The mathematical model of trace conditioning

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Introduction

Why trace conditioning is difficult to model?

- It requires the filling in of the seconds/minutes time gap between CS and US by means of neurons and synapses that are operating on a millisecond time scale
- It requires an intact hippocampus, mPFC, amygdala, cerebellum, and functional interactions between them, by means of theta oscillations

The main problems of trace conditioning:

1. What is the neuronal mechanism of gap filling?
2. What does the hippocampus do during trace conditioning? What are specific roles of the mPFC, amygdala, and cerebellum?
3. Where is the trace in trace conditioning?

The aim of this present work is to build a unified trace conditioning model based on the three main principles of brain functioning: the principle of dominanta by Ukhtomsky (1908/2002), the author's principle of metastability (Kryukov et al, 1990) and Vinogradova's (2001) comparator principle.

Method and theory

The method used is a mathematical reduction of the multidimensional system of differential equations which describes the “Neurolocator” model of memory and attention (Kryukov, 1991, 2008) adjusting it for Pavlovian trace conditioning. Since the “Neurolocator” model is based on Vinogradova’s (2001) Theta-Regulated-Attention Theory the basics of this theory are presented first (see Figure 1A):

1. The Hippocampal CA3-field is acting as a comparator.
2. CA1-based limbic circle is being used as a delay line.
3. Medial septum is acting as a global pacemaker.
4. Theta rhythm is playing a crucial organizing role in perception, attention and memory as an attentional filter.

In addition, the “Neurolocator” model takes the following premises:

5. The functional unit of the model is not a single neuron but a unified submodule, much like the cortical microcolomn. It represents a relatively small network of locally connected excitatory integrate-and-fire neurons with a small number of

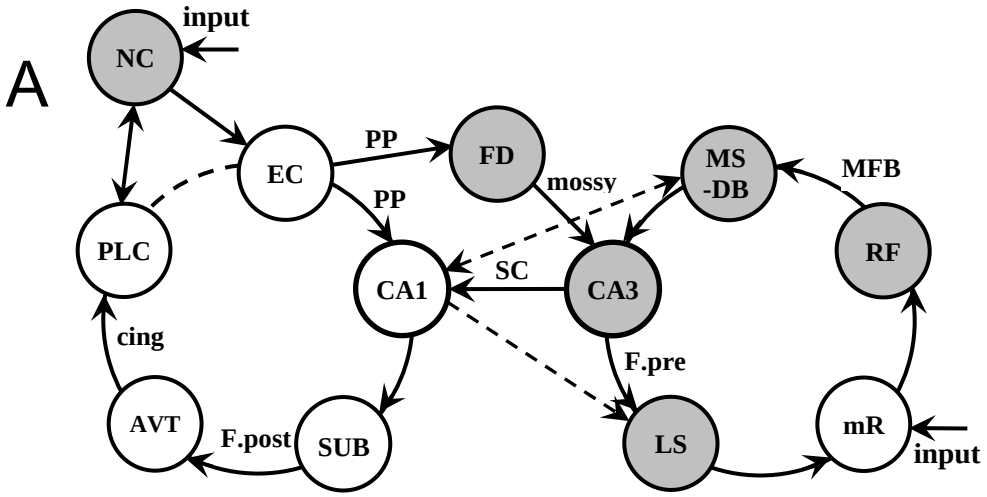
inhibitory interneurons (see Figure 1D) that are capable of oscillatory activity with specific natural frequency.

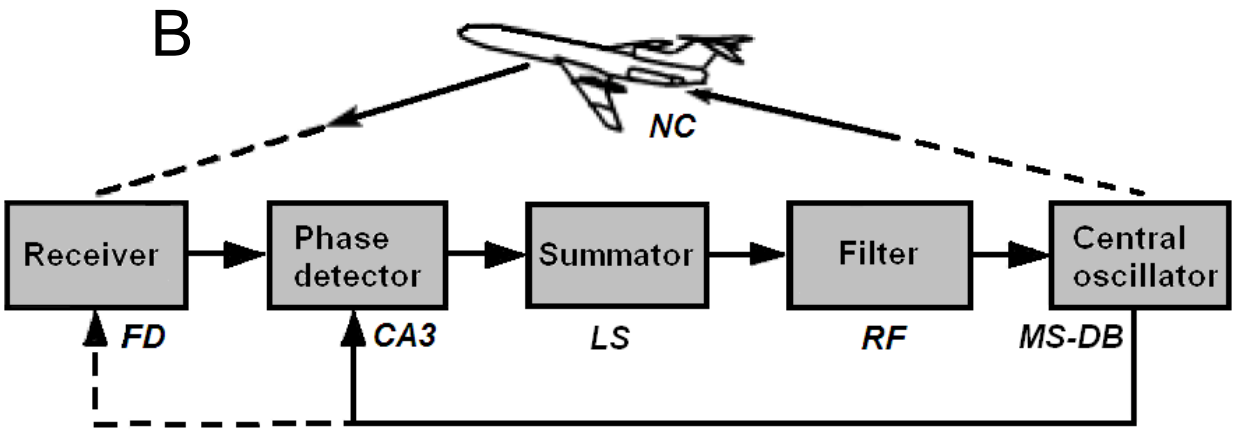
6. A new type of learning in an oscillatory system - *Isolability Assumption* - is assumed:

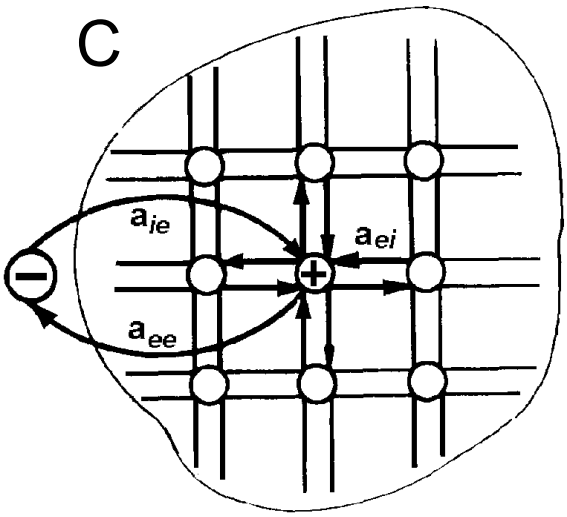
In this system when a number of cortical oscillators are locked in an ensemble and reaches the critical value, their labilities (i.e. natural frequencies) tend to be equalized by means of new protein synthesis during the consolidation/reconsolidation process

7. A star-like architecture is assumed consisting of cortical and sub cortical peripheral oscillators (PO) and one central oscillator (CO) (see Figure 1C); the theta frequency of CO is automatically regulated by POs through the control phase lock loop (PLL) system (see Figure 1B).

A large system of non-linear differential equations that are describing the behavior of the above model (not given here) is used to reduce it to a single differential equation, which explains most of the data on the trace conditioning. Two variants of reduction are used: one is the mean field approximation, and the another one is the random walk stochastic process with a deterministic drift.







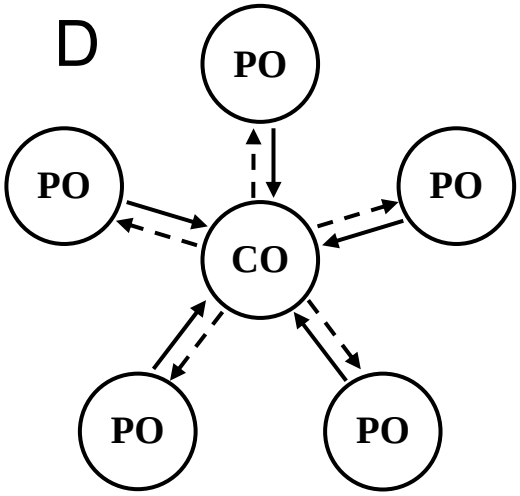


Figure 1 (A) Vinogradova's bicycle. Only principal blocks are shown as follows: CA1 and CA3 – hippocampal fields; FD – fascia dentate; LS – lateral septal nucleus; MS-DB – medial septal nucleus and nucleus of diagonal band; NC – neocortex; RF – reticular formation; (B) Corresponding block-scheme of PLL control system of "Neurolocator" model; (C) Scheme of interconnections of unified submodule; (D) Simplified star-like architecture with central oscillator CO and peripheral oscillators POs.

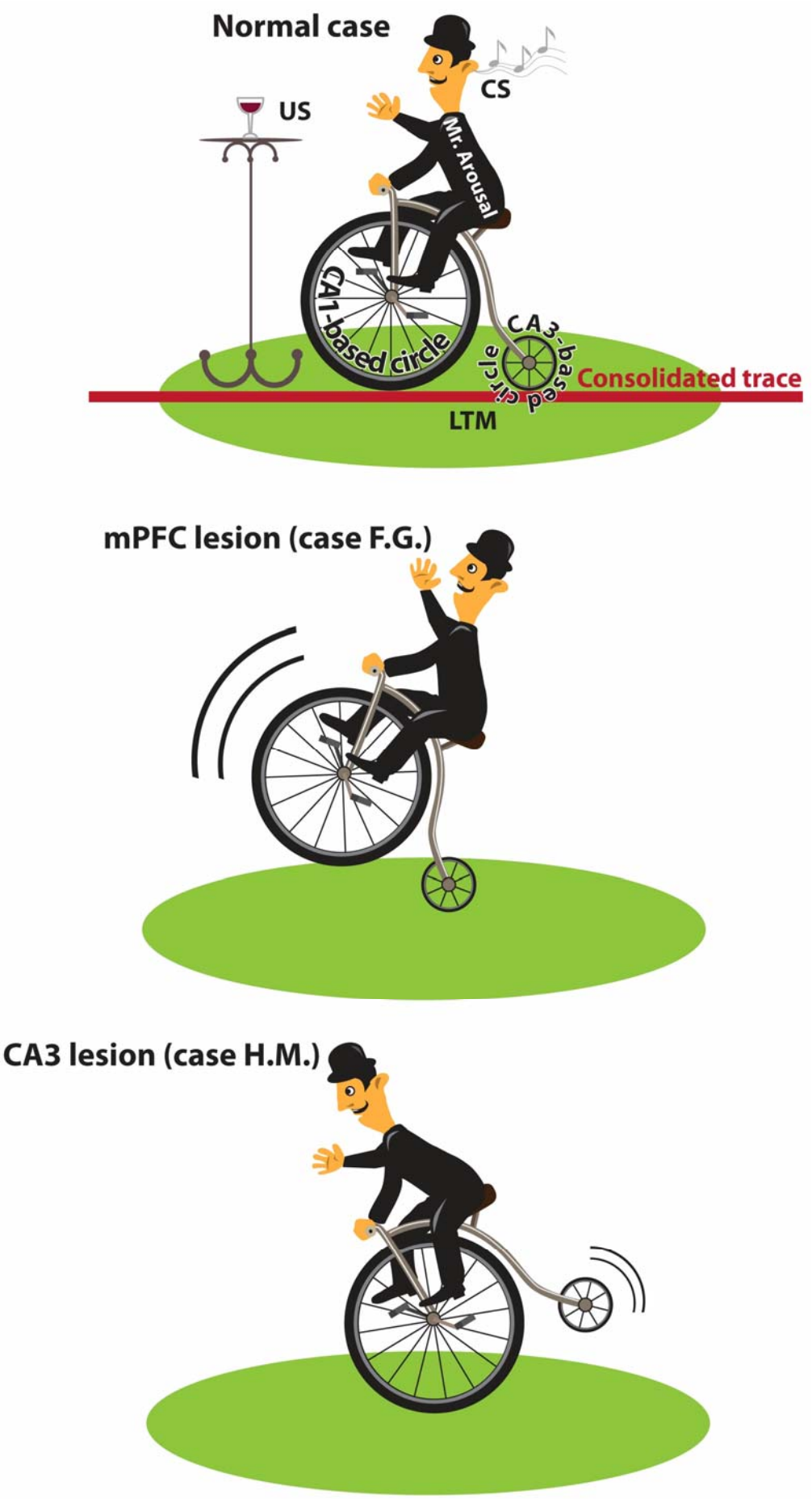


Figure 2 Vinogradova's bicycle as a mechanical analog of trace conditioning

Results

1. The numerous experimental data (e.g. the hippocampal and prefrontal cortical lesions) can be explained in terms of dynamical system properties of the “Neurolocator” model (see Figure 2, Table 1).
2. The mean field approximation equation of the original system provides the strict boundary conditions for the hippocampal involvement in trace conditioning in terms of the model parameters (see Figure 3).
3. The random walk approximation explains the scalar property (constancy of variation coefficient) of the timing in trace conditioning (see Figure 4).

Table 1

Main effects explained by the "Neurolocator" model:

7 hippocampal lesion effects (11 refs),
e.g. retrieval of memories is possible without hippocampus at short ISI (Chowdhury et al, 2005; Moyer et al, 1990)

6 attentional effects (15 refs),
e.g. attention is the key trigger for plasticity in A1 (Fritz et al, 2007)

5 timing effects (15 refs),
e.g. Weber's law (Kehoe et al, 2010) and backward conditioning (Quinn et al, 2002)

7 learning effects (23 refs),
e.g. if training takes place during computer defined specific time intervals of theta activity the number of required trials is reduced by factor up to 4 (Griffin et al, 2004; Berry and Hoffman, 2011)

$$g_{3min} < \frac{\Lambda_0}{A_{01} A_{02} K} \frac{ISI}{\tau_{CS}} < g_{3max}$$

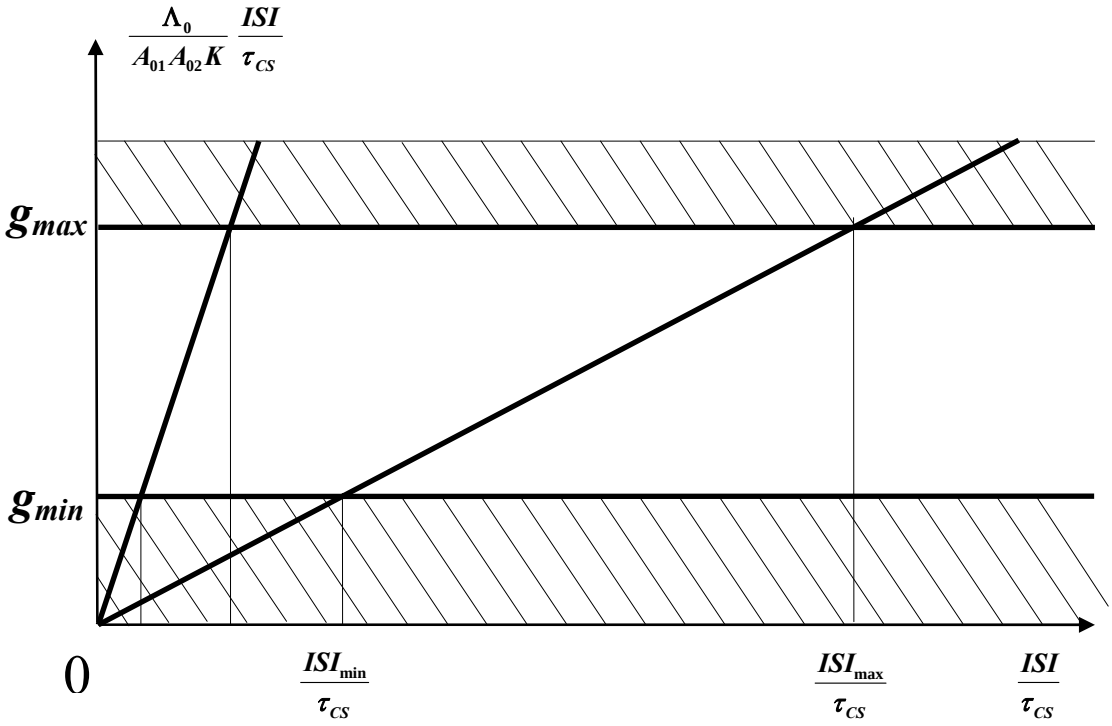


Figure 3 The upper and lower ISI-boundaries for the trace conditioning according to the above inequality, where ISI – interstimulus interval; A_{01}, A_{02} – CS and US intensities; τ_{CS} – duration of CS; K – loop gain; Λ_0 – initial detuning; g_{max}, g_{min} – constants < 1

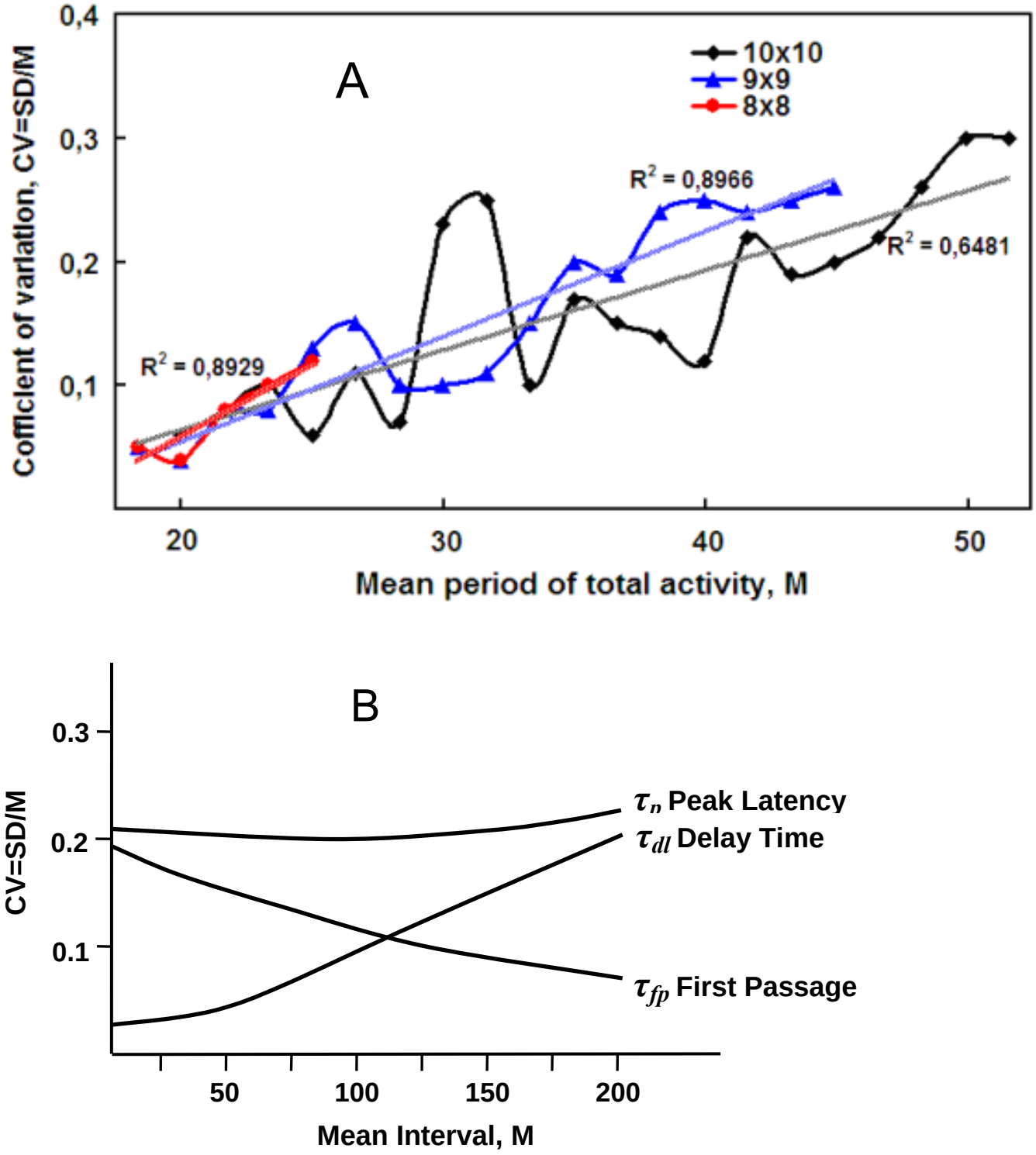


Figure 4 (A) Simulation results on CA1-based limbic delay line; (B) Constant CV of Peak Latency ($\tau_p = \tau_{dl} + \tau_{fp}$) can be at least partially explaind as a result of two opposite tendencies

Further Work

The preliminary author's (2011a, 2011b) results show that the "Neurolocator" model can solve both extinction and reconsolidation problems. However, the validity of these solutions can be firmly established only after the model can explain the new data of reactivation-extinction paradigm.

Further computational work is needed to build a large-scaled simulation variant of the "Neurolocator" model to check the main predictions of the mathematical theory.

Discussion

The above results show that the hippocampus can not be either a memory store or a novelty detector, but it has more of a general function of the double (space and time) comparator. Also, the model is described as non-Hebbian. This may strike readers as questionable. However, the Isolability Assumption as a new learning hypotheses can be considered as a spatial and temporal generalization of Hebbian learning – see Kryukov(2012).

Conclusions

1. The “Neurolocator” model with some adjustments provides a solution for the main trace conditioning problems stated in the Introduction.

2. Derived boundary conditions for the hippocampal involvement in trace conditioning in the “Neurolocator” model are providing unexpectedly simple explanation for the main trace conditioning “critical” effects, including some seemingly contradictory ones.

3. The “Neurolocator” model of trace conditioning predicts the existence of a unified model of general Pavlovian conditioning in which various conflicting findings are resolved and integrated into a single theory.

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