standpoint. Recent studies on immunology have clarified that the immune system does not just detect and eliminate the non-self materials called *antigen* such as virus, cancer cells and so on, rather plays important roles to maintain its own system against dynamically changing environments through the interaction among *lymphocytes and/or antibodies*. Therefore, the immune system would be expected to provide a new methodology suitable for dynamic problems dealing with unknown/hostile environments rather than static problems.

Based on the above facts, We have proposed a new decentralized consensus-making system inspired by the biological immune system in [5, 6, 7]. We expect that there would be an interesting AI technique suitable for dynamically changing environments by imitating the immune system in living organisms. However, what competence modules (antibodies) should be prepared still remains an open question. In this paper, we try to incorporate adaptation mechanisms based on the innovation mechanisms into the previously proposed artificial immune network in order to autonomously construct appropriate immune networks. We verify our method by carrying out simulations.

2 Biological Immune System

2.1 Overview

The basic components of the biological immune system are *macrophages*, *antibodies* and *lymphocytes* that are mainly classified into two types, namely *B-lymphocytes* and *T-lymphocytes*. B-lymphocytes are the cells maturing in *bone marrow*. Roughly 10^7 distinct types of B-lymphocytes are contained in a human body, each of which has distinct molecular structure and produces "Y" shaped antibodies from its surfaces. The antibody recognizes specific *antigens*, which are the foreign substances that invade living creature, such

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as virus, cancer cells and so on. This reaction is often likened to *a key and keyhole relationship* (see Fig.??). To cope with continuously changing environment, living systems possess enormous repertoire of antibodies in advance. On the other hand, T-lymphocytes are the cells maturing in *thymus*, and they generally perform to kill infected cells and regulate the production of antibodies from B-lymphocytes as outside circuits of B-lymphocyte network (idiotypic network) discussed later.

For the sake of convenience in the following explanation, we introduce several terms from immunology. The key portion on the antigen recognized by the antibody is called an *epitope* (antigen determinant), and the keyhole portion on the corresponding antibody that recognizes the antigen determinant is called a *paratope*. Recent studies in immunology have clarified that each type of antibody also has its specific antigen determinant called an *idiotope* (see Fig.??).

2.2 Jerne's idiotypic network hypothesis

Based on this fact, *Jerne* proposed a remarkable hypothesis which he has called the *"idiotypic network hypothesis"*, sometimes called *"immune network hypothesis"* [8, 9, 10, 11, 12]. This network hypothesis is the concept that antibodies/lymphocytes are not just isolated, namely they are communicating to each other among different species of antibodies/lymphocytes. This idea of *Jerne's* is schematically shown in Fig.1. The idiotope **Id1** of antibody 1 (**Ab1**) stimulates the B-lymphocyte 2, which attaches the antibody 2 (**Ab2**) to its surface, through the paratope **P2**. Viewed from the standpoint of **Ab2**, the idiotope **Id1** of **Ab1** works simultaneously as an antigen. As a result, the B-lymphocytes 1 with **Ab1** are suppressed by **Ab2**. On the other hand, antibody 3 (**Ab3**) stimulates **Ab1** since the idiotope **Id3** of **Ab3** works as an antigen in view of **Ab1**. In this way, 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 a new parallel distributed processing.

2.3 Metadynamics and selection mechanism

In the biological immune system, the structure of the network is not fixed, but variable continuously. It flexibly self-organizes according to dynamic changes of environment. This remarkable function, called metadynamics function[13, 14, 15], is mainly realized by incorporating newly-generated cells/antibodies and/or removing useless ones. Fig.2 schematically shows the metadynamics function. The new cells are generated by both gene recombination in bone marrow and mutation in the proliferation process of activated cells (the mutant is called *quasi-species*). 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. Due to such enormous loss, the metadynamics function works to maintain appropriate repertoire of cells so that the system could cope with environmental changes. The metadynamics function would be expected to provide feasible ideas to engineering field as emergent system.

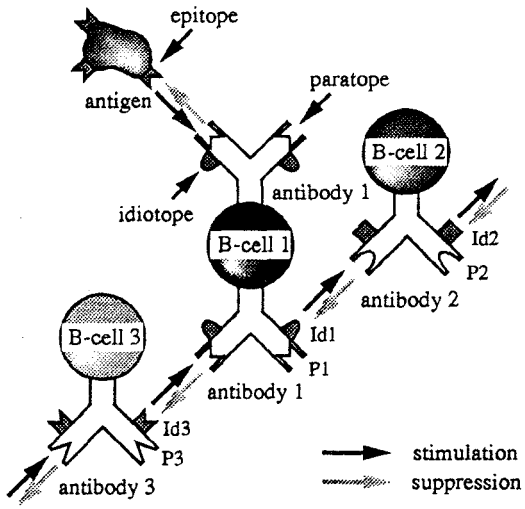


Figure 1: Jerne's idiotypic network hypothesis.

Furthermore, new types of T-cell, which are also generated by gene recombination, undergo the selection in the thymus before they are incorporated into the body. In the selection mechanism, over 95 percent of them would be eliminated (*apoptosis*). The eliminated T-cells would strongly respond to self or not respond to self at all. In other word, the selection mechanism accelerate the system to incorporate new types effectively.

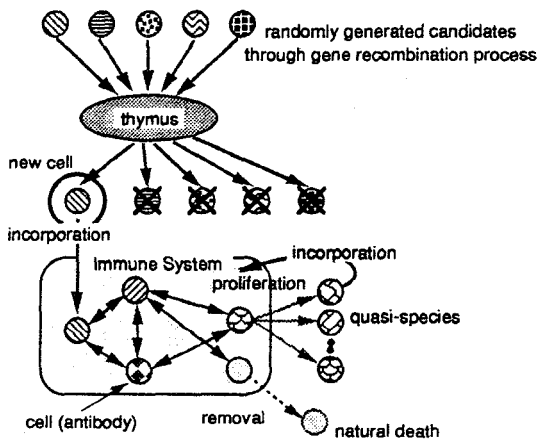


Figure 2: Metadyamics function and selection mechanism.

3 Decentralized Consensus-Making Network Based on the Immune System

3.1 Action selection problem and the immune system

As described earlier, in the behavior-based AI, how to construct a mechanism that realizes appropriate arbitration among the prepared competence modules must be solved. We have approached to this problem from the immunological standpoint, more concretely with the use of immune network architecture [5, 6, 7]. In this section, we discuss our proposed decentralized consensus- making network based on the biological immune system. Fig.3 schematically shows the action selection system for an autonomous mobile robot and the immune network architecture. As shown in this figure, current situations detected by installed sensors work as multiple antigens, and a prepared competence module (*i.e.* simple behavior) can be regarded as an antibody (or B-lymphocyte), while the interaction between modules is represented by stimulation and suppression between antibodies. The basic concept of our method is that the immune system equipped with the autonomous mobile robot selects a competence module (antibody) suitable for the detected current situation (antigens) in a bottom-up manner. For convenience, we have dubbed the autonomous mobile robot with the immune network-based action selection mechanism *"immunoid"*.

3.2 Description of the antigens and antibodies

Antigen informs the situation of the detected object. For example, in Fig.3 (a),the antigens inform the distance and the direction to the obstacle and the destination. In this study, the epitope of each antigen is represented in a binary string form according to the sensor readings.

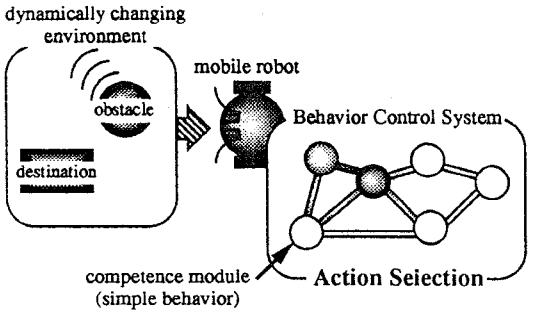
Next, we explain how we describe an antibody in detail. To make immunoid select a suitable antibody against the current antigens through interacting among antibodies, we must look carefully into the description of the antibodies. To realize the above requirements, we defined the description of antibodies as follows: We define each antibody has one action to be executed when it is selected. And we assign a precondition to the paratope, and a disallowed condition to the idiotope, respectively. The structure of the paratope and the idiotope is the same as the epitope of the antigen (*i.e.* binary string form).

In addition, in order to represent the appropriateness of each antibody, we introduce one state variable called *concentration of antibody*.

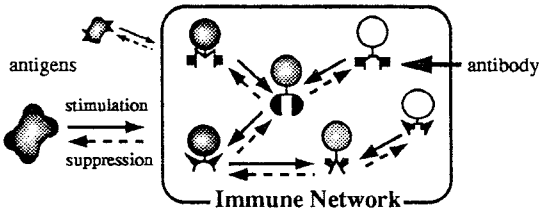
3.3 Interaction between antibodies

We explain the interaction among antibodies, that is, the basic principle of our immunological consensus-making networks in detail. For the ease of understanding, we use the example depicted in Fig.4.

Consider the listed two antibodies that respond to the antigens with epitopes C_1 and C_2, respectively. These antigens stimulate the antibodies, consequently



(a) Action selection system for an autonomous mobile robot.



(b) Immune network architecture.

Figure 3: Basic concept of our proposed method.

the concentration of antibody 1 and 2 increase. If there is no interaction between antibody 1 and antibody 2, these antibodies will have the same concentrations. Suppose that the idiotope of antibody 1 and the paratope of antibody 2 are the same. This means that antibody 2 is stimulated by antibody 1, and oppositely antibody 1 is suppressed by antibody 2 (indicated the arrow). In this case, unlike the previous case, antibody 2 will have higher concentration than antibody 1. As a result, antibody 2 is more likely to be selected. This means that antibody 2 has higher priority over antibody 1 in this situation. As observed in this example, the interactions among the antibodies work as a priority adjustment mechanism.

3.4 Dynamics

We discuss the dynamics of the system, namely, the changes of the concentration of antibody. First, we define the affinity m_{ji} (*i.e.* the degree of interaction) between antibody j and antibody i and the affinity m_i between the detected antigens and antibody i as:

$$m_{ji} = F\left(\sum_{k=1}^{L}\left(w_k\overline{I_j(k)\oplus P_i(k)}\right)\right), \qquad (1)$$

$$m_i = \sum_j\left\{F\left(\sum_{k=1}^{L}\left(w_k\overline{E_j(k)\oplus P_i(k)}\right)\right)\right\}, \qquad (2)$$

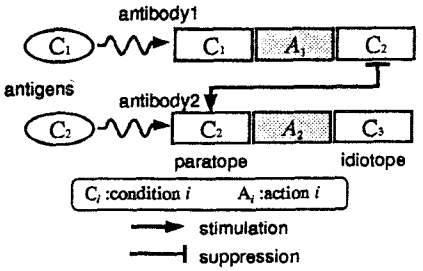


Figure 4: Interaction among antibodies.

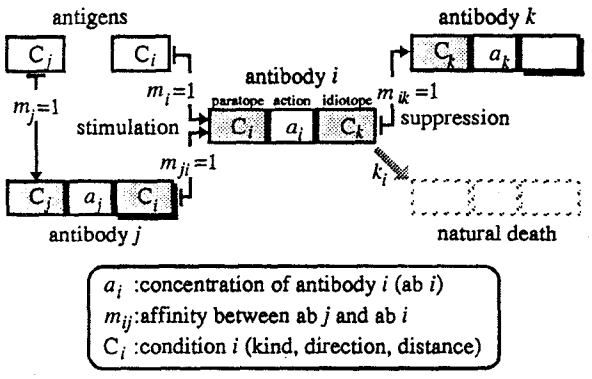


Figure 5: Dynamics.

where $I_j(k)$, $P_i(k)$ and $E_j(k)$ represent the k-th binary value of idiotope of antibody j, that of paratope of antibody i, and that of epitope of antigen j, respectively. L is the length of epitope, paratope and idiotope in binary string form, and w_k is the weight of k-th binary value. $\oplus$ denotes *exclusive-or* operator. The function $F(x)$ is 1 if $x \geq \theta$, $F(x) = 0$ if $x < \theta$, , where θ is the threshold of affinity. In this study, for simplicity, all w_k is equal to w, and $\theta = Lw$. Based on this assumption, m_{ji} is 1 only if the idiotpe of antibody j completely matches the paratope of antibody i.

Next, the concentration of i-th antibody, which is denoted by a_i, is calculated as follows:

$$\frac{dA_i(t)}{dt} = \left(\alpha \sum_{j=1}^{N} m_{ji} a_j(t) - \alpha \sum_{k=1}^{N} m_{ik} a_k(t) + \beta m_i - k_i \right) a_i(t) , \quad (3)$$

$$a_i(t+1) = \frac{1}{1 + \exp(0.5 - A_i(t+1))} , \quad (4)$$

where, in equation (3), N is the number of antibodies, and α and β are positive constants. The first and second terms of the right hand side denote the stimulation and the suppression from other antibodies, respectively. The third term represents the stimulation from the antigen, and the forth term the dissipation factor (*i.e. natural death*) [12]. Equation (4) is a squashing function to ensure the stability of the concentration. In this study, selection of antibodies is simply carried out on a *roulette-wheel manner* basis according to the magnitude of concentrations of the antibodies. Note that only one antibody is allowed to be selected and act its corresponding action to the world (*i.e. winner-take-all manner*).

4 Proposed Innovation Mechanism

4.1 Innovation mechanism

For more usefulness, as some researchers have been pointed out, the introduction of some adaptation mechanisms is highly indispensable. The adaptation mechanism is usually classified into two types: *adjustment* and *innovation* [16, 17]. Adjustment is an adaptation realized by changing parameters of the system (*e.g.* the modification of synaptic weights in neural networks). On the other hand, innovation is an adaptation realized by topological changes of the system, that is, the learning based on selection. An example of innovation is evolutionary process through genetic operators.

From this point of view, we address the candidates of adaptation mechanisms for artificial immune system. Adaptation for adjustmanet is realized by modifying molecular shapes of antibodies (*i.e.* affinity m_{ij} among antibodies). On the other hand, adaptation for innovation is realized by metadynamics function and selection mechanism.

In our previous research [7], we proposed the online adjustment mechanism that initially starts from the situation where the idiotopes of the prepared antibodies are undefined, and then obtains appropriate idiotopes using reinforcement signals. However, in our adjustment mechanism, we should notice that we must still describe the paratope of each antibody in a top-down manner.

One obvious candidate to avoid such problem is to incorporate an innovation mechanism. Therefore, we propose the following innovation mechanism inspired by the biological immune system, that is, metadynamics function and selection mechanism. Figure 6 schematically depicts the proposed innovation mechanism. As shown in this figure, the innovation mechanism is composed of two mechanism: the incorporation/removal mechanism and the selection mechanism. We discuss the detail of each mechanism.

4.2 Incorporation/removal mechanism

Initially, the immune network consists of N antibodies, each of them is generated by gene recombination and given one state variable named *"concentration of B-cell"*. In order to relate this variable to the action selection process, we modify the equation (2)

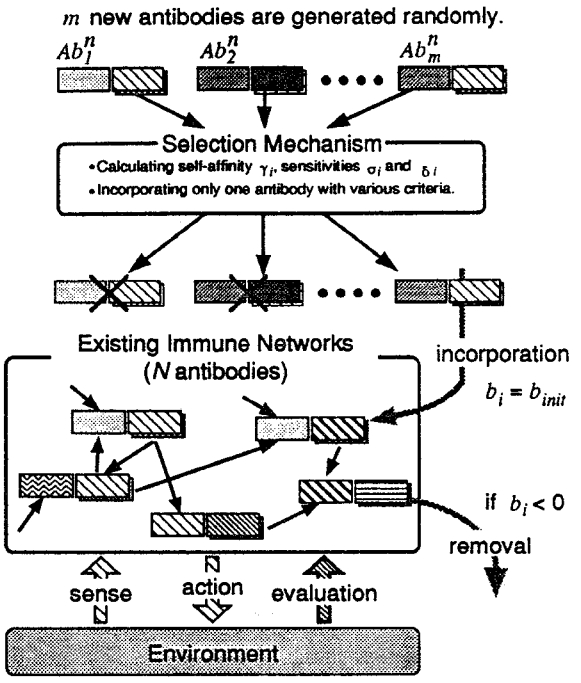


Figure 6: Proposed innovation mechanism.

as follows:

$$\frac{dA_i(t)}{dt} = \left(\alpha \sum_{j=1}^{N} m_{ji} a_j(t) - \alpha \sum_{k=1}^{N} m_{ik} a_k(t) + \beta m_i - k_i \right) a_i(t) b_i(T) , \quad (5)$$

where $b_i(T)$ is the concentration of B-cell i in T-th time step. This equetion means that the higher the concentration of B-cell is, the more offen its corresponding antibody is selected. If an antibody receives a reinforcement signal as a result of its action, the corresponding concentration of B-cell is varied as:

$$\frac{b_i(T)}{dT} = r_i \Delta b - K b_i(T) , \quad (6)$$

where K is the dissipation factor of B-cell. $r_i = 1$ if the antibody i is selected and receives a reward signal, and $r_i = -1$ if the antibody i receives a penalty signal. In addition, if antibody i stimulates the antibody that receives a reward, antibody i also receives a reward proportional to its concentration, that is, $r_i = a_i$, otherwise $r_i = 0$.

If $b_i(T)$ becomes below 0, the corresponding antibody is removed, then a new antibody is incorporated through the selection mechanism explained belows.

4.3 Selection mechanism

Next, we explain a selection mechanism inspired by the biological immune system. Before incorporating a new cell into the existing immune network, we randomly generate m candidates for antibodies by gene recombination process. In this study, only one antibody is allowed to be incorporated based on the predetermined criterion.

The possible candidates for the selection criteria are self-affinity γ_i, sensitivities σ_i and δ_i. These are expressed as:

$$\gamma_i = \sum_{k=1}^{L} \left(w_k \overline{I_i(k) \oplus P_i(k)} \right) , \quad (7)$$

$$\sigma_i = \sum_{j=1}^{N} m_{ji} a_j , \quad (8)$$

$$\delta_i = \sum_{j=1}^{N} m_{ij} a_j . \quad (9)$$

The self-affinity γ_i could be a measure of recurrent interactions, and the sensitivities σ_i and δ_i can be used to measure the impact of a newly created cell on the existing immune network.

5 Simulation

5.1 Problem

To confirm the ability of the proposed innovation mechanism, we carried out some simulations. The simulated environment contains immunoid, one predator and one prey (Fig.7). The predator attacks immunoid if it detects immunoid within the prespecified detectable range, otherwise, it moves randomly. On the other hand, the prey always moves randomly. The aim of immunoid is to be captured by the predator as little as possible and capture the prey as much as possible. To realize the aim, immunoid should select an appropriate action by flexibly varying the priorities between the predator and the prey according to the detected condition of each object.

Figure 7 also indicates the sensory inputs and actions of immunoid used in the simulation. Immunoid can detect the direction and distance to the predator and the prey. For simplicity, the detected direction and distance are categorized as:

- direction → front, front-right, front-left back, back-right and back-left
- distance → near, middle, far

And we assume that immunoid can move toward the above six direction. Based on the above assumption, we concretely define the condition (*i.e.* the epitope, the paratope and the idiotpe) and the action of each antibody(see Fig.8).

In the simulations, the following reward and penalty signals are used:

Reward ($r_i = 1$)

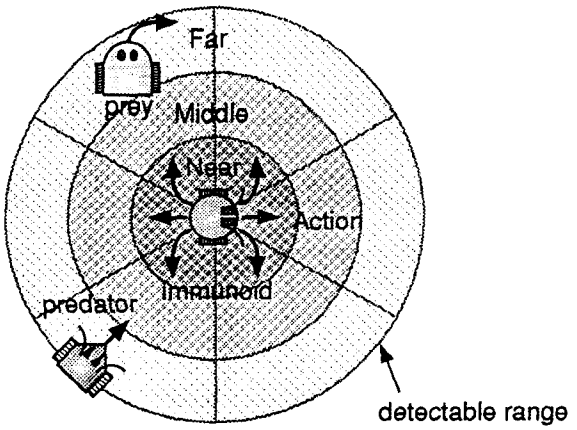


Figure 7: Simulated environment.

Condition (Antigen, Paratope, Idiotope)

Object	Direction		Distance	
Predator : 0	None	: 000	None	: 00
Prey : 1	Front	: 001	Near	: 01
	Front-Right	: 010	Middle	: 10
	Front-Left	: 011	Far	: 11
	Back	: 101		
	Back-Right	: 110		
	Back-Left	: 111		

Action

Front	: 001
Front-Right	: 010
Front-Left	: 011
Back	: 101
Back-Right	: 110
Back-Left	: 111

Figure 8: Conditions and actions.

- Immunoid approaches the prey.
- Immunoid escapes from the predator.

Penalty ($r_i = -1$)

- Immunoid escapes from the prey.
- Immunoid approaches the predator.

Additionally, we assume $N = 45$, $m = 20$, $\alpha = \beta = 0.7$, $k_i = 0.1$ and $K = 0.0003$. Δb is proportional to the distance as follows:

$$\Delta b = \begin{cases} 0.05 & \text{Near} \\ 0.10 & \text{Middle} \\ 0.15 & \text{Far} \end{cases} \quad (10)$$

5.2 Simulation Results

Figure 9 denotes the transition of number of capturing the prey and number of being captured by the predator in two cases. In case (a), the selection mechanism is not used, namely, one random generated antibody is incorporated, while in case (b), the selection mechanism is used with the criterion $\min \gamma_i$. From these results, the performance with the selection mechanism is improved more rapidly than that without the selection mechanism. We are currently analyzing these results in detail.

6 Conclusions and Further Work

In this paper, we proposed a new adaptation mechanism inspired by the innovation mechanism of the biological immune system, that is, metadynamics function and selection mechanism. And we applied it to the action arbitration for an autonomou mobile robot in the simulated environment and examined the ability of our proposed method. In the further work, we must analyze obtained results quantitatively and qualitatively and examine various criteria.

Acknowledgements

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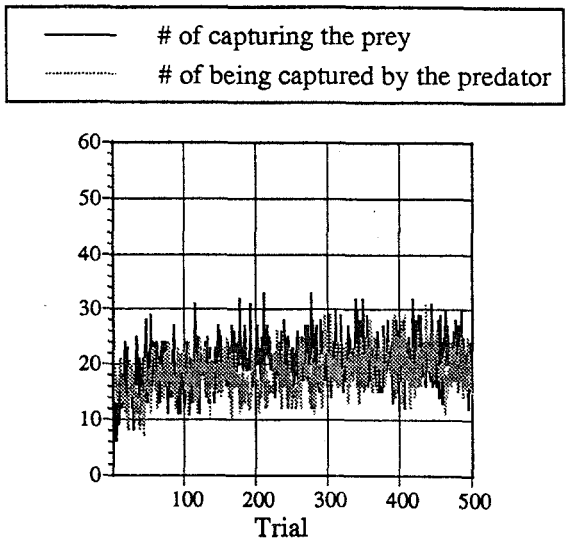
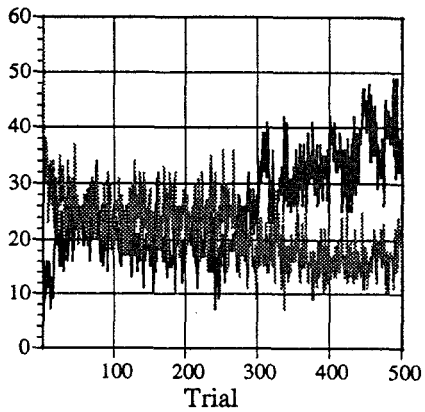


(a) Without selection mechanism.



(b) With selection mechanism (min γ_i).

Figure 9: Simulation results.