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A model of associative learning and classical conditioning in Aplysia californica: A preliminary study.

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Abstract

A simple invertebrate animal like the marine mollusc Aplysia exhibits an impressive variety of learning behaviour. The underlying biophysical and biochemical cellular mechanism has been for the most part well described by the group of Kandel. We propose a mathematical model which applies to the simple circuit of a synapse between three neuronal cells with non-hebbian learning rules and with temporal associations of conditioned and unconditioned stimuli. This model mimics the synaptic plasticity of the sensorimotor synapses responsible for the habituation, sensitization,classical conditioning of gill-withdrawal reflex. The evolution of the neuronal states is described by a set of differential equations for the dynamics of membrane potential, potassium conductance, calcium conductance and calcium concentration in dendritic and assonic regions.

Introduction

Learning behaviour of the marine mollusc Aplysia has been studied extensively and has become a good model for artificial neural networks which take into account interesting aspects of elementary forms of learning such as habituation, sensitization and classical conditioning[1].

Aplysia reacts to a stimulus on its siphon by the defensive response of contracting its gill through a neural circuit which consists of a population of about 25 sensory neurons that innervate the siphon and make monosynaptic connections with interneurons and with gill motoneurons[2-4].

A repeated mild stimulus to the siphon causes habituation with a decrease in the response which can last for several hours. An unconditioned stimulus as a shock to the tail cause an enhancement of defensive response with a sensitization to even the mild siphon stimulus that has caused the habituation behaviour. In the classical conditioning experiments the mollusc learns to anticipate an unconditioned shock to its tail following a conditioned stimulus[2-4]. All these learning mechanisms can be schematized by the same system of sensory neurons from the siphon that make excitatory monosynaptic connections with motor neurons that control the gill withdrawal (see Fig.1). The weight of the sensorimotor synapse is reduced during his habituation through a diminished influx of calcium ions into the presynaptic terminal. The associative changes produced by conditioning occur through presynaptic facilitation of the synaptic connection. This pre-modulatory coincidence mechanism is different from the pre-post coincidence proposed by Hebb[5]. The group of Card[6,7] has developed analog VLSI circuits to mimic qualitatively the learning

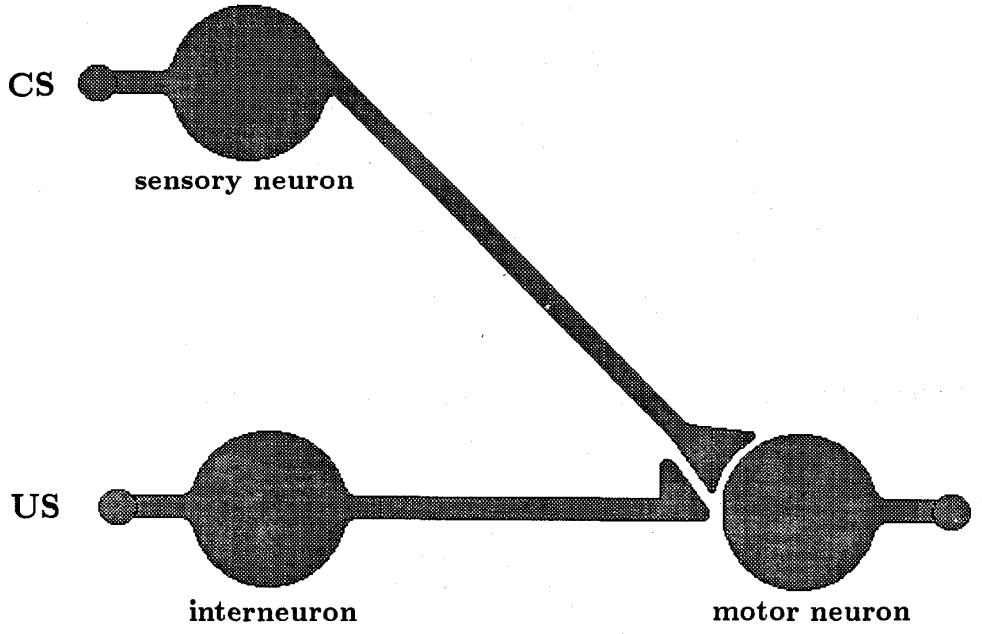

Fig.1. Diagram of gill-withdrawal reflex neural circuit in Aplysia. This ternary synapse includes inputs from both the sensory neuron (conditioned stimulus CS) and the the interneuron (which conveys unconditioned stimulus US).

mechanism of Aplysia. In this paper we describe a more realistic model which is based on the circuit of Fig.1 and takes into account ionic concentrations and conductances in the neuron cell.

The proposal model

The neuronal state is characterized by the membrane potentials, potassium conductances, calcium conductance and calcium concentration. Their values are kept in two distinct regions, the dendritic tree and the axon. The evolution of the neural state as a dynamical system is ruled by the following set of differential equations, modified from the two-point model neuron described by McGregor[8] :

$$\tau_M \frac{dV^D}{dt} = \begin{cases} -V^D + I^D + g_{DA}(V_S - V^D) + g_{Ca}(V_{Ca}^{Eq} - V^D) + g_K^D(V_K^{Eq} - V^D) & V^A \geq \theta^A \\ -V^D + I^D + g_{DA}(V^A - V^D) + g_{Ca}(V_{Ca}^{Eq} - V^D) + g_K^D(V_K^{Eq} - V^D) & V^A < \theta^A \end{cases}$$

$$\tau_K^D \frac{d}{dt} g_K^D = \begin{cases} -g_K^D + \varphi & \rho_{Ca} \geq \rho_0 \\ -g_K^D & \rho_{Ca} < \rho_0 \end{cases}$$

$$\tau_{Ca} \frac{d}{dt} \rho_{Ca} = -\rho_{Ca} + \gamma \cdot g_{Ca}$$

$$\tau_{Ca}^g \frac{d}{dt} g_{Ca} = \begin{cases} -g_{Ca} + \delta \cdot (V^D - \theta^D) & V^D \geq \theta^D \\ -g_{Ca} & V^D < \theta^D \end{cases}$$

$$\tau_M \frac{dV^A}{dt} = -V^A + g_{DA}(V^D - V^A) + + g_K^A (V_K^{Eq} - V^A)$$

$$\tau_K^A \frac{d}{dt} g_K^A = \begin{cases} -g_K^A + \beta & V^A \geq \theta^A \\ -g_K^A & V^A < \theta^A \end{cases}$$

The dendritic current I^D being the input stimulus, the response or output function is

$$V_O = V^A + s \cdot (V_S - V^A) + \eta$$

where V_S is the spike amplitude, η is a small white noise, and s is the spike variable

$$s = \begin{cases} 1, & \text{if } V^A \geq \theta^A \\ 0, & \text{if } V^A < \theta^A. \end{cases}$$

The apices A,D indicate the values of the variable in the axon and dendritic tree respectively. V^A,V^D are the membrane potentials,g_K^D,g_K^A the potassium conductances,ρ_{Ca} and g_{Ca} are the calcium concentration and calcium conductance. These variables characterize the state of the system, that can be therefore represented with a point $\mathbf{x}(t) = (V^D, g_K^D, \rho_{Ca}, g_{Ca}, V^A, g_K^A)$ moving in a six-dimensional vector space.

The properties of the neuron are fixed by the following list of parameters:
τ_M, τ_K^A,τ_K^D,τ_{Ca}, τ_{Ca}^g are the relaxation times for the membrane potential, dendritic and assonic potassium conductance, calcium concentration and calcium conductance in the dendritic region respectively.
V_K^{Eq},V_{Ca}^{Eq} are the equilibrium potential for the ions K^+,Ca^{++}.
θ^D,θ^A are the spike threshold for the potential in the dendrites and in the axon.
g_{DA} is the conductance between the two regions.
β is the rise in potassium conductance in response to a spike.
γ is the coefficient which bind the change of the calcium concentration whit the calcium conductance.
δ is the coefficient of calcium conductance change in response to a spike.
φ is the increasing in dendritic potassium conductance in response to an increase in calcium concentration.
ρ_0 is the threshold level of calcium ions which trigger potassium conductance.

We propose as learning rule the following time variation of synaptic efficacies $W(t)$ between three neurons:

$$W(t + \Delta t) - W(t) = \begin{cases} +W_0, & \alpha(t) > \alpha_2 \\ 0, & \alpha_1 \leq \alpha(t) \geq \alpha_2 \\ -W_0, & \alpha(t) < \alpha_1 \end{cases}$$

where W_0 is the amplitude of a sudden change in W, and $\alpha(t)$ is introduced as an additional variable for the short-term memory. The value of $\alpha(t)$ can be increased from a unconditioned stimulus only in a temporal window following the conditioned stimulus

$$\alpha(t+\Delta t) = \alpha(t) + \omega(t-t^*)\cdot(V_{US}-\theta_{US})$$

where t^* is the time of the last conditioned stimulus V_{CS},θ_{US} is a threshold for reinforcing action of V_{US} on α, and ω is the function

$$\omega(t) = \omega_0 \cdot e^{-\frac{t}{\tau_2}} \cdot (1 - e^{-\frac{t}{\tau_1}})$$

which is about zero except for the time interval (τ_1, τ_2). No biological interpretation of the variable $\alpha(t)$ is done, being his introduction justified only by a phenomenological modelling of simple neural system of the Aplysia.

The proposed learning rule for $W(t)$ is sufficient to mimic qualitatively the cellular mechanism of learning behaviour, and it can be used to build neural nets in which the temporal association of stimuli plays a fundamental role. Work is in progress to analyze the results of the simulations and to locate the limits of the model and the possible extensions.

The implementation of the model

Numerical simulations was carried on graphical workstation (SGI, Linux486).
The simple integration scheme used is

$$\frac{dx(t)}{dt} = -a(t)x(t) + b(t) \longrightarrow x(t+\Delta t) = x(t)e^{-a\Delta t} + \frac{b}{a}(1 - e^{-a\Delta t})$$

and this alghoritm was implemented in C language.

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