« Effects of Cellular Homeostatic Intrinsic Plasticity on Dynamical and Computational Properties of Biological Recurrent Neural Networks »

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Goal of the talk

To study the joint effects of synaptic and intrinsic plasticity on a neural network model.
Neurons and synapses.
Synapses and Plasticity

• The synaptic weight (cellular and molecular factors)
  – As a function of the quantity of neurotransmitters released at a time
Synapses and Plasticity

- The synaptic weight (cellular and molecular factors)
  - As a function of the quantity of receptors
Synapses and Plasticity

- The synaptic weight (cellular and molecular factors)
  - As a function of the type of receptors
Synapses and Plasticity

- The synaptic weight (cellular and molecular factors)
  - As a function of the uptake and the degradation
Synaptic plasticity.

**Long-Term Potentiation**: long lasting enhancement of synaptic transmission

**Long-Term Depression**: long lasting reduction in synaptic transmission
Let us assume that the persistence or repetition of a reverberatory activity (or "trace") tends to induce lasting cellular changes that add to its stability. When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.

Dynamical effects of Hebbian plasticity

Daucé & al, *Neural Networks*, 1998
The SNT model again

\[ u_i(t+1) = \gamma u_i(t) + \sum_{j=1}^{N} J_{ij} f(u_j(t)) - \theta_i + \sigma B_i(t) \]

\[ u_i(t + 1) = \sum_{j=1}^{N} W_{ij} f(u_j(t)) - \theta_i \]

\[ x_i(t) = f(u_j(t)) \]

\[ x_i(t + 1) = f \left( \sum_{j=1}^{N} W_{ij} x_j(t) - \theta_i \right) \]
Effects of Hebbian learning.

Hebbian learning.

\[ W_{ij}^{(T+1)} = \lambda W_{ij}^{(T)} + \frac{\alpha}{N} \Gamma_{ij}^{(T)} \]

« Small » parameter.

depends on pre- and post-synaptic neuron « activity ».

Decay rate (forget)

« passive LTD »

Neurons dynamics

\[ x_i^{(T)}(t+1) = f \left( \sum_{j=1}^{N} W_{ij}^{(T)} x_j^{(T)}(t) - \theta_i^{(T)} \right) \]

Example.

\[ m_i^{(T)} = \frac{1}{\tau} \sum_{t=1}^{\tau} x_i^{(T)}(t) - d_i \]

\[ \Gamma_{ij}^{(T)} = m_i^{(T)} m_j^{(T)} H \left( m_j^{(T)} \right) \]
Effects of Hebbian learning.

Coupled evolution of neurons and synapses.

Nonlinear neurons dynamics depends on the synaptic graph structure.

Synaptic graph evolves according to the neurons activity.
Generic effect of Hebbian learning.

Changes in the network structure.

Increase in the number of positive circuits.
Generic effect of Hebbian learning.

Changes in the network structure.

Increase in the number of positive circuits.

Dynamical effect.

Reduction of chaos.
Generic effect of Hebbian learning.

\[ m(T) = \frac{1}{N} \sum_{i=1}^{N} x_i^{(T)}(t_0) \]
Generic effect of Hebbian learning.

Th. (Siri, Berry, Cessac, Delord, Quoy, 2007)

\[
\| W^{(T)} \| \leq \lambda^T \| W^{(1)} \| + \frac{\alpha}{N} \sum_{n=1}^{T} \lambda^{T-n} \| \Gamma^{(n)} \| \\
\lambda_1^{(T)} \leq \log(\| W^{(T)} \|) + \left\langle \log(\max_i f'(u_i)) \right\rangle^{(T)}
\]
Empirical distribution of the mean local field

Before learning for the stimulus-forced dynamics.

After learning for the stimulus-forced dynamics.

After learning, with the dynamics induced by another random non-learned stimulus
Generic effect of Hebbian learning.

\[ \Delta^{(T)} = \frac{1}{N} \sqrt{\sum_{i=1}^{N} \left[ \langle f'(u_i) \rangle^{(T)} - \langle f'(u_i') \rangle^{(T)} \right]^2} \]
Reactivity to random stimuli on a population of 50 networks before and after learning, with $g=15$.

Reactivity to noisy versions (noise=20%) of the learned stimulus on a population of 50 networks before and after learning, with $g=15$.

Generic effect of Hebbian learning.

Changes in the network structure. → Increase in the number of positive circuits.

Dynamical effect. → Reduction of chaos.

Fonction acquisition → Pattern recognition by dynamical reduction.
A movie
Is there a bio physically relevant mechanism allowing to stabilize the network at the edge of chaos?
Persistent modification of a neuron’s intrinsic electrical properties by neuronal or synaptic activity.

Mediated by changes in the expression level or biophysical properties of ion channels in the membrane.

Can affect such diverse processes as synaptic integration, subthreshold signal propagation, spike generation....

regulator of synaptic plasticity underlying learning and memory

Homeostatic regulation.

(Collective phenomena by which neurons alter their intrinsic or synaptic properties to maintain a target level of electrical activity).
Intrinsic plasticity in vivo (courtesy H. Berry)

motor or somatosensory cortex

activity-dependent modifications of the threshold or the slope of the f-I relation

Molecular mechanisms of IP (courtesy H. Berry)

\[ K(Ca) = K_{max} \frac{Ca^n}{Ca^n + K_K^n}, \quad P(Ca) = P_{max} \frac{Ca^n}{Ca^n + K_P^n} \]

\[ \frac{dg_x^*}{dt} = K(Ca)(1 - g_x^*) - P(Ca)g_x^* \]

\[ g_x^*(\infty) = \frac{K(Ca)}{K(Ca) + P(Ca)}; \quad \tau = \frac{1}{K(Ca) + P(Ca)} \]


- activity-dependent plasticity & memory in single molecular module
- activity-dependent time scale
Molecular mechanisms of IP

From Naudé et al., Plos Comp Bio, 2013.

**Figure 3. Inverse gain sensitivities of the standard HH model.**
(A) Inverse gain sensitivity map of the standard HH model, $s_c (mV/s)$. Black line: isoline $x_0 = 0.25$. Colorbar as in (D). (B) Increasing the maximal conductance $g_X$ of the sodium conductance in the standard HH model with $V_{Xh} = -55mV$ and $k_X = 1mV$ increases the gain (decreases the inverse gain) of the $f - I$ (arrow). Blue to red curves: $g_X = [0.1g_{X, sup} - g_{X, sup}]$ with $g_{X, sup} = 4 \times 10^{-2} mS/cm^{-2}$. (C) Increasing the maximal conductance $g_X$ of the sodium conductance in the standard HH model with $V_{Xh} = -62mV$ and $k_X = 1mV$ decreases the gain (increases the inverse gain) of the $f - I$ but this effect is masked by the much larger change of firing frequency due to modification of the threshold. Blue to red curves: $g_X = [0.1g_{X, sup} - g_{X, sup}]$ with $g_{X, sup} = 2.3 \times 10^{-2} mS/cm^{-2}$. (D) Theoretical inverse gain sensitivity map derived from the pre/post-spike IAF theory with sodium conductance. $I = 0.15 nA/cm^{-2}$, $k_C = 2.75$. doi:10.1371/journal.pcbi.1002349.g003
Although generated at a molecular scale, synaptic and intrinsic plasticity induce prominent effects at all scales in the brain (neurons, neural networks, brain area).

However, at the moment, there is no mathematical theory to handle properly these effects.

Let us see some mathematical results and obstructions toward such a theory by studying the SNT.
Dynamical effects of intrinsic plasticity at the network level.

Jeremie Naude, Bruno Cessac, Hugues Berry, and Bruno Delord, "Effects of Cellular Homeostatic Intrinsic Plasticity on Dynamical and Computational Properties of Biological Recurrent Neural Networks"

J. of Neuroscience, 2013.
Dynamical effects of intrinsic plasticity at the network level.

\[
x_i^{(T)}(t+1) = f\left( \sum_j w_{ij} x_j^{(T)}(t) - \theta_i^{(T)} \right)
\]

\[
\theta_i^{(T+1)} = \theta_{max} \left( 4g_i^{*(T+1)} - 1 \right)
\]

\[
g_i^{*(T+1)} = K_i^{(T)} \left( 1 - g_i^{*(T)} \right) - P_i^{(T)} g_i^{*(T)}
\]

\[
K_i^{(T)} = K_{max} \frac{\left( \bar{x}_i^{(T)} \right)^4}{\left( \bar{x}_i^{(T)} \right)^4 + K_K^4}
\]

\[
P_i^{(T)} = P_{max} \frac{\left( \bar{x}_i^{(T)} \right)^4}{\left( \bar{x}_i^{(T)} \right)^4 + K_P^4}
\]
Dynamical effects of intrinsic plasticity

A

\[ \langle x(t) \rangle \]

\[ T \]

0 50 100 150 200 250

0 0.2 0.4 0.6 0.8 1

B

\[ f(u), p(u(t)) \]

\[ u(t) \]

-1 0 1 2 3

0 0.2 0.4 0.6 0.8 1

C

\[ p(x(t)) \]

\[ x(t) \]

0 0.2 0.4 0.6 0.8 1

0.1 0.2 0.3 0.4 0.5

H/IP

no H/IP

H/IP

no H/IP
Dynamical effects of intrinsic plasticity

Dynamic Mean-field theory

Largest Lyapunov exponent computed with the dynamical mean-field approach, as a function of the input amplitude, in the presence (yellow) or absence (red) of HIP.

The neuronal transfer function, (black curve) and its derivative (black dashed curve), as a function of the local field. In the absence of HIP, input fluctuations (red bands) give rise to a contracted output fluctuations. On the contrary, in the presence of HIP, output fluctuations are amplified, i.e. the difference between the input and the neuron’s threshold (yellow bands).

Local field variance at the end of the simulation in the presence (yellow curve) or absence (red curve) of HIP. (B-C)
Combining Hebbian and intrinsic plasticity.
Combining Hebbian and intrinsic plasticity.
Conclusion
Synaptic and Intrinsic plasticity are interesting examples of multi scales dynamics.

We have a qualitative understanding of the conjugated of SP and IP on this model dynamics, but a mathematical theory is still missing.

Missing steps:

Dynamic mean-field theory for correlated weights (the challenge is not to obtain the equations but rather to study them and their links with the finite system dynamics).

Finite size effects.

Developing such a theory could provide methods for the analysis of many other examples of dynamical systems with adaptation and memory.