

Paper:

# A Neuromodulation Model for Adaptive Behavior Selection by the Cricket

## – Nitric Oxide (NO)/Cyclic Guanosine MonoPhosphate (cGMP) Cascade Model –

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Physiological research has shown the importance of neuromodulators such as nitric oxide (NO) in the pheromone behavior such as fighting behavior in insects. We focused on modeling function of neuromodulator in fighting behavior of crickets, and to emerge adaptive behavior selection by synthetic approach. In this paper, we propose a model for adaptive behavior selection by nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) cascade based on the physiological knowledge, and discuss the result of computer simulation.

**Keywords:** adaptive behavior selection, neuromodulator, nitric oxide, NO/cGMP cascade, cricket

### 1. Introduction

A major subject of study in the development of autonomous robots involves learning and adaptation. Although techniques such as neural networks [1] and reinforcement learning [2] remains inadequate.

Organisms select behavior adaptively based in part of according to their environment in real time. This is attributable to neural circuit network plasticity. Many studies have sought to clarify high-order motion control in animals through behavioral observation and physiological analysis.

Just how fragmented information obtained from analytical results operates in an actual system remains unclear, and we believe it is important to alternately repeat synthetic and analytical approaches in which a dynamic hypothetical model is simulated based on information from static physiological experiments and to verify the results obtained in further physiological experiments [3].

Meyland et al. [4] reported rhythmic motor behavior generated by flexible coupling with central pattern generators (CPGs) in the lobster's central nervous system, indicating such pattern generation is triggered by neuromodulatory stimulation of the connection among CPGs.



Fig. 1. Male crickets fighting.

Neuromodulation may also be responsible for individual adaptive interaction.

As example of adaptive behavior selection among individuals is the fighting behavior of crickets (Fig. 1), which is representative pheromone behavior in insects. Pheromone behavior emerges when pheromones are detected. We sought to clarify mechanisms behind adaptive behavior selection by modeling neuromodulator (NM) functions in the fighting behavior of crickets.

In behavior selection, nitric oxide (NO) is thought to function as a neuromodulator (NM) for extracting a specific behavior program from polymorphic circuits in the brain and that the NO/cyclic guanosine monophosphate (cGMP) cascade plays an important role [5].

Smith et al. [6, 7] proposed a controller, GasNet, using the concept of neuromodulation by NO gas and acquiring the architecture through evolutionary computation. Moreover, the purpose of their work was not to investigate an accurate neural model using the neuromodulation.

Kondo et al. [8] proposed a neural network model with dynamically-rearranged functions for an autonomous robot controller, realizing novel functions by incorporating the concept of dynamic rearrangement in biological neural networks using neuromodulators. While this model considered neuromodularity in an artificial neural network, the modularity effect is artificially pre-designed.

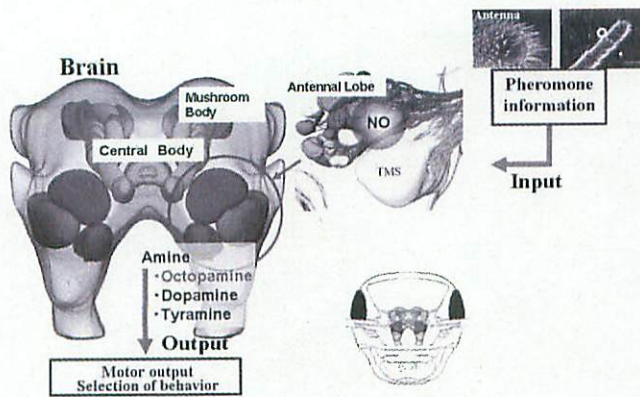


Fig. 2. Information pathway in the cricket brain.

We propose an adaptive behavior selection model inspired by the NO/cGMP cascade seen from the physiological viewpoint in cricket's fighting behavior. Our model consists of knowledges or results obtained by biological research on the cricket. We attempted to connect individual static biological properties and construct a dynamic model explaining adaptive functions by neuromodulation.

## 2. Cricket Fighting Behavior and Neuromodulators

### 2.1. Cricket Fighting Behavior

While pheromone behaviors of insects was once considered "hard-wired" and specific pheromones thought to cause specific behavior, it is becoming clear that the insect pheromone behavior involves plasticity by modification, as in cricket fighting behavior. Fig. 2 diagrams the information pathway in the cricket brain.

A cricket's body is covered with cuticular substances considered a so-called pheromone, probably for individual identification [9]. A male cricket encountering another cricket first touches it with its antennae to determine its gender, then attempts courtship behavior if the other cricket is female or fighting behavior if it is male (Fig. 3).

Such fighting behavior usually continues 15 to 30 minutes [10]. If the loser later encounters the winner or senses the same pheromone, it shows avoidance behavior i.e., experiencing defeat causes the cricket to select different behavior for the same stimulus adaptively based on the circumstances. Understanding the mechanism behind the switching of a program behavior could lead to clarify neural adaptation.

### 2.2. Neuromodulators and NO/cGMP Cascade

Organic neural systems involve a variety of neurotransmitters and NMs. NO, for one, is thought to play an important role in selecting program behavior from polymorphic circuits. NO diffuses in the brain at about 100 μm/s in three dimensions to control neurotransmitter emission [11]. Due to diffusion and reverse propagation, NO is

Singing, reproductive, and aggressive behaviors in a male cricket

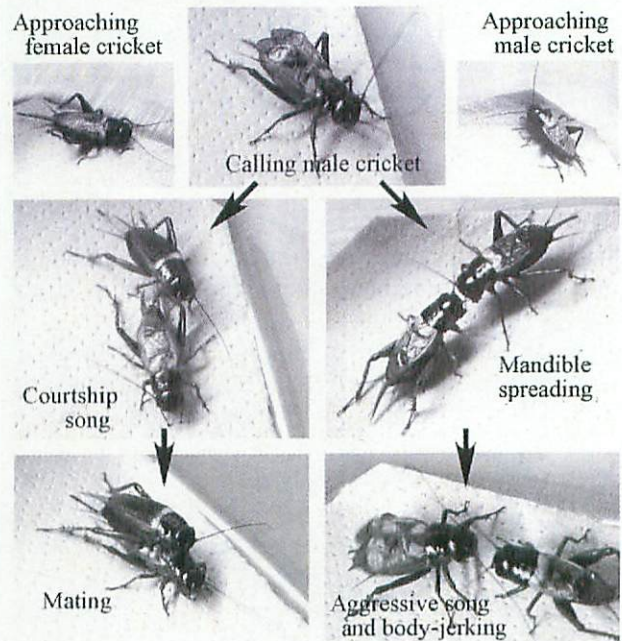


Fig. 3. Cricket behavior selection.

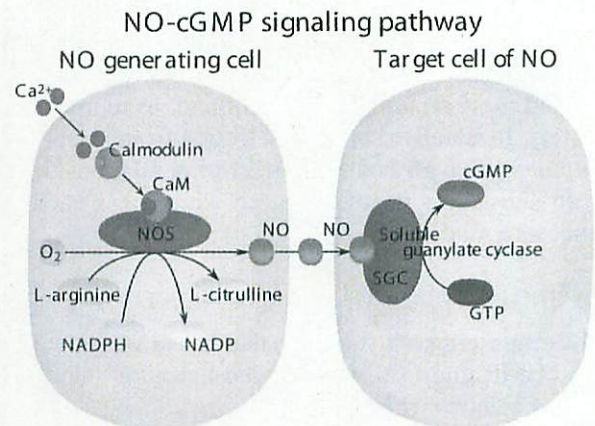


Fig. 4. NO/cGMP signaling pathway.

generally considered to be involved in neural plasticity as the basis of learning and memory [12].

The NO/cGMP cascade is considered particularly closely related to fighting behavior selection by crickets. Fighting behavior is observed when an NO synthesis inhibitor is injected into the head of a crickets. When two males encounter one another, fighting starts as usual and soon ends. If these crickets again encounter each other after a 15-minute interval, even the defeated cricket may exhibit fighting behavior instead of avoidance, indicating appropriate behavior is not selected if the NO/cGMP cascade functions normally in the brain. Even under these circumstances, the pheromone is identified [6] and NO is closely related to pheromone behavior modified by experience [13].

In the NO/cGMP signaling pathway (Fig. 4), the

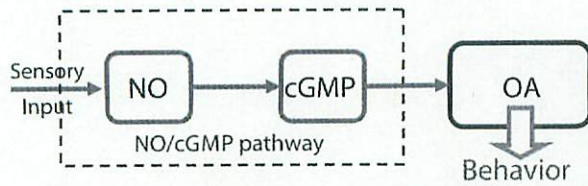


Fig. 5. Model of action selection from NO/cGMP cascade.

NO/cGMP cascade mediates the efficacy of different neural pathways, or circuits, which in turn affect the cricket's physiological condition. One change octopamine (OA) levels in the brain, for example [15, 16]. As recent research indicates (*Aonuma, unpublished data*), the amount of OA differs significantly before and after fighting and in winner's and loser's OA levels. The NO/cGMP cascade is thus considered deeply related to behavior selection through OA.

Given this background, we constructed a dynamic hypothetical model, discussed below.

### 3. Modeling Behavior Selection

In attempting to model NO/cGMP cascade function in cricket fighting behavior taking account with the relationship between OA concentration and behavior selection. Many neurons are complexly related in behavior generation and these relationships are difficult to reproduce accurately. In adaptive behavior selection from insect's programmed behavior however, describing relationships between substances is sufficient, so we express each substance as a module.

#### 3.1. Internal State Modeling of NO/cGMP Cascade

Noting a cricket's internal state based on amounts of NO, cGMP, and OA, we modeled behavior selection in fighting behavior (Fig. 5).

Simply put our model takes two states which are aggression and avoidance. Basing the internal state on the amount of OA, aggression causes the cricket to select fighting behavior and avoidance causes it to stay away from a previous winning cricket.

Based on sensory information from its antennae, a cricket first generates NO, and pheromones from another individual increase the NO concentration in the antennal lobe (AL). When NO is generated in the AL, the target range is considered the membrane surrounding the AL. If neural circuits are considered to consist of modules, no information is needed about neuronal regarding the linkage in the AL and only distance information is important. The AL is thus considered as a dimension of  $n$  [ $\mu\text{m}$ ]. The amount of NO in the AL is expressed as vector  $\mathcal{N}$  of  $(n + 1)$  dimensions and that at position  $x$  ( $0 \leq x \leq n, x \in \mathbb{Z}$ ) as  $\mathcal{N}^x$ .

$$\mathcal{N} = [\mathcal{N}^0 \ \mathcal{N}^1 \ \dots \ \mathcal{N}^n] \dots \dots \dots (1)$$

NO is a radical that reacts with metal ions immediately after diffusion throughout the brain, after which it disap-

pears, presumably present for about 10 seconds. To express this effect, the equation of NO diffusion with constant  $\gamma_N$  and diffusion constant  $D$  is expressed as follows:

$$\frac{\partial \mathcal{N}}{\partial t} = D \frac{\partial^2 \mathcal{N}}{\partial x^2} - \gamma_N \mathcal{N} + \mathcal{N}_{in} - \mathcal{N}_{out} \dots \dots (2)$$

where,  $\mathcal{N}_{in}$  and  $\mathcal{N}_{out}$  represent the amount of NO generation and consumption. Because NO mainly remains inside the membrane enveloping the AL, the following reflecting boundary is given as a boundary condition:

$$\mathcal{N}^0 = \mathcal{N}^1, \quad \mathcal{N}^n = \mathcal{N}^{n-1} \dots \dots \dots (3)$$

The NO diffusion property in the cricket brain is then modeled based on Eqs. (2) and (3).

cGMP in the NO target cell is generated when soluble guanylate cyclase (SGC) and guanosine triphosphate (GTP) mutually react, and SGC is driven by diffused NO from NO-generating cells (Fig. 4). cGMP affects the amount of OA for behavior selection function. The amount of cGMP in NO target cell  $C$  is expressed as a scalar value using the constant  $\gamma_C$  based on the idea that excessive amounts of cGMP are decomposed in the body:

$$\frac{\partial C}{\partial t} = -\gamma_C C + C_{in} - C_{out} \dots \dots \dots (4)$$

where  $C_{in}$  and  $C_{out}$  represent amounts of cGMP generation and consumption in the NO target cell.

The cricket selects behavior based on the amount of OA,  $A$  and OA is generated by cGMP and used to generate behavior. Likewise, the amount of OA is expressed by using constant  $\gamma_A$ :

$$\frac{\partial A}{\partial t} = -\gamma_A A + A_{in} - A_{out} \dots \dots \dots (5)$$

where  $A_{in}$  and  $A_{out}$  represent the amount of OA generation and consumption

The dynamics of each substances are modeled Based on Eqs. (2), (4) and (5).

In physiological experiments, technical constraints limit the ability to measure NO concentration in the AL, so we measure the NO concentration leaking from AL. The increased NO concentration in the AL is certain, but the degree of increase is unclear, so we assume that the amount of NO generation  $\mathcal{N}_{in}^x$  at position  $x$  is determined for position set  $\mathbb{I}$  at the source:

$$\mathcal{N}_{in}^x = \begin{cases} 0.0 & \text{if } x \notin \mathbb{I} \\ 1.5 & \text{if } (x \in \mathbb{I} \wedge \text{fighting}) \\ 1.0 & \text{otherwise.} \end{cases} \dots \dots (6)$$

This means that the amount of NO generated when the cricket fights becomes 1.5 times that of the normal amount without fighting.

Given that NO is completely consumed in cGMP production, NO consumption depends only on the NO concentration, so, the amount of NO consumption  $\mathcal{N}_{out}^x$  at position  $x$  is determined for position set  $\mathbb{O}$  at the source. According to [12], when the amount of NO in the brain is increased by a NO donor, the amount of cGMP in the NO target cell is multiplied by NO consumption. Modeling

property  $\mathcal{N}_{out}^x$  as a smooth function such as Eq. (7),

$$\mathcal{N}_{out}^x = \begin{cases} a \times (1 + \tanh(50 \times (\mathcal{N}^x - 0.4)))/2 & \text{if } x \in \mathbb{O} \\ 0 & \text{otherwise.} \end{cases} \dots \dots \dots (7)$$

where  $a$  is a constant and the amount of  $\mathcal{N}_{out}^x$  is expressed as  $[0:a]$ . Fig. 6(a) shows an example of the mapping function.

As stated, the amount of NO consumption in the NO target cells is related to cGMP generation. Expressing the amount of cGMP in the NO target cell as scalar value  $C_{in}$ ,  $C_{in}$  equals the total amount of NO consumption. Eq. (8) indicates that total NO consumption is used to generate cGMP.

$$C_{in} = \sum_x \mathcal{N}_{out}^x \dots \dots \dots (8)$$

Based on physiological experiments, OA is divided into that which is NO-independent and that which is NO-dependent. cGMP suppresses OA production, so cGMP consumption is modeled as a smooth function.

$$C_{out} = b \times (1 + \tanh(10 \times (C - 0.64)))/2 \dots (9)$$

If the amount of OA,  $A$ , is normalized to  $[0:b]$ , the amount of NO-dependent is about  $0.4b$  (Aonuma, unpublished data).

$$A_{in} = b - 0.6 \times C_{out} \dots \dots \dots (10)$$

Figure 6(b) shows an example of the mapping function using Eqs. (9) and (10).

OA is used in the cricket, to obtain energy from fat. Using the constant  $c$ ,  $A_{out}$  is:

$$A_{out} = \begin{cases} c & \text{if fighting} \\ 0 & \text{otherwise.} \end{cases} \dots \dots \dots (11)$$

This indicates that fighting behavior consumes large amounts of energy.

### 3.2. Fighting Behavior and Behavior Selection

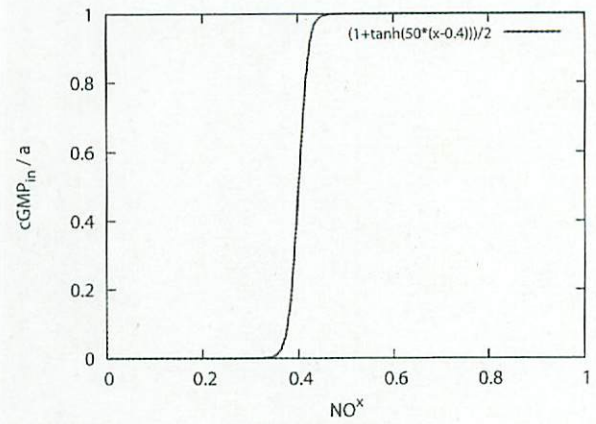
Bonabeau et al. reported the need of probability factors for ranking in a group [14] and Ashikaga et al. modeled the effects of breeding density and fighting behavior by introducing probability factors into behavior selection [17, 18]. A deterministic method appears suitable for analyzing the internal state of a single cricket.

As stated, the NO/cGMP cascade in the cricket brain affects its physiological condition changing OA levels. OA is a biogenic amine used to extract energy from fat.

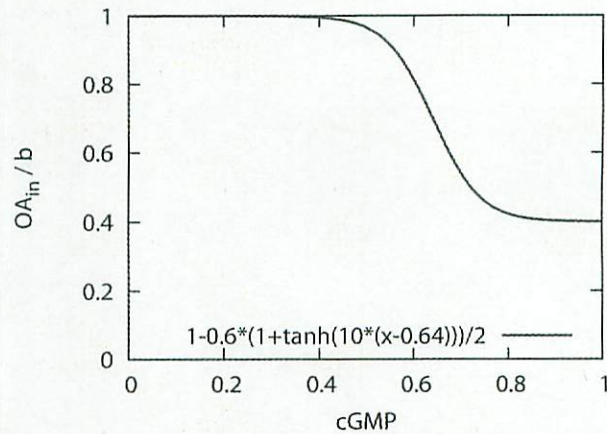
The simplest model assumes behavior selection based only on the amount of OA. When modeling behavior selection, however, fighting behavior itself must be modeled. Assuming that a cricket selects behavior at each step ( $\Delta t$  [sec]) during fighting, probability  $P$  of fighting in the next step is expressed as follows:

$$P = \text{sgn}(A - \Theta) \dots \dots \dots (12)$$

where fighting behavior is selected when  $A$  is over threshold  $\Theta$  and avoidance behavior is selected in other cases.



(a) NO to cGMP



(b) cGMP to OA

Fig. 6. Functions in our model.

We also assume that penalty  $-c$  is given to a defeated cricket after fighting. So, the post-fighting state is obtained deterministically if the pre-fighting state is given.

## 4. Computer Simulation

The behavior of the proposed model was verified in computer simulation in which the internal states of crickets were compared for changes between victory and defeat.

### 4.1. Experimental Setup

As stated, one position was determined for NO generation and consumption, and  $\mathbb{I} = \{50\}$  and  $\mathbb{O} = \{60\}$  were set. With diffusion coefficient  $D = 1000$ ,  $\gamma_N = 1.0 \times 10^{-1}$ ,  $\gamma_C = \gamma_A = 5.0 \times 10^{-4}$  was set as the effect where NO is diffused and disappears in about 10 seconds by reacting with metal ions.

Based on physiological experiments, the stationary state of cGMP was  $C = 0.6$  and that of OA  $A = 0.8$ . To satisfy these values,  $a = 1.82 \times 10^{-2}$  and  $b = 4.91 \times 10^{-4}$  were set. Hypothesis that NO operates as a type of switch, stationary states were ignored. The values were set  $n = 100$ ,  $\Delta t = 0.001$ ,  $\Theta = 0.5$  and  $c = 0.1$ . Fig. 7 gives NO states when the cricket is in an ordinary non-

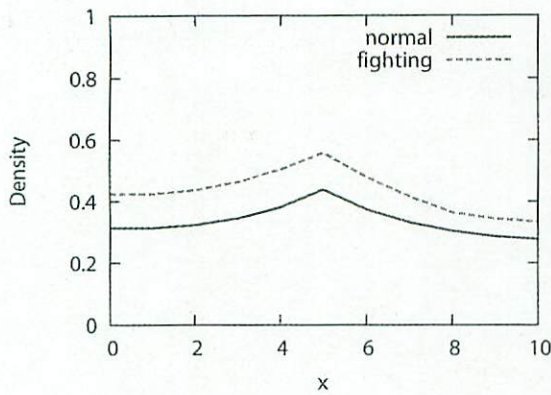


Fig. 7. Density of NO in antennal lobe.

fighting state and at 30 seconds after the start of fighting (ordinary state during fighting). With these settings, fighting behavior clearly increases NO levels in the brain and affects cGMP and OA values.

#### 4.2. Experimental Results and Discussion

In simulated of a single fight between two crickets and the internal state transition in the simulated winner and loser (Fig. 8). Fighting behavior starts at  $time = 3000$  and continues for 15 seconds in (a) and 30 seconds in (b).

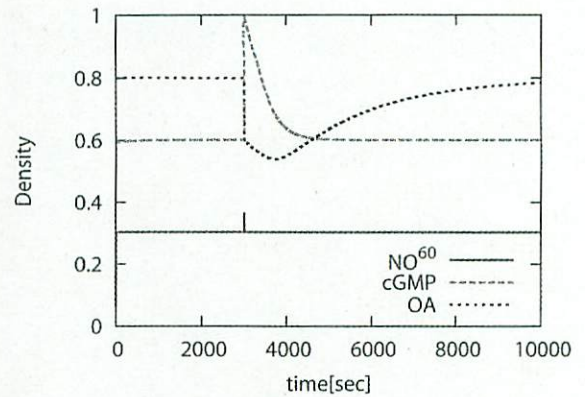
The proposed model takes either or state of aggression or one of avoidance, determined by the amount of OA (threshold  $\Theta = 0.5$ ). With aggression state leading to the selection of fighting behavior or avoidance behavior leading to non-fighting.

This model accurately represent behavior in which the cricket exhibits avoidance behavior after a loss and fighting behavior after sufficient time has passed. The effective time effect lasts just over 30 minutes, similar to actual observations, required for a defeated cricket to exhibit fighting behavior or for recovery of  $A = 0.5$ . This shows that time effects can be matched with actual data by varying  $\gamma_C$  and  $\gamma_A$ .

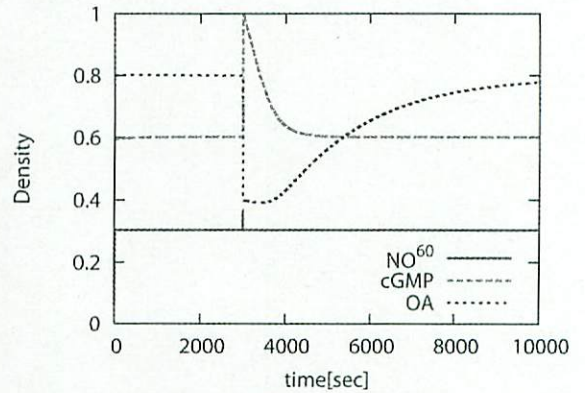
Results confirms whether another response can be expressed. When NO inhibitor is injected into the cricket's brain, fighting behavior selection increases even though the cricket has been defeated [10] so NO inhibition weakens that the memory of past experience. In simulation utilizing same model, if NO synthesis is inhibited ( $N_{in}^x = 0$ ),  $C$  becomes 0, and  $A = 1$  indicating that recovery will become shorter (Fig. 9) and that memory based on experience is weakened by blocking the NO/cGMP cascade. Our proposed model thus appears sufficient to explain adaptive behavior selection in crickets.

#### 5. Conclusions

We have modeled the cricket's neural system to understand principles of adaptive behavior selection, focusing on cricket fighting behavior based on pheromone behavior as a basic example of adaptive behavior selection. Neuro-modulators such as NO in the cricket brain are concerned



(a) The winner



(b) The loser

Fig. 8. Simulation result of internal state after fighting ( $\Theta = 0.5$ ).

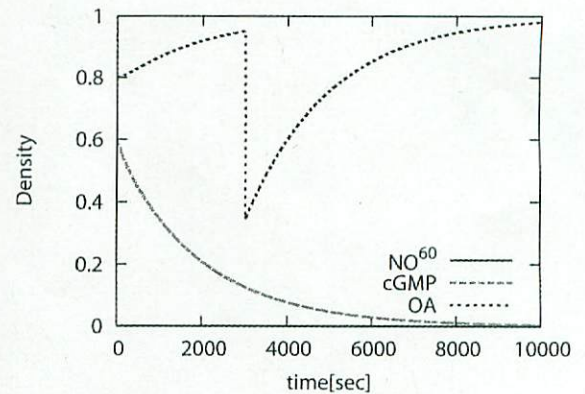


Fig. 9. Simulation result of internal state of loser after fighting (NO generation inhibited).

with the behavior selection. We have proposed a model of adaptive behavior selection involving the NO/cGMP cascade in cricket fighting behavior.

In projected work, we plan to establish a more accurate model of the neural system for adaptive behavior selection by continuing discussions with biologists on maintaining consistency between developed model behavior and physiological experimental results for the cricket. We also plan to evaluate the model in computer experiments involving a multi-individual environment.

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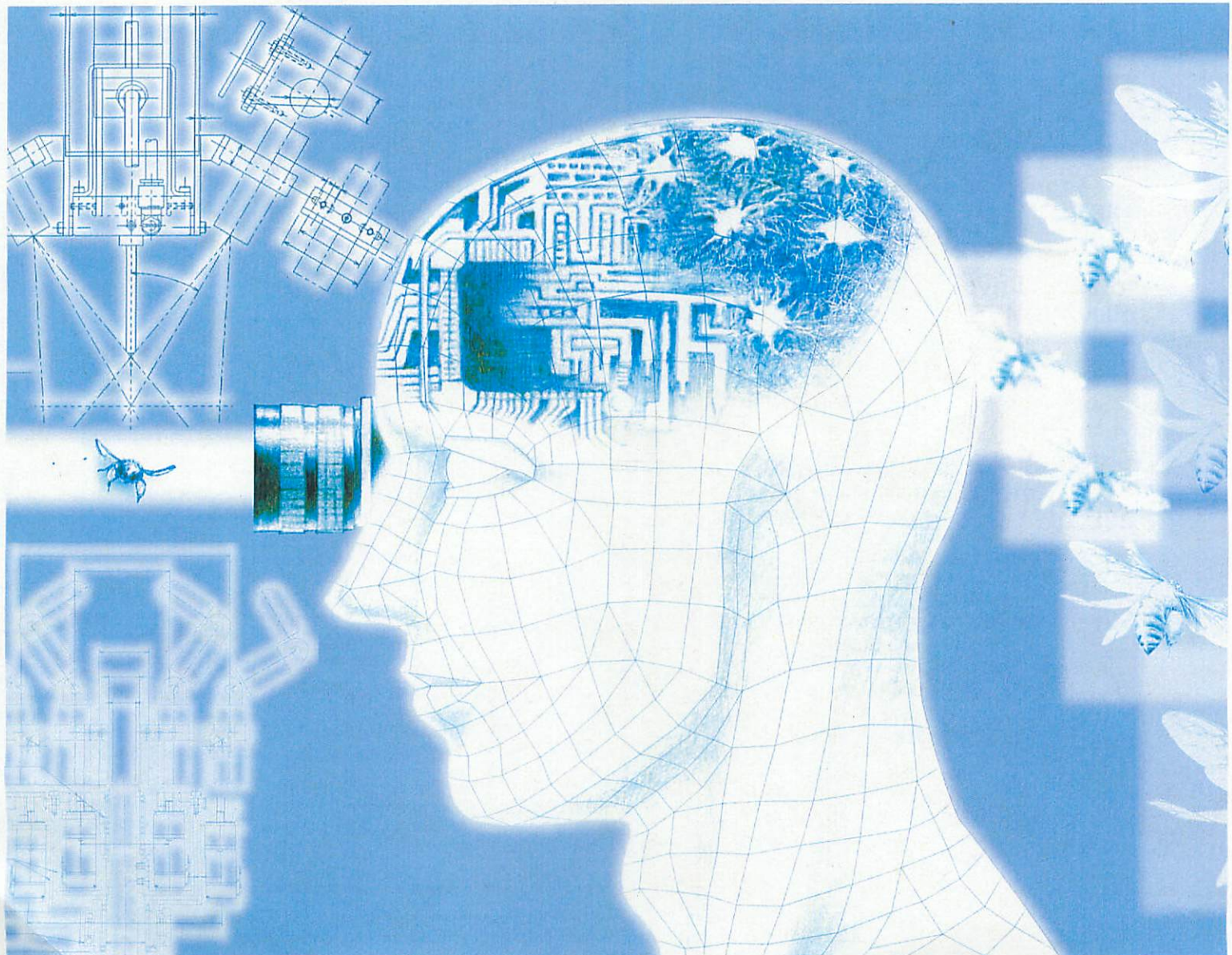
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