Epigenesis of the Global Neural Workspace: from local Hebbian to global Reinforcement Learning

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Objectives: The Global Neuronal Workspace hypothesis (GNW) posits a specific cortical architecture of specialised and distributed modules which perform low-level tasks at a non-conscious level, and under defined conditions, information processed by these modules becomes conscious by reaching a global network (the Global Neuronal Workspace) of distant pyramidal cells connected by long range axons. The message is then amplified and broadcasted to many brain regions through an ignition process which defines the subject's conscious access to the piece of information. GNW entails that the development of certain patterns of connectivity between territories of the global workspace is crucial in learning high-level abilities, such as social cognition, and effortful cognitive tasks (Dehaene, Kerszberg, and Changeux, 1998). We present a project of multi-scale model of the GNW through mechanisms of synaptic plasticity combining Hebbian and Reinforcement Learning (Izhikevich, 2007). The overall objective is to capture how synaptic epigenesis by selection/elimination of connections is shaping the GNW through the interaction of a genetic envelope —here embodied by the initial structure at micro-, meso-, and macro-levels—, the spontaneous neural dynamics, and the activity elicited by the interaction with the environment.

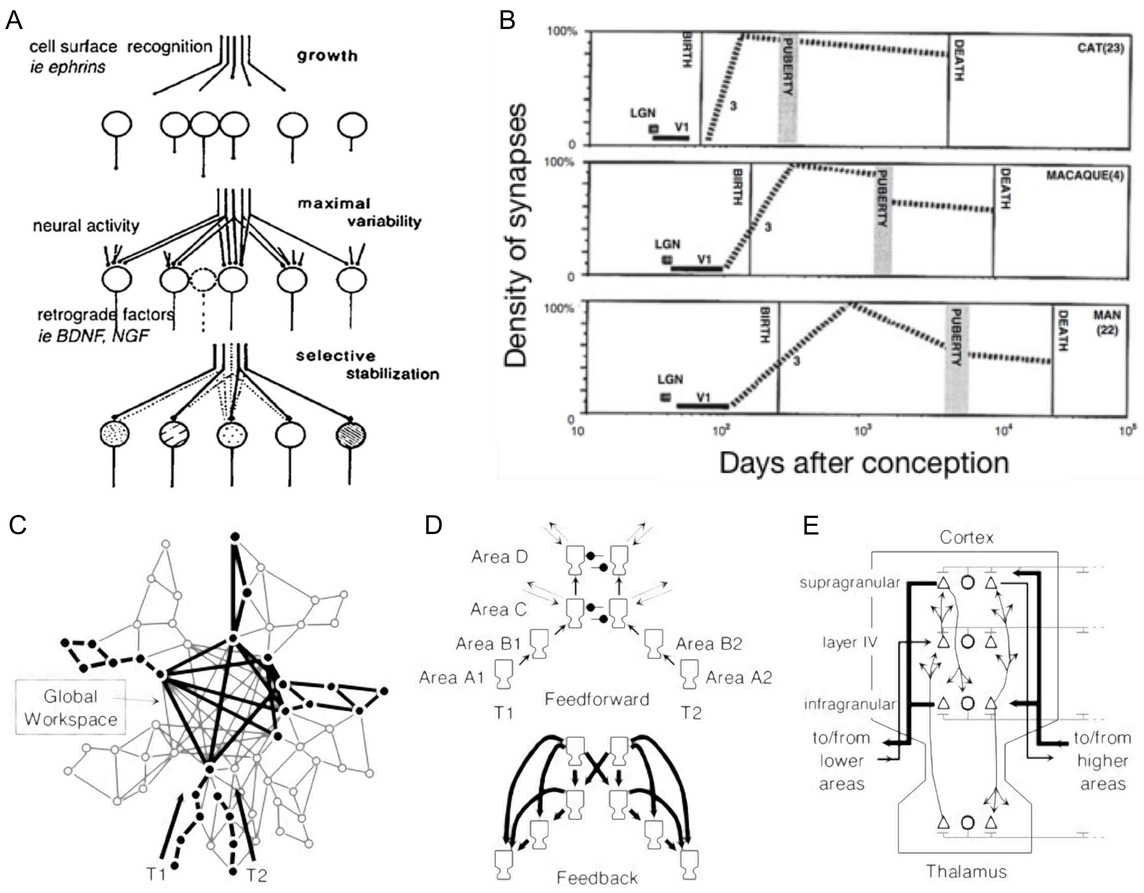

Figure 1: Core concepts of Synaptic Epigenesis & Global Neuronal Workspace model of conscious access. (A) The three core mechanisms of synaptic epigenesis: growth, variability, and stabilization of synaptic connections. (B) Comparison of the evolution of synaptic density in cat, macaque and human. Notice the relative longer period between birth and puberty, but also the massive synaptic pruning in human. (C) Schematic architecture of brain areas (redrawn from ref. 14) comprising multiple specialized processors and a central network of high-level areas temporarily interconnecting them. During the attentional blink, T1 invades the workspace, where areas lock into a single assembly supporting conscious reportability. This invasion by T1 blocks the processing of T2 at a similar depth. (D) Subset of thalamo– cortical columns included in the present simulation. (E) Structure of a single simulated thalamo– cortical column, reproducing the laminar distribution of projections between excitatory neurons (triangles) and inhibitory neurons. (adapted from Changeux et al. 1973; and Dehaene, Sergent, Changeux 2003)

Methods: All the computational models were simulated with the Brian2 python library (Goodman et al. 2008). Current simulations being at the micro- and meso-scales, we were still able to run them on a standard laptop (Core i5 2.4 GHz, RAM 8GB). For the micro-scale, we implement in Brian2 a simple phenomenological model capturing the essence of dopamine modulation of STDP (Izhikevich, 2007). The state of each synapse uses 2 variables: the synaptic strength or weight (s) but also the activation of an enzyme important for plasticity (c; e.g. autophosphorylation of CaMK-II, oxidation of PKC or PKA).

The dynamics is governed by the set of equations:

$$\begin{cases} \dot{c} = -c/\tau_c + \mathrm{STDP}(\tau)\delta(t - t_{\mathrm{pre/post}}) \\ \dot{s} = cd. \end{cases}$$

Here *d* describes the extracellular concentration of dopamine and $\delta(t)$ is the Dirac delta function that step-increases the variable *c*. Firings of pre- and postsynaptic neurons, occurring at times $t_{pre/post}$, respectively, change *c* by the amount STDP. This variable decays to c = 0 exponentially with the time constant τ_c = 1 s. This decay controls the sensitivity of plasticity to delayed reward, *c* acting as the "eligibility trace" for synaptic modification.

For the meso-scale simulation, we used 100 Poisson spiking neurons and selectively reinforced near-coincident firing of both neurons by dopamine release throughout the whole network, with a 1 second delay. We ran the simulation for 3600 seconds, and monitored synaptic eligibility, weight and extracellular dopamine.

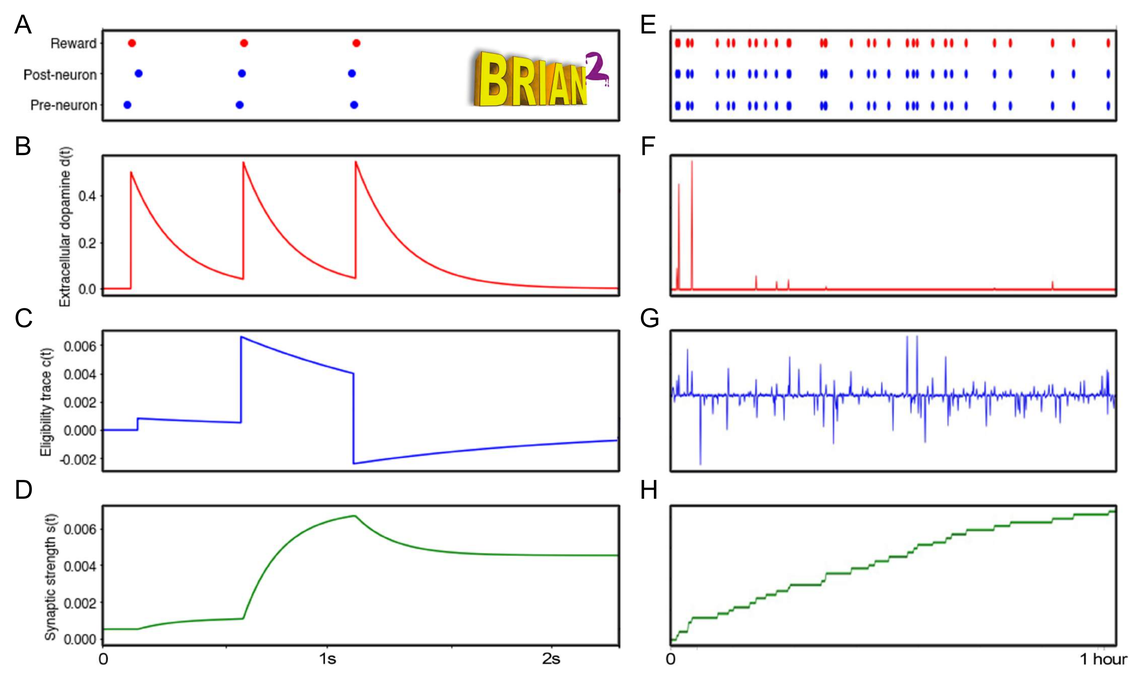

Figure 2: Dopamine modulated STDP synapse. Simulation at both single synapse level (A-D; notice the interaction between dopamine release and spike timing) and network level (E-H).

Results: At the micro-scale level, our implementation of the dopaminergic modulated STDP successfully replicated the original results by Izhikevich (2007), at a single synapse (Figure 2, panels A to D) but also at the network level (Figure 2, panels E, F). At the meso-scale, we indeed obtained an increase in synaptic weight, as predicted by the model. We also successfully implemented the sand box for the XOR task, using Inputs, Core, and Output areas composed of cortical columns (Figure 3).

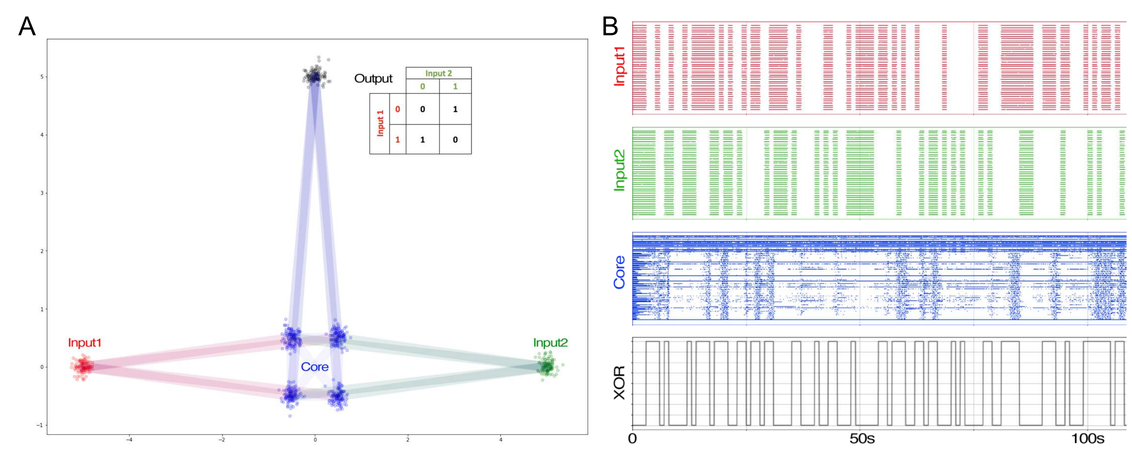

Figure 3: XOR task. (A) Schema of the current model to solve the XOR function. (B) Example of a 2 min simulation.

Perspectives: Next step will be to implement the reward loop of the XOR task by triggering dopamine release in function of the activity of the supra-granular layer in the Output cortical column. We will be able to analyse how performance of the global network is affected by the plasticity at intra-columnar, inter-columnar, and inter-area levels of organization. Then, we will move from XOR to a more complex tasks such as the addition of two digits (with input and output as 8 x 8 columns areas, using MNIST) or binocular reconstruction from stereo-pair images. The long term goal is to go beyond single whole brain modelling to probe the impact of GNW epigenesis in the context of social interaction (Figure 4).

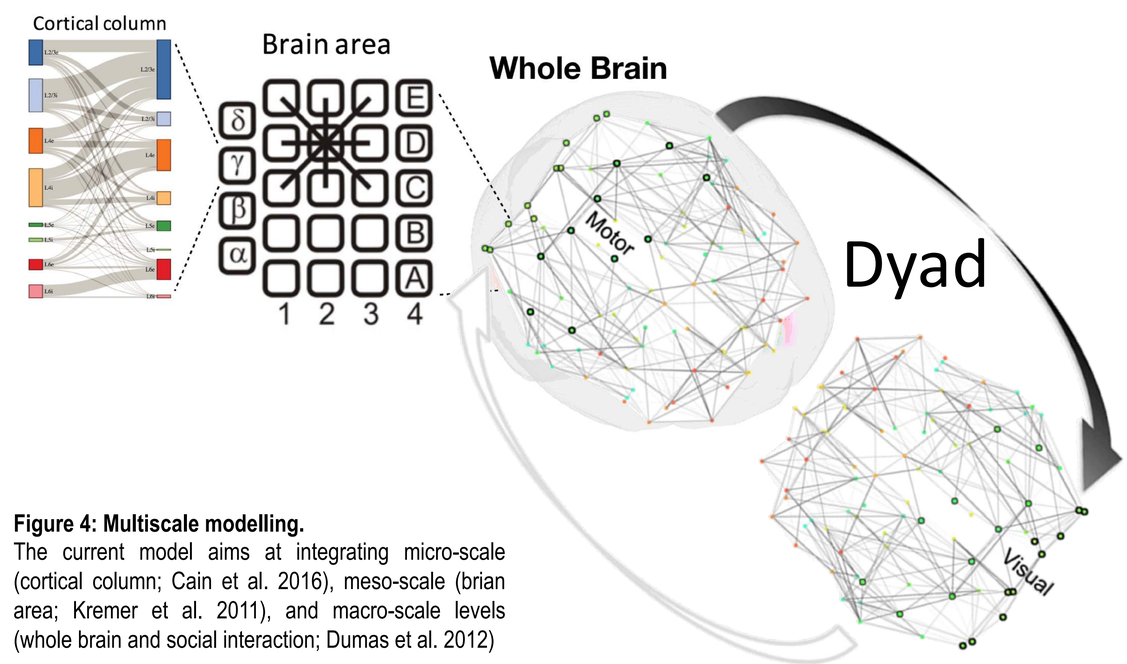

Figure 4: Multiscale modelling.
The current model aims at integrating micro-scale (cortical column; Cain et al. 2016), meso-scale (brian area; Kremer et al. 2011), and macro-scale levels (whole brain and social interaction; Dumas et al. 2012)

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