

A MODEL FOR MEMORY AND FORGETTING

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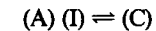
Abstract

Taking into account the idea of a biochemical equilibrium between "memory molecules", inhibitors and complexes, it is possible to find again the general shape of learning and extinction curves, and to explain various peculiarities of the learning phenomena, from short term habituation to reactive inhibition. The implications of this model are discussed in the context of current learning theories.

Several authors suggested the possibility of a molecular code for memory (see review by Chapouthier, 1983). Some experiments even suggested that the hypothetical "memory molecules" could be inhibited by some kind of substances (Braud, 1970). The purpose of the present paper is not to discuss the reliability of these results. Since inhibition is a very classical phenomenon in the nervous system, we attempted to study the properties of a mathematical model for memory and forgetting on the basis of a biochemical equilibrium between:

- 1) memory molecules
- 2) inhibitors

We will now study the following biochemical equilibrium (α):



where (A): is the molar concentration in memory molecules

(I): is the molar concentration in inhibitor

(C): is the molar concentration in complex (association of I and A)

The equilibrium follows the law of mass action:

$$(I) (A) / (C) = K$$

In all cases, we will admit that the behavioural response to a stimulus is proportional to the quantity (A) of free memory molecules. It is, therefore, interesting to study how this quantity varies. We will study two types of phenomena:

- the synthesis of new molecules A or I
- the modification of the equilibrium (α) without synthesis of new molecules.

Synthesis of Memory Molecules: Learning

At the instant 0, we have:

$$(I_0)(A_0) / (C_0) = K$$

Each trial (or set of trials) of training involves the synthesis of (a) new molecules. If the animal has been subjected to N trials, it will have synthesized (Na) molecules.

At the instant t , we have:

$$(A_t + Na)(I_t) / (C_t) = K$$

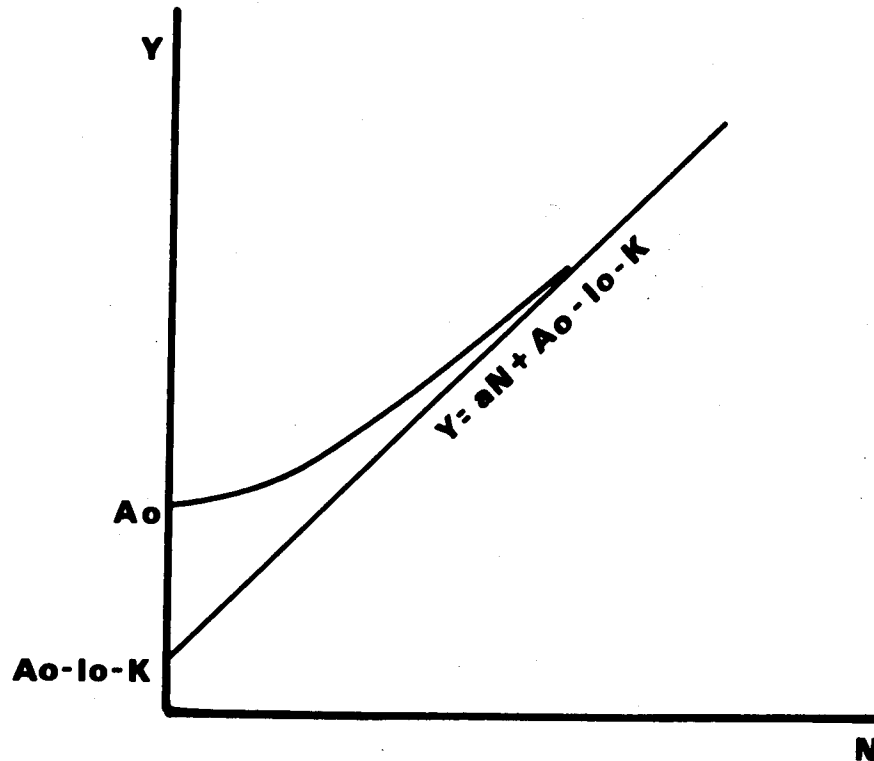


Fig. 1. $y = f(N)$ in the case of learning.

where ($A_t + Na$) is the new concentration of free memory molecules, (I_t) the new concentration of inhibitors, (C_t) the new concentration of complex.

We also have the following equations:

$$\begin{aligned} (I_0) + (C_0) &= (I_t) + (C_t) \\ (A_0) + (C_0) &= (A_t) + (C_t) \end{aligned}$$

which mean that the *initial quantities* of memory molecules and inhibitors are *conserved* during the phenomenon.

The important quantity is $y = A_t + Na$ (quantity of free memory molecules which determines the behavioural response). We will now study how this quantity varies with the rank N of the training trials.

An easy count gives the expression of y as a function of N and of the constants A_0 , I_0 , C_0 and K . We find:

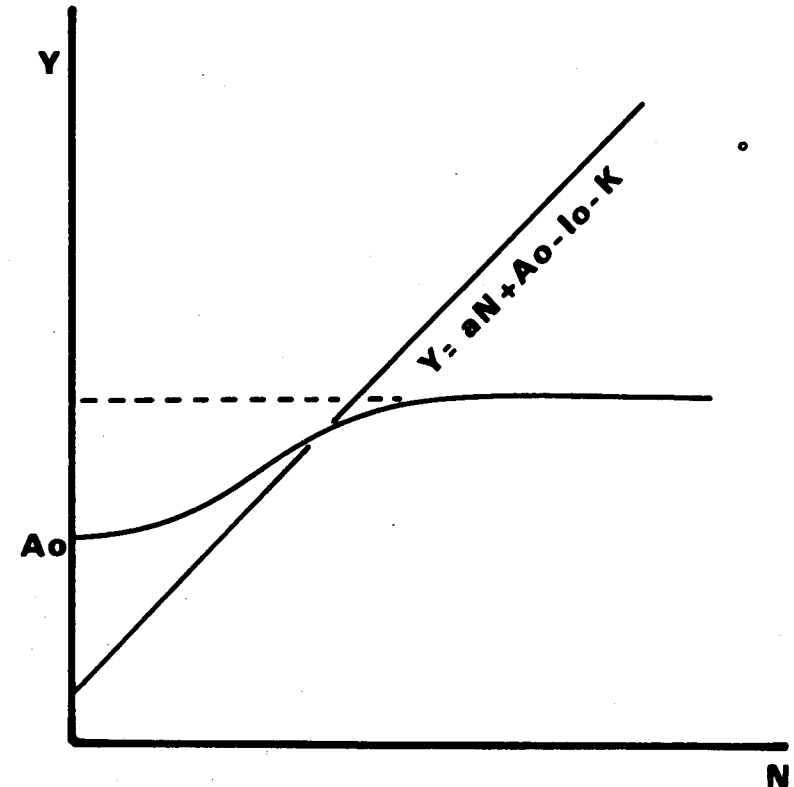


Fig. 2. $y = f(N)$ in the case of learning with saturation in memory molecules.

$$y = 1/2 \left[aN + A_0 - I_0 - K + \sqrt{(K - A_0 + I_0 - aN)^2 + 4K(A_0 + C_0 + aN)} \right]$$

For proper values of the constants, the graph of the function $y = f(N)$ is shown in Fig. 1.

Since the quantity y cannot be infinite, we will admit that, after a certain number of trials, a phenomenon of saturation in memory molecules (A') appears. The resulting graph is shown in Fig 2.

We obtain a graph which has the general shape of a learning curve.

Synthesis of Inhibitors: Extinction

As in the preceding study, we have, at the instant 0:

$$(A_0)(I_0) / (C_0) = K$$

Each trial or period of extinction produces the synthesis of (i) new inhibitors. If there is N trials or periods, we have the synthesis of (Ni) inhibitor molecules. At the instant t , the equilibrium is:

$$(A_t)(I_t + Ni) / (C_t) = K$$

The equations of conservation are:

$$\begin{aligned} (I_0) + (C_0) &= (I_t) + (C_t) \\ (A_0) + (C_0) &= (A_t) + (C_t) \end{aligned}$$

The important parameter is $y = A_t$ (quantity of free memory molecules which determines the behavioural response). Let us study how this quantity varies with the rank N of the extinction trials.

An easy count gives the expression of y as a function of N and of the constants A_0 , I_0 , C_0 and K . We find:

$$y = 1/2 \left[A_0 - I_0 - iN - K + \sqrt{(K + I_0 - A_0 + iN)^2 + 4K(A_0 + C_0)} \right]$$

For proper values of the constants, the graph of the function $y = f(N)$ is shown in Fig. 3.

There again, it is possible to imagine a phenomenon of saturation in the inhibitor.

We see anyway that the Fig. 3 has the general shape of extinction curves.

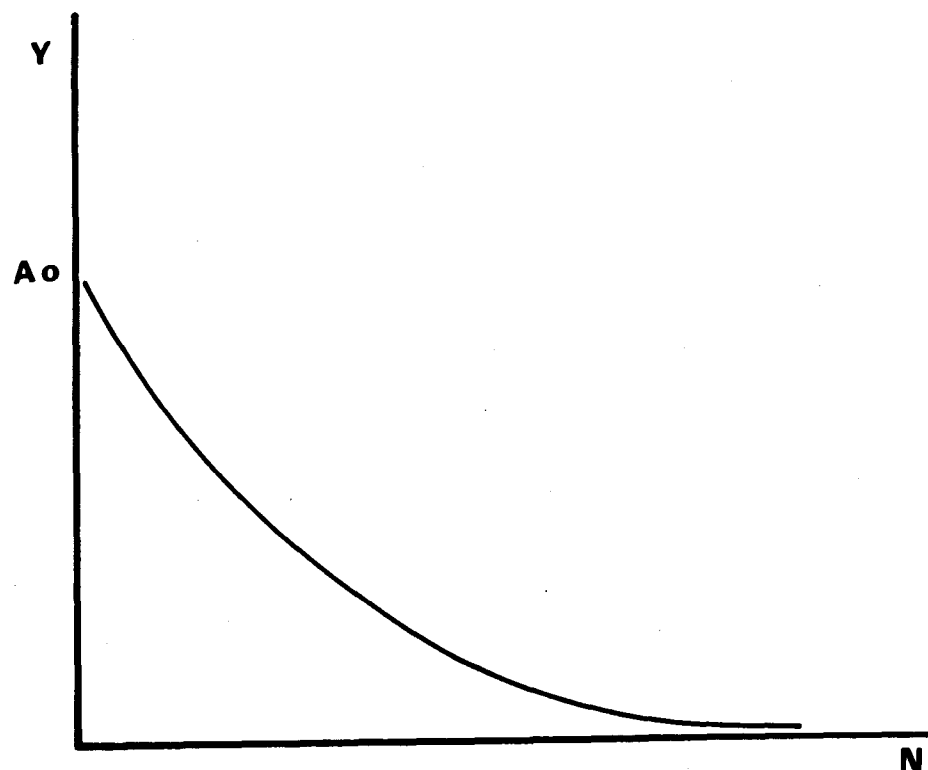
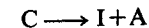


Fig. 3. $y = f(N)$ in the case of extinction.

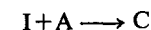
Short Term Variations

It is possible that some environmental factors, acting on the equilibrium, increase or decrease the quantity of (A) without any new synthesis. The *sensitization* could be explained by a destruction of C according to the reaction:



which increases the quantity of free memory molecules.

Short term habituation could be explained by the reaction:



in which A decreases.

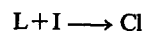
Verifications of the Model

Shape of the curves

The model explains the behavioural modifications found in the learning phenomena, including sensitization and short term habituation. It gives a theoretical explanation of the shape of learning and extinction curves.

Interpretation of reactive inhibition

Let us suppose that two "memory molecules" (one L responsible for a left turn, the other R for the right turn) have the same inhibitor I. If the animal makes x turns to the left without reinforcement, it will develop a short term habituation to the left, which will enhance the reaction:



The quantity of free inhibitors will therefore decrease. Because of the equilibrium:

$$(R) / (Cr) = Kr$$

this decreasing of I will enhance the reaction:



The quantity R will be increased. *An animal which has made many left turns will tend to make right turns.*

Discussion

This model has some interesting implications for memory mechanisms and forgetting. Because it is based on a biochemical equilibrium and thus the law of mass action, this model implies the conservation of the initial quantity of memory molecules, either in a free form (A), or within a complex (C). Moreover, new molecules (Na) can be synthesized as a consequence of learning.

The kinetic of acquisition is the same in this model as in Rescorla and Wagner's model of classical conditioning (1972): there is an increment of the number of free memory molecules from trial to trial, and these increments decrease as $(A_1 + Na)$ comes closer to the saturation value (A'). This point does not differ from any other current model.

A more important implication is when a certain amount of memory molecules are acquired: whatever happens at a behavioural level (extinction or forgetting), this quantity $(A_1 + Na)$ will always be conserved, but transformed in an inactive or "unusable" form (C). This conservation of the acquired memory molecules could

explain active and inactive memory as defined by Lewis (1979), or forgetting as a retrieval failure (Spear, 1971).

For example, the possibility to act on the equilibrium by some environmental factors (described in paragraph "Short term variations") could offer a biochemical explanation to the phenomenon of memory reactivation by some cues of the learning situation, as after a spontaneous forgetting (Deweere *et al.*, 1980) or an artificially induced amnesia (Miller *et al.*, 1974).

This conservation of acquired memory molecules is the main point of this model, and the important difference from learning theories (Rescorla and Wagner, 1972; Mackintosh, 1975), which have no clear implications on the long-term evolution of what is acquired during learning.

Acknowledgement

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