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Computational Model of Amygdala Network Supported by Neurobiological Data

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Abstract. The amygdala has repeatedly been involved in the processing of emotional reactions and conditioning. This paper presents a neurobiologically inspired computational model of the emotional memory in aversive behaviors. This artificial neural network aims at partially reproduce the same characteristics as the amygdala when it induces the aversive state experienced by individuals with a withdrawal effect.

1 Introduction

The amygdala is one of the basal ganglia, small islands of gray matter that lie deep within the white matter. It is located within the temporal lobes on each side of the brain and forms part of the limbic system. The amygdala is composed of two almond-shaped, fingernail-sized structures that are connected to most brain areas, especially advanced sensory-processing areas. Its principal task is to filter and interpret incoming sensory information in the context of our survival and emotional needs, and then to help initiate appropriate responses[5].
The objective of our project is to propose a computational model of emotional memory in order to validate neurobiological data observed in the amygdala nuclei in the morphine weaning experiment. This study is based on the following hypothesis. The associative memory process (which is the root of relapse) is caused among other things by a persistent modification of synaptic transmission and by the activity of the neural network of amygdala and associated structures. The aim is then to elaborate a neural network with the following constraints. On the one hand the learning or the associative memory only exploits the Hebb's rule. On the other hand connections and the different types of neurons respect the neurobiological data.

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2 Neurobiological data

2.1 Amygdala

The amygdala is a complex structure, consisting of about twelve distinct nuclei[6]and we decided to group them into two parts: the basolateral nucleus *BlA* and the central nucleus *CeA*. We consider there are only inhibitory neurons in the CeA and there is approximatively 10 percent of inhibitory neurons in the BlA. The amygdala is linked to other cerebral areas. We are particularly interested in the link with the hippocampus which has to centralize the different sensory inputs in order to activate some neurons that can recognize a specific environment. Sensory information is not only processed by the conscious system but also by the amygdala. It allows animals to automatically react to emotional stimuli (the fear for example)[2]. This is precisely the kind of behavior that has been considered. Finally a simplified input/output diagram(fig 1) of the amygdala has been used.

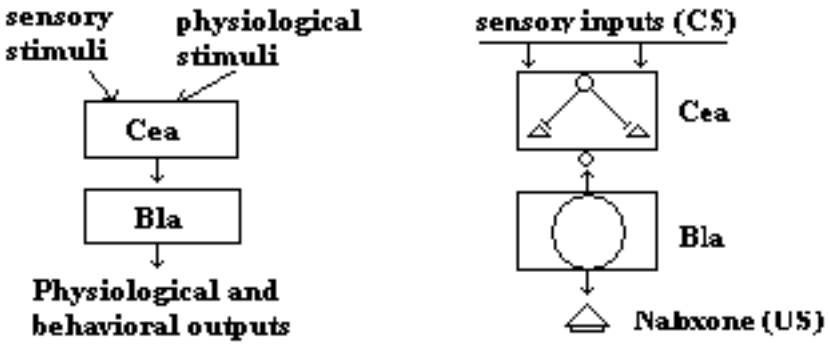

Figure 1: Simplified diagram for the amygdala. Bla is the basolateral nucleus CeA is the central nucleus and ITC is the inferior temporal cortex. A triangle represents an excitatory neuron and a circle represents an inhibitory one.

2.2 Drug addiction's effects

The presence of drug creates an overproduction of dopamine which creates a sensation of pleasure[1]. Our goal is to create a model of the withdrawal and this state cannot be considered as an external stimulus. Therefore the amygdala should play a role in the withdrawal effect. It can be explained by a dopaminergic network: the axons take the meso-cortico-limbic way. Many studies have shown the essential role of this way in the effect of relapse[4]. We have made the hypothesis in our study that this way is operative in the withdrawal effect.

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3 Methods and Experimental results

3.1 Methods

Some meatballs of morphine are given to the rats. Once they are swallowed morphine is regularly released in the body. Rats are then weaned by injection of naloxone. This action is called *US* (Unconditioned stimuli) because it does not depend on the rat's past. It has a direct effect on the withdrawal. This action takes place in a box with separated compartments. The box is in a room we call the *Env+* (Environment). The presentation of one of the compartments to the rats will be the *CS*(Conditioned Stimuli), respectively CS+ for the one where the weaning of morphine is and CS- for the other. The rats are used to 2 rooms. Six days long, they are alternatively placed in these different rooms, one day in the first room and the next day in the other. They are weaned in the same compartment three times, the three other days they receive in the other compartment a saline solution, which is assumed to be neutral.

	Injections		tests
Group	CS+	In the box	Env+
Control	Saline	Saline	Hazard
Env-	Saline	Naloxone	Hazard
Env+/CS+	Naloxone	Saline	CS+
Env+/CS-	Naloxone	Saline	CS-

Table 1: summary of the different steps of the experiment

4 Experimental results

Experimental results (fig 2):

- A strong injection of naloxone: the CeA is very active contrary to the BlA but the few active neurons have a stronger activity than expected with a saline injection.
- A new exposure to the weaning context: the BlA is clearly active and the CeA shows a slight activity.
- The associative learning is located in the BlA.

The biological study of these nuclei determines the internal structure of the layers of our model

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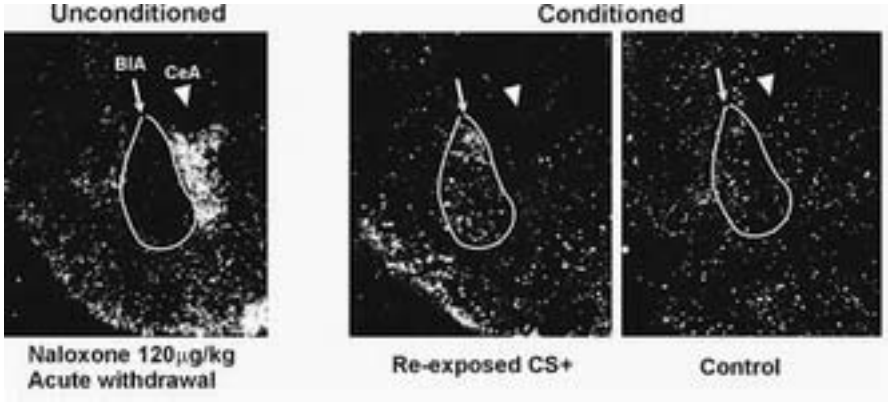

Figure 2: Measure of c-fos mRNA expression at the anatomical and cellular levels after in situ hybridization with radioactive probes

5 Simulations

5.1 Parameters

We have simulated the different amygdala's nuclei by a neural network. The neural activity surrounding a synapse can modify the synaptic weight of a connection. This phenomenon induces the synaptic plasticity [3]. In our case, we consider that the change is always an intensification. Then this rule can be mathematically defined by the following formula (1):

$$w_i^{t+1} - w_i^t = a.y.x_i \tag{1}$$

with a a constant which defines the learning rate, w_i^t the weight of the connection at time t , y the activity of the effective neuron and x_i the activity of the receptor neuron. A threshold is also used to prevent an infinite increase of the weight.
The output function of the neuron is a sigmoid (2):

$$\begin{cases} f(x) = 0 & \text{if } x \leq 0.2 \\ f(x) = N.\frac{1}{1+\exp\theta x} & \text{if } x > 0.2 \end{cases} \tag{2}$$

In fact, neurons follow the law all or nothing but this behavior has not been implemented in our model because we have chosen to represent several neurons by one. It allows us a quantification. In fact, instead of having neurons with their output values between 0 and 1 the obtained result is in the range $[0, N]$. This option allows simplifying the output values even if the quantification is conserved. In order to make an associative learning we propose a network with 7 inputs among which an inhibitory one, 3 for the environment (a known one and several others), 2 for the context, 1 for the presence of naloxone and the other

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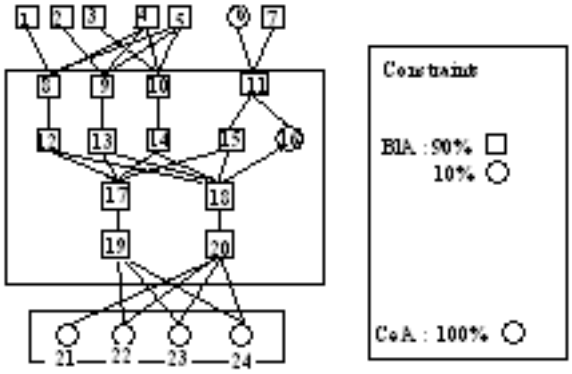

Figure 3: Obtained network: it respects data given by the team of neurobiologists. Squares represent excitatory neurons and circles inhibitory ones.

for the presence of dopamine (fig 3). We have chosen an inhibitory neuron for the naloxone. Indeed the naloxone prevents the activity of the neuron activated by the morphine. According to neurobiological data, a mechanism has to be found to obtain a stronger activation of a neuron thanks to the inhibitory action of another one. It is defined by the successive processing of neurons 11, 15, 16 and 18. When 6 is activated (injection of naloxone) 11 is less active and thereafter 16 is less active too. As a consequence 18 is more active due to the primary inhibitory action of the naloxone. Furthermore neurons 8, 9, 10 allow the association of the environment and the context. Neurons 12, 13, 14 and 17 are useful for the synchronization with neurons 11, 15, 16 and 18. Neuron 19 is useful for the inhibitory action of the CeA and neuron 21 corresponds to the group of neurons included in the CeA that are not connected to the BlA. Indeed it is necessary to allow the CeA to be active even if neuron 19 is not.

5.2 Tests

We have tested the network before learning and after in order to compare the simulated data to the experimental ones. We have analazed the number of activated neurons in the two nuclei and the activity of the neurons whether or not they are a little activated. Indeed, it's important to dissociate the quantity of the quality because the experimental results do.
We have then analazed 4 cases the one with naloxone and the one without before and after learning.
Before learning when there is no injection of naloxone, neuron 11 is very active. This implies the activation of 16 and then 18 is not active enough to enable Hebbian learning. The result is that the CeA is not active. When there is an injection of naloxone, neuron 18 is active, and neuron 20 is active enough to excite all neurons of the CeA.

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After learning when there is no injection of naloxone, the connection between neurons 12 and 18 is sufficient to generate the firing of neuron 20 despite the inhibitory action of neuron 16. Since the seventeenth to nineteenth connection has been reinforced there is only one neuron active in the CeA: neuron 21. We obtained the same result with an injection of naloxone.

6 Conclusion

It is a first attempt to take into consideration neurobiological information and the result of behavioral experiments in the design of an artificial neural network. All simulations performed well, indicating that the model is appropriate. However as experiments are still running, it is possible that we will have to add some connections or neurons in order to include in the model the new discovered properties. In addition, many questions remain. How to take into account that the learning stage last several minutes? Are there other parts in the brain that play an important role? Is there a unique interpretation of neurobiological data? Finally we can also suggest new experiments to confirm or invalidate new hypothesis on the structure and the functioning of the emotional memory in aversive behaviors.

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