



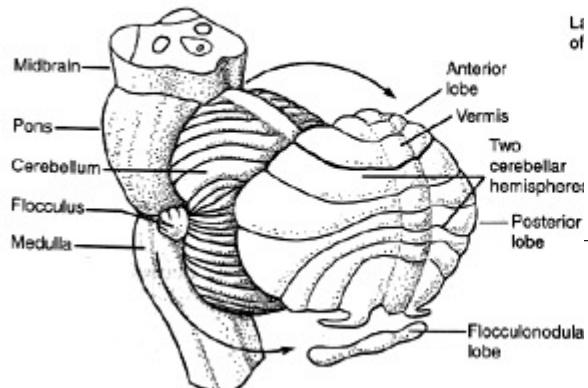
# Neuroengineering 2021/22

COMPUTATIONAL NEUROSCIENCE 1 –  
Part 3- Neural bases of Motor Control – Focus on the  
Cerebellum

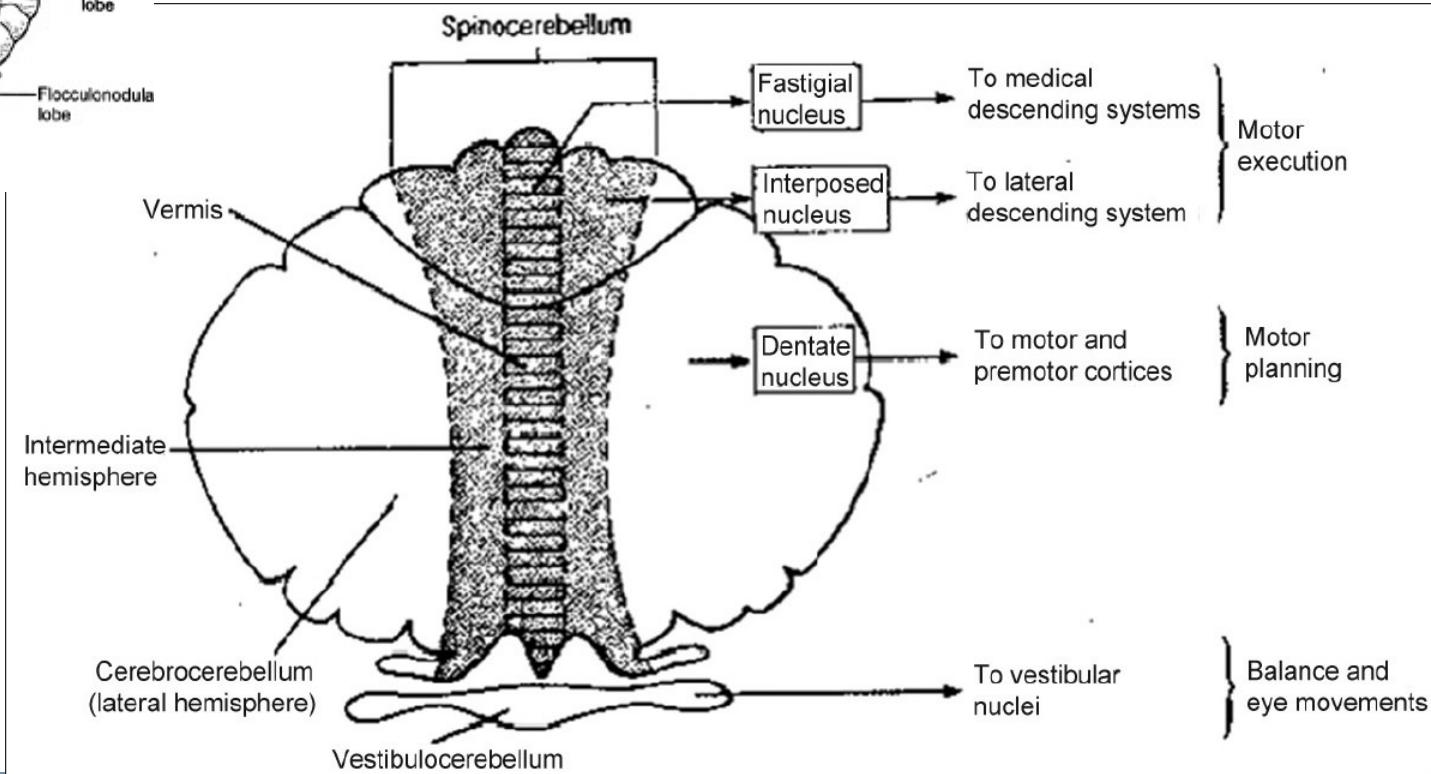
- What does it receive as input? (Inputs=40\*outputs)
  - Info on the objective of motor actions
  - Info on the motor commands
  - Sensorial feedback signals associated to the planning and execution of movements
- What does it produce as output?
  - The output projections of the cerebellum are focused mainly on the premotor and motor systems of the cerebral cortex and brain stem, systems that control spinal interneurons, and motor neurons directly
- It has the property of modulation of the input/output connections (adaptation and motor learning: synaptic plasticity)
- The cerebellum makes up only about 10 percent of the mass of the human brain but contains more than half of its neurons. Stretched out, its surface area would be nearly 80% that of the cerebral cortex.

# Anatomical structure

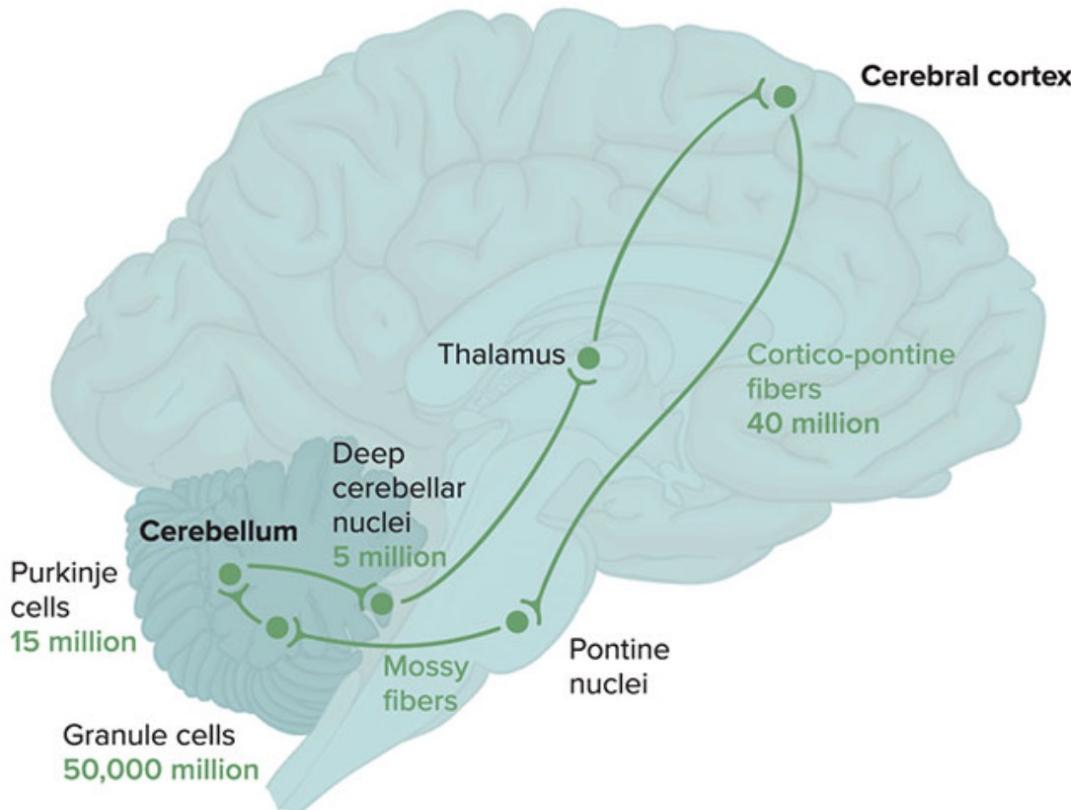
A



B



## The cerebellum-cerebral cortex loop



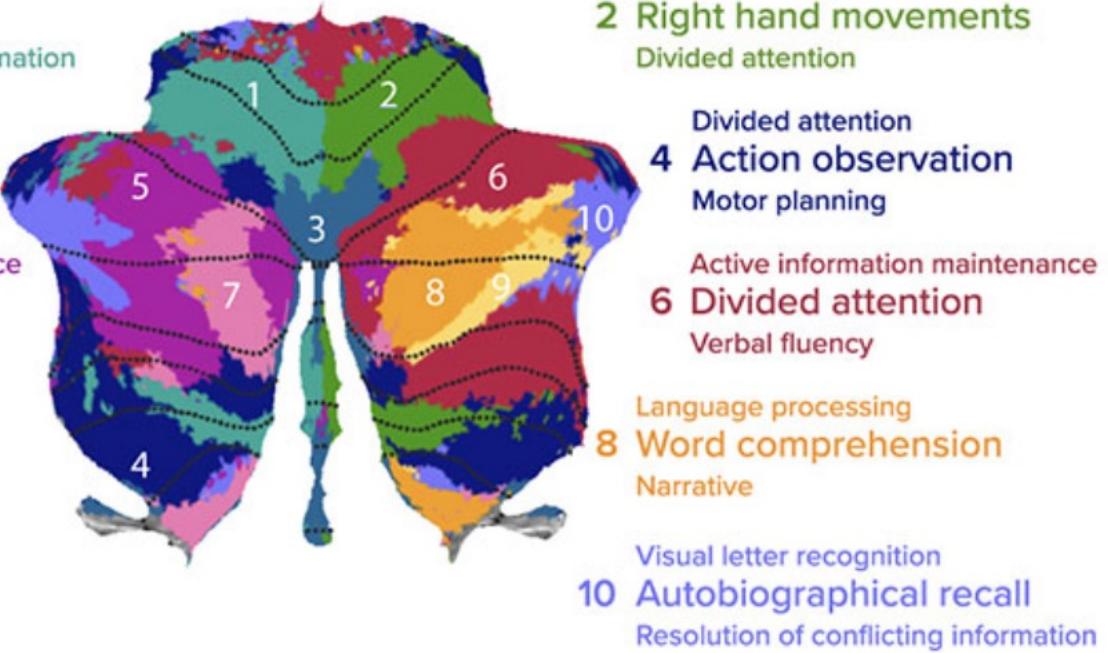
SOURCE: J. DIEDRICHSEN ET AL / NEURON 2019

KNOWABLE MAGAZINE

While the cerebral cortex's role in cognition has long been recognized, the cerebellum's role has largely been ignored. But these two parts of the brain share an elaborate network of connections. Some 40 million neurons in the cerebral cortex have fiber-like axons extending to the pontine nuclei in the brainstem, an area intimately connected to the cerebellum. Extensive connections go in the other direction, too, from the cerebellum up to the cortex.

# Functions of the cerebellum

- Motor planning
- 1 Left hand movements**
  - Resolution of conflicting information
- Visual working memory
- 3 Rapid eye movements**
  - Visual letter recognition
- Active information maintenance
- 5 Divided attention**
  - Mental arithmetic
- Emotion processing
- 7 Narrative**
  - Language processing
- Word comprehension
- 9 Verbal fluency**
  - Mental arithmetic



SOURCE: M. KING ET AL / NATURE NEUROSCIENCE 2019

KNOWABLE MAGAZINE

The cerebellum plays a role in several motor and cognitive tasks. Neuroscientists used functional magnetic resonance imaging to scan people's brains while they carried out a wide variety of activities. The researchers then mapped key functions associated with different regions of the cerebellum. Stronger associations are indicated by larger text in this flattened version of the cerebellum's surface.

<https://www.brainfacts.org/brain-anatomy-and-function/anatomy/2020/the-mysterious-multifaceted-cerebellum-120320>

# Effects of cerebellar removal

## CONTROL OF LOCOMOTION

Reticulo spinal pathway

After cerebellum removal

Vestibulospinal pathway

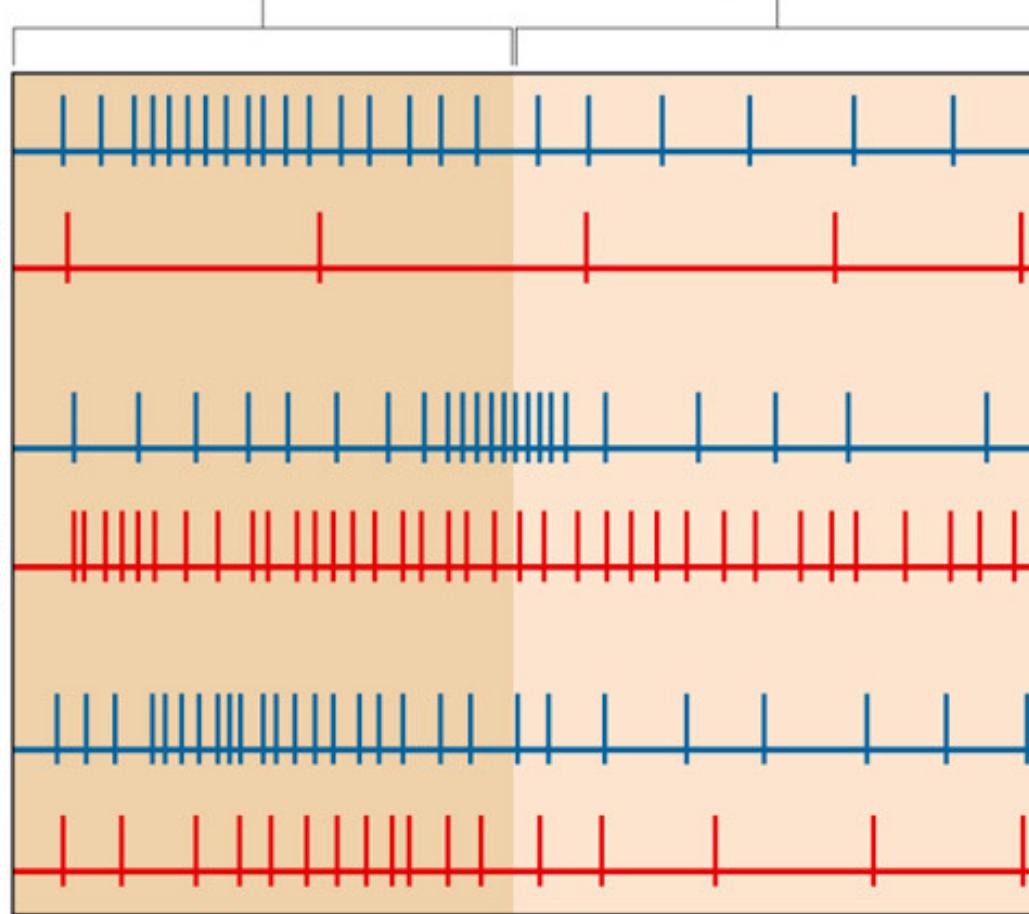
After cerebellum removal

Rubrospinal pathway

After cerebellum removal

SWING

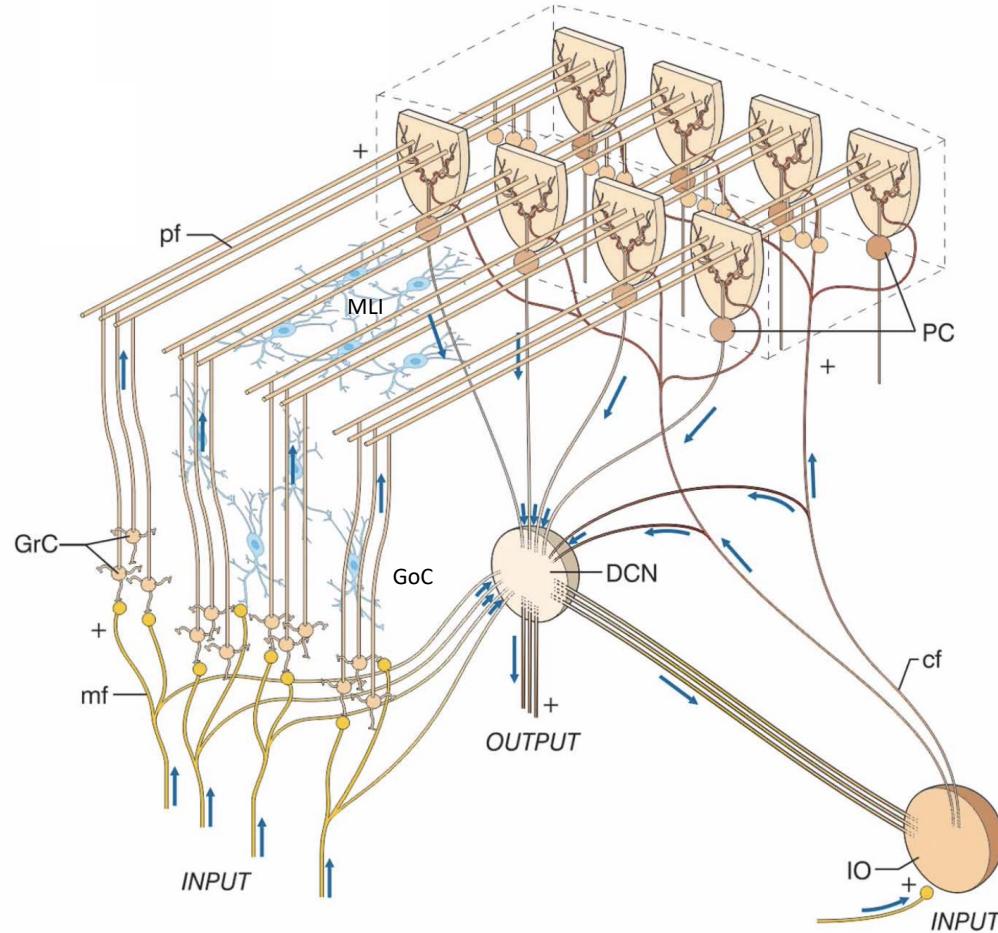
STANCE



Ooba © 2006 edi.ermes milano

# Cerebellar microcircuits

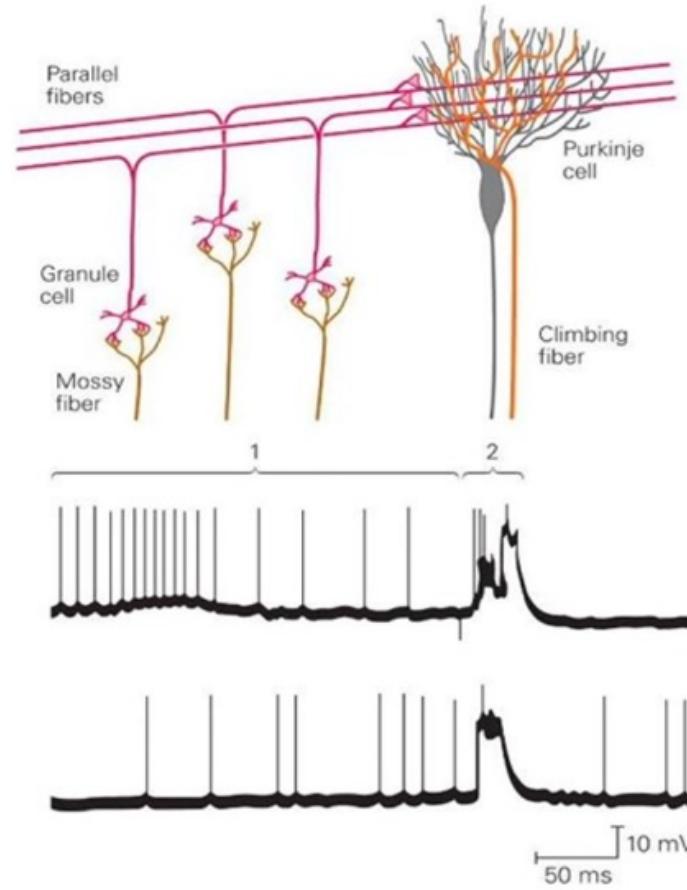
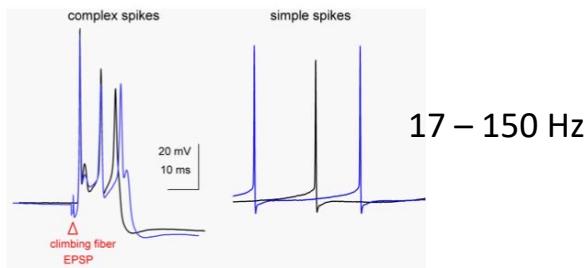
Mossy Fibers (mf)  
Granule cells (GrC),  
parallel fibers (pf)  
Golgi cells (GoC),  
Molecular Interneurons (MLI)  
Stellate cells(SC)  
Basket cells (BC).  
Inferior Olive cells (IO-C)  
Purkinje cells (PC),  
Deep Cerebellar Nuclei cells (DCN-C)



[Ito 2006]

# Electrical Activity of Purkinje Cells

1-3 Hz

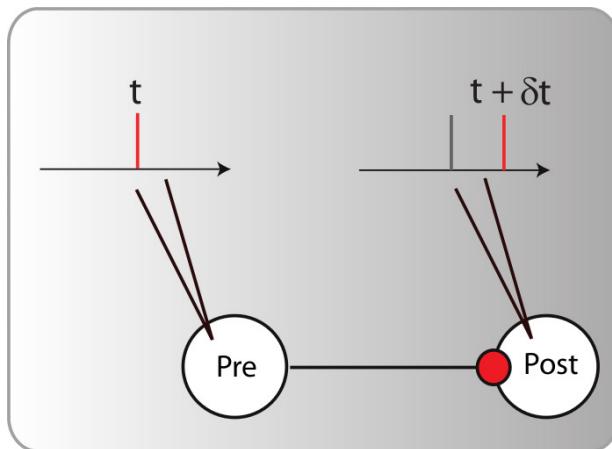


# Hebbian plasticity: Spike Timing Dependent Plasticity (STDP)

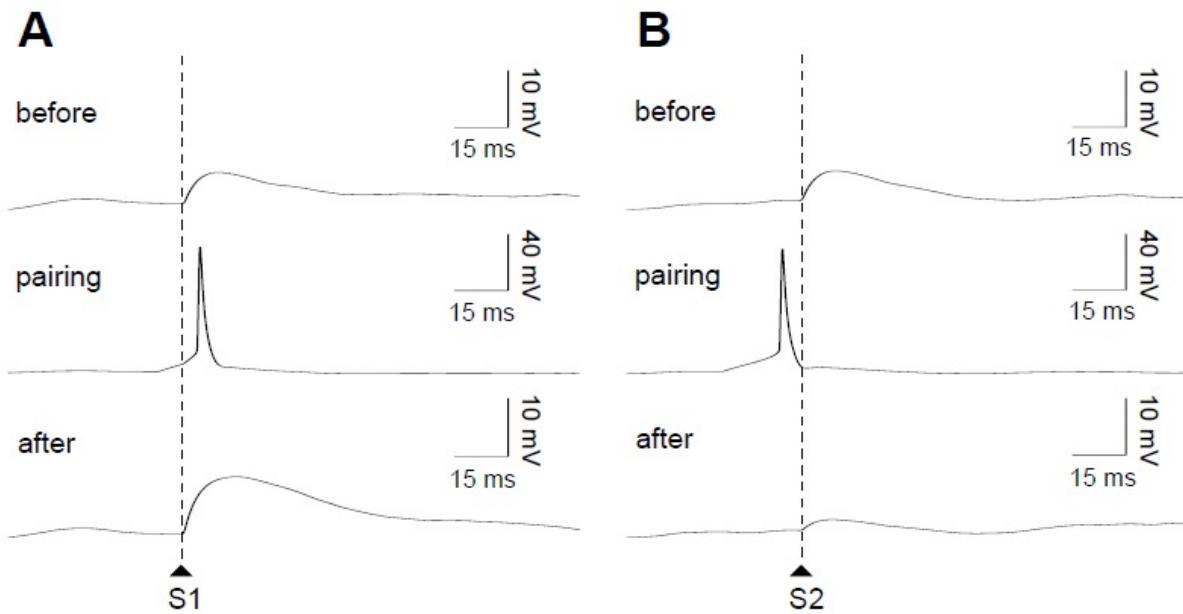
Hebb hypothesis 1949

Experimental proofs:

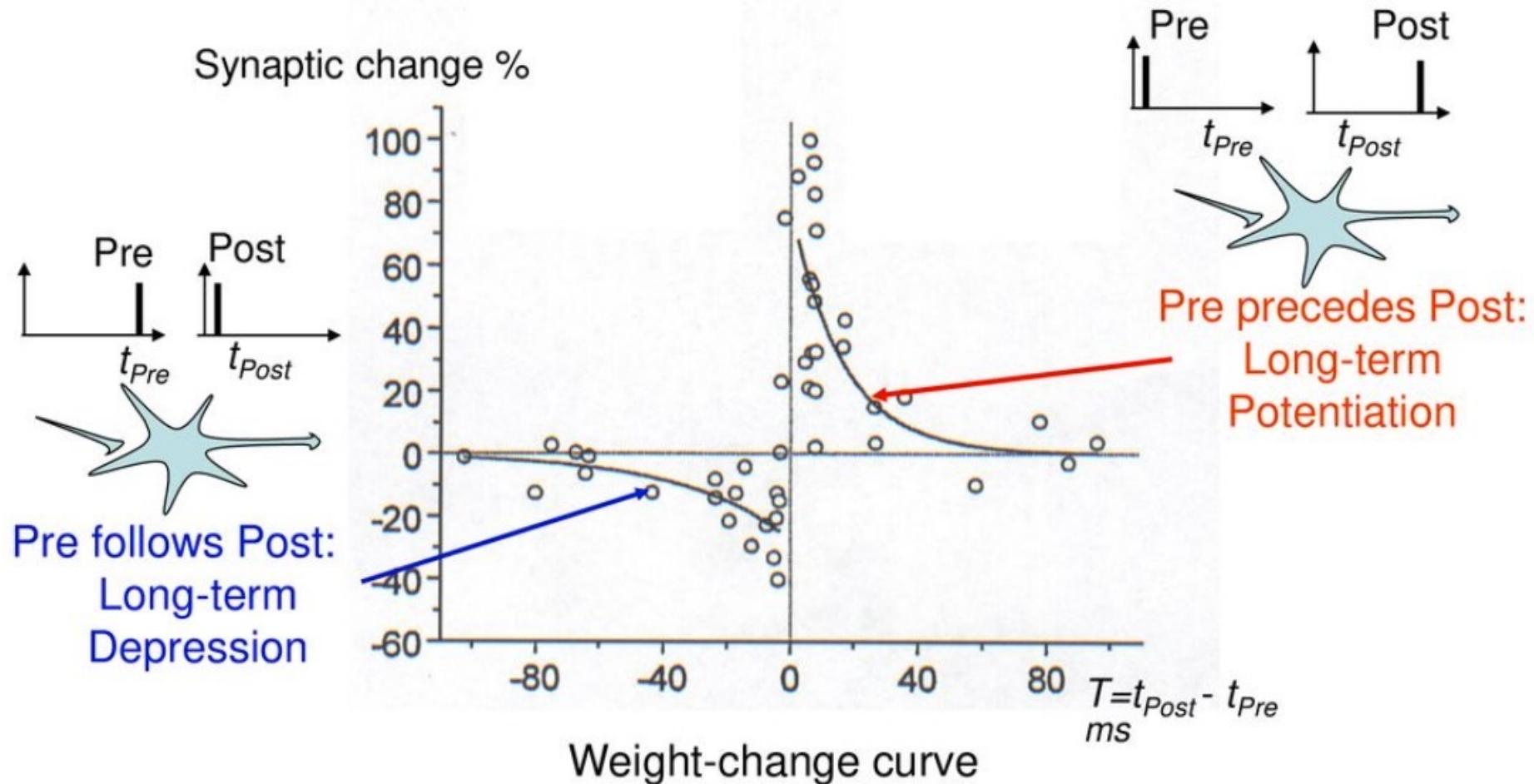
Bi 1998, Markram 1997,  
Gerstner 1996



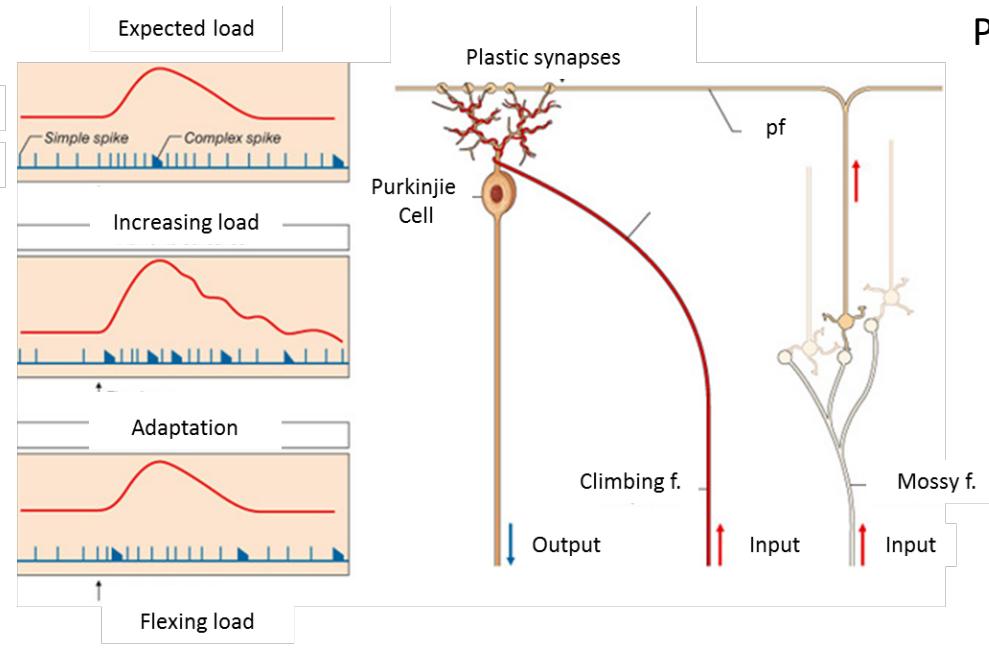
Experimental protocol of Spike Timing Dependent Plasticity in vitro. Pre and Post synaptic neurons are patched and forced to fire with a time difference, while the modification of the synaptic strength is monitored



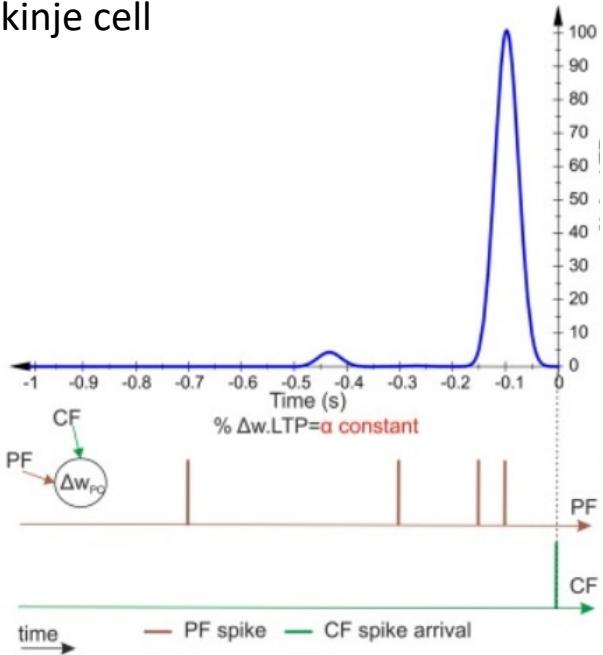
# STDP



# Cerebellum output



Synaptic plasticity at parallel fiber–Purkinje cell



Changes in the strengths of parallel fiber–Purkinje cell synapses could store stimulus-response associations by linking inputs with appropriate motor outputs, following a **Hebbian learning** approach but **with supervision of Cf discharge**.

# Summary

Structure and organization of the brain suggest computational analogies:

- INFORMATION STORAGE: Physical and chemical structure of neurons and synapses
- INFORMATION TRANSMISSION: electrical and chemical signaling
- PRIMARY COMPUTING ELEMENT: the neuron
- COMPUTATIONAL BASIS: still unknown!!!

# Summary

- What is motor control and motor learning and high level models of motor control by the Human Brain
- The human brain areas involved in motor control
- Focus on Cerebellum
- The physiology of the cerebellum accounting for its functional features

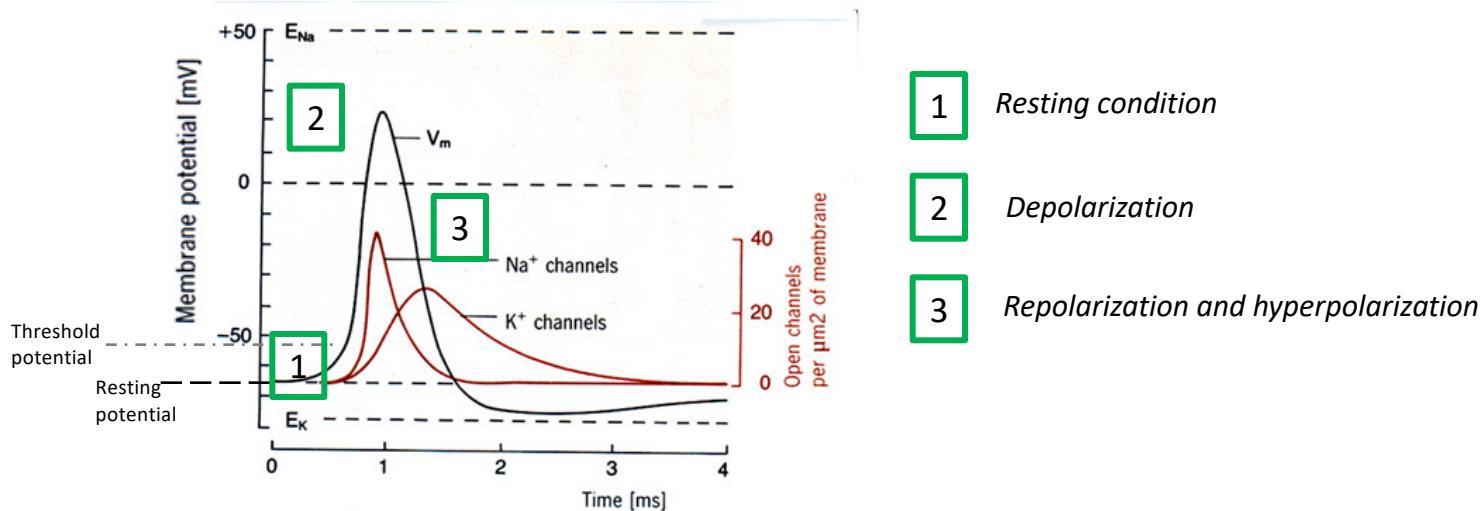


# Neuroengineering 2021/22

COMPUTATIONAL NEUROSCIENCE - Part 4 Modelling and Simulations

# Neuronal communication: the Action Potential

Neurons communicate through Action Potentials (AP) = change in membrane voltage depending on subcellular ion channel-mediated mechanisms

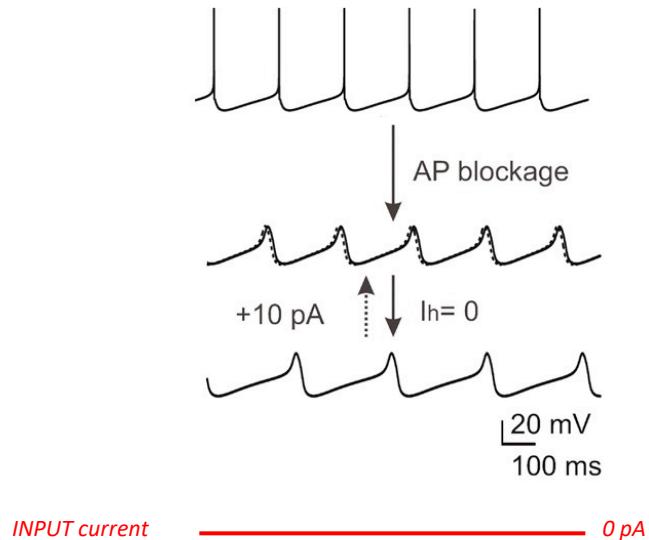


AP can be approximated with SPIKES, the basic units of neuronal coding

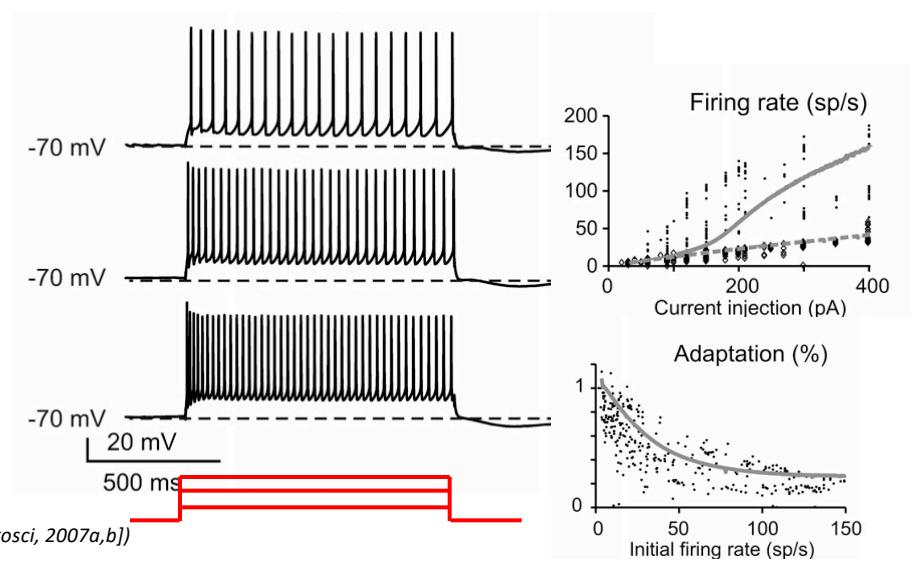
# Neuronal communication: single neuron dynamics

Neurons can exhibit different **spiking** patterns (**electroresponsive properties**):

- Autorhythm = spontaneous firing of neurons due to neurons' intrinsic electro-responsiveness
- SubThreshold Oscillations (STO) = sinusoidal oscillations of the membrane potential around the threshold potential value
- Depolarization induced bursting = increased firing rate of neurons following the starting of a depolarizing external stimulation
- Linear current-frequency relationship
- Spike-Frequency Adaptation (SFA) = Decrease in the neuron's firing rate when stimulated with a constant input.



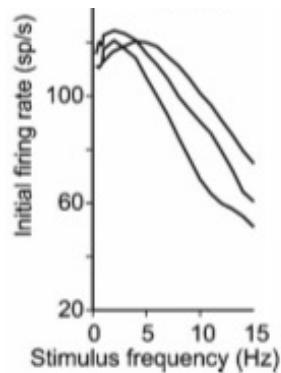
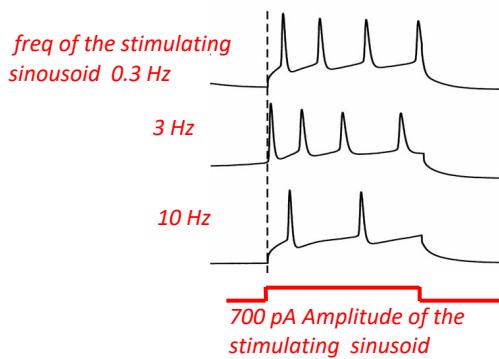
Membrane voltage of a cerebellar Golgi cell (adapted from [Solinas et al, Front Cell Neurosci, 2007a,b])



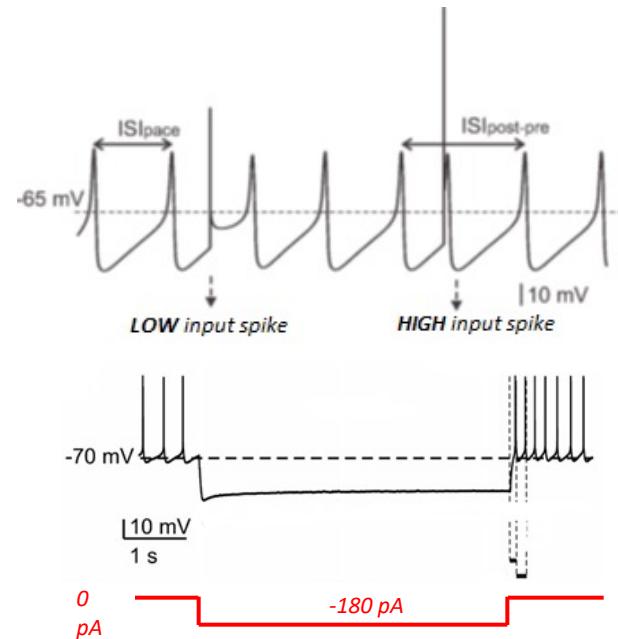
# Neuronal communication: single neuron dynamics

Neurons can exhibit different **spiking** patterns  
(electroresponsive properties):

- Phase reset
- Post-inhibitory rebound burst = increased firing rate following the end of a negative hyperpolarizing input (amplitude and duration of the negative input are fundamental to cause the rebound burst)
- Resonance = maximum firing response of a neuron at a preferred frequency of the input current stimulus



Initial firing rate is the inverse of the ISI between the first two spikes after the stimulus



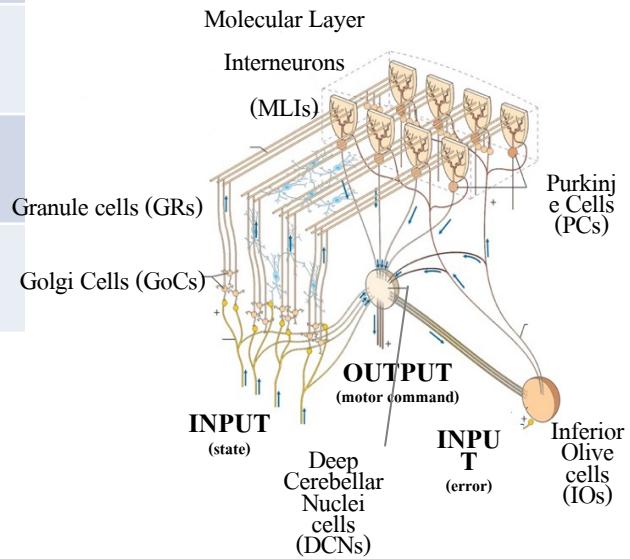
**FUNDAMENTAL** for:

- ✓ Generating network dynamics
- ✓ Noise filtering
- ✓ Plasticity enhancement
- ✓ Communication within and among brain areas

# Cerebellar single neuron dynamics

<u>CEREBELLAR CELLS</u>	Auto-rhythm	Sub-Threshold Oscillations	Depolarization-induced burst	SFA	Phase reset	Post-inhibitory rebound burst	Resonance
Golgi Cell	✓ 5-15 Hz	✓	✓	✓	✓	✓	✓ (θ band)
Granule Cell	-	✓	-	-	-	-	✓ θ band)
Purkinje Cell	✓ 40-80 Hz	-	✓	-	-	-	-
Molecular Layer Interneurons	✓ 10-20 Hz	-	-	-	-	-	-
Deep Cerebellar Nuclei	✓ 10-30 Hz	-	✓	✓	-	✓	-
Inferior Olive cells	-	✓ 1-4 Hz	n.a.	n.a.	✓	-	-

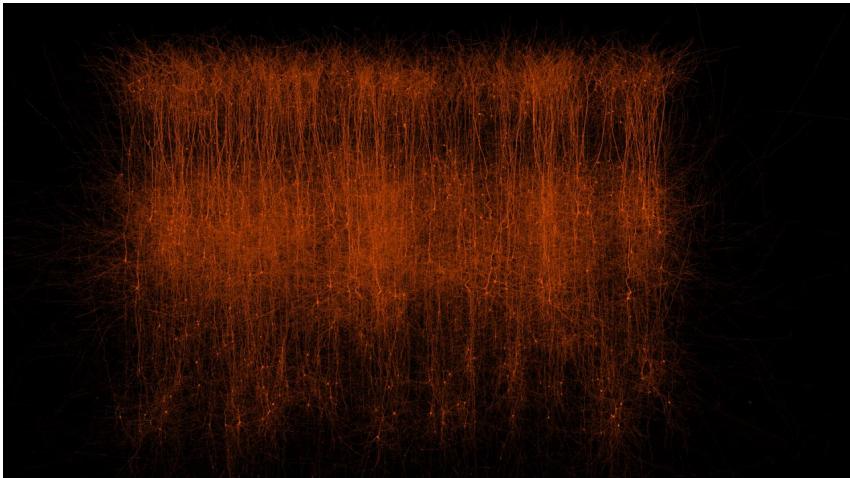
θ band = 1-4 Hz; fundamental for brain oscillations and communication with the cerebral cortex



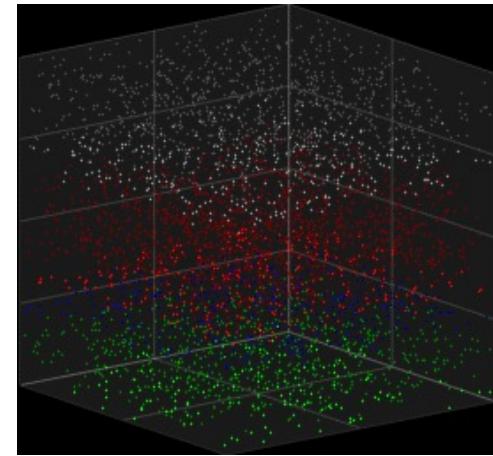
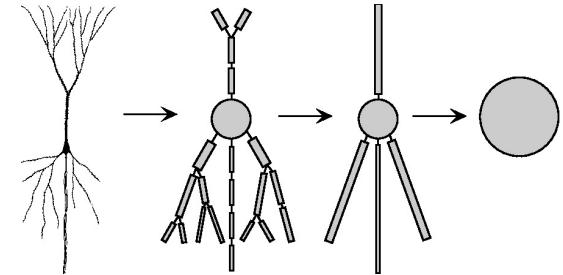
# Modelling single neurons

Different levels of morphological detail: from multi-compartment to point neuron models

**Multi-compartment** neuron models describe the activity of each neuron element (dendrites, axons, ...) taking into account morphological features. Example from the neo-cortex microcircuit [Markram et al., *Cell Reports*, 2015]:



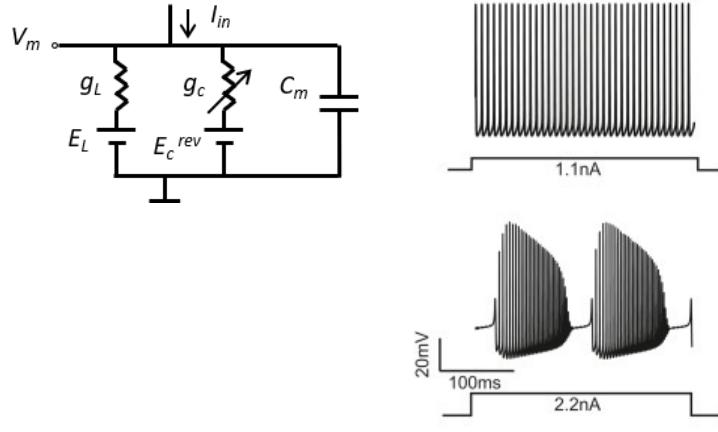
**Point** neuron models describe the activity of neurons as collapsed in a single point, neglecting compartment differences and morphological features. They represent more the computational properties of neurons, than the electrical activity and its spatial distribution



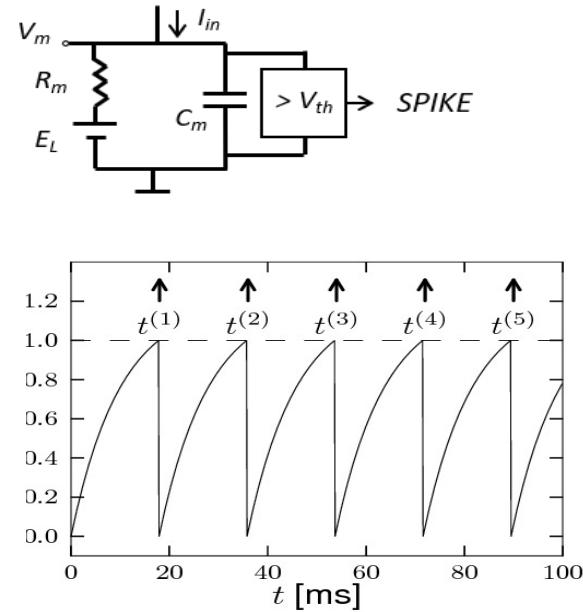
# Modelling single neurons

Different levels of electrical detail: Hodgkin-Huxley (HH) and Leaky Integrate-and-Fire (LIF)

HH: membrane potential  $V_m$  computed considering the resting potential ( $E_L$ ) and the contribution of each membrane **ion channel** (represented by the conductance  $g_c$  and reversal potential  $E_c^{rev}$ ).



LIF: Only **passive** membrane properties are considered (capacitance  $C_m$  and resistance  $R_m$ ). The output is a spike train, corresponding to time instants of threshold overcoming



# Modelling the neuron: HH Compartmental models

The above HH description is for a single compartment. If one wants to describes propagation of the spike in the axon, one has to couple the compartments like we did in the cable equation. The equation holds for each compartment in the axon.

## HIGH PERFORMING COMPUTERS (CINECA)

Stefano Masoli, Sergio Solinas, and Egidio D'Angelo

Front Cell Neurosci. 2015; 9: 47.

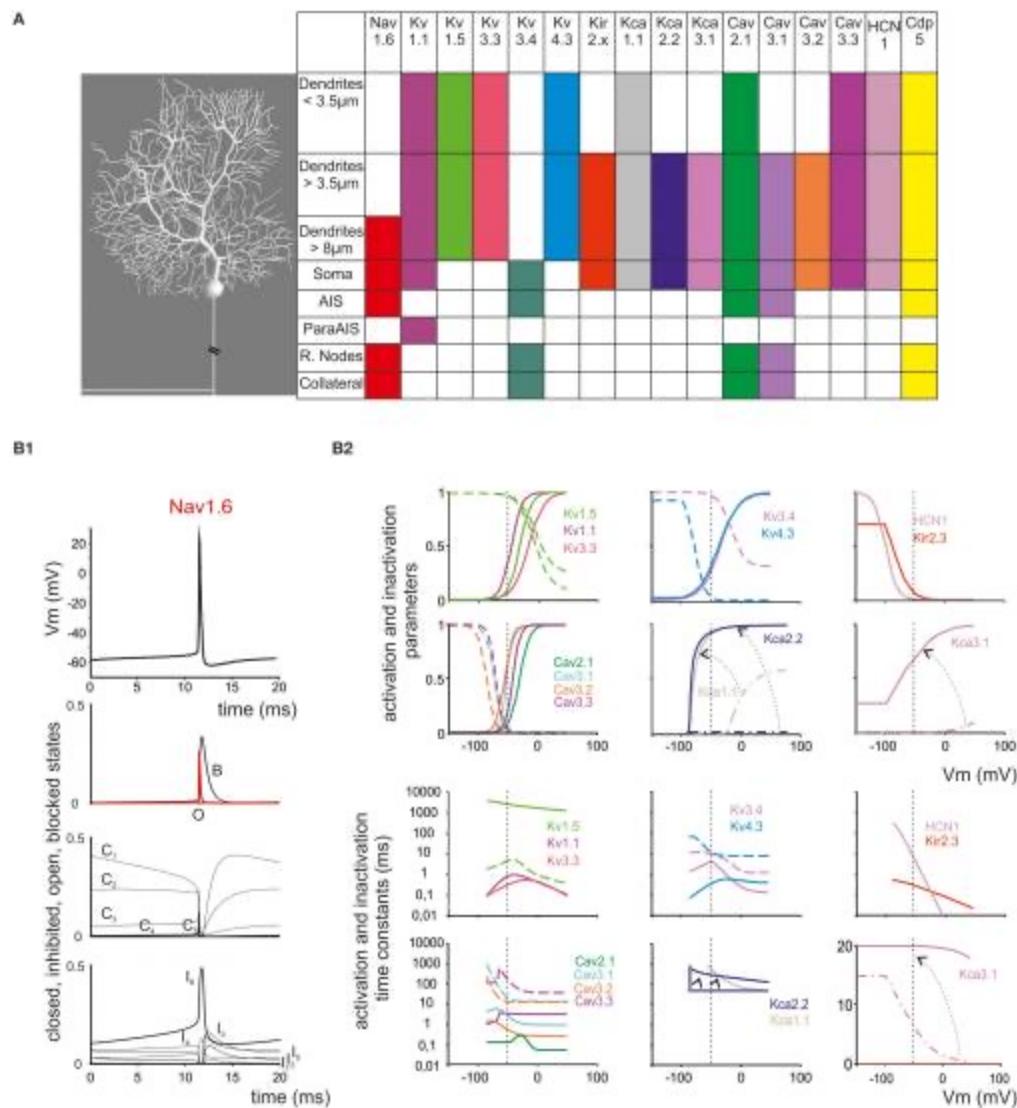
In each compartment, membrane voltage was obtained as the time integral of the equation (Yamada, [1989](#)):

$$\frac{dV}{dt} = -\frac{1}{C_m} * \left\{ \sum [g_i * (V - V_i)] + i_{inj} \right\}$$

Adjacent compartments communicated through an internal coupling resistance (Diwakar et al., [2009](#)).

# Modelling the Purkinje Cell (PC): HH Compartmental models

Section name	Diameter ( $\mu\text{m}$ )	Length ( $\mu\text{m}$ )	Nº sections
Dendrites	0.67–9.22	1–10	1599
Soma	29.8	29.8	1
AIS Axon Initial Segment	0.97	17	1
ParaAIS	0.97	4	1
Myelin	0.73	100	4
Ranvier Nodes	0.73	4	3
Collateral	0.6	100	2



**Table 1 -Electrotonic compartments in the PC model.**

The table shows the sections of the PC model along with their number, diameter and length.

# Modelling PC: HH Compartmental models

Table 2

## Ionic mechanisms in the PC model: I.

The table shows ionic channel localization, maximum conductance and reversal potential. Gating equations were written either in Hodgkin-Huxley (HH) style or Markovian style according to indicated references.

Conductance/Location	Gmax ( $S/cm^2$ )	Erev (mV)	Description of channel (H.H or Markovian)	References
<b>Na CHANNEL</b>				
Nav1.6	Dendrites	0.016	60	Markovian 13 states
	Soma	0.214		Raman and Bean, <a href="#">2001</a>
	AIS	0.5		
	Nodes	0.03		
	Collateral	0.03		

# Modelling PC: HH Compartmental models

Conductance/Location	Gmax (S/cm <sup>2</sup> )	Erev (mV)	Description of channel (H.H or Markovian)	References
<b>K CHANNELS</b>				
Kv1.1	Dendrites	0.0012	-88	HH Akemann and Knopfel, <a href="#">2006</a>
	Soma	0.002		
	ParaAIS	0.01		
Kv1.5	Dendrites	$1.3 \cdot 10^{-4}$	-88	HH Courtemanche et al., <a href="#">1998</a>
Kv3.3	Dendrites	0.01	-88	HH Akemann and Knopfel, <a href="#">2006</a>
Kv3.4	Soma	0.05	-88	HH Raman and Bean, <a href="#">2001</a> ; Khalil et al., <a href="#">2003</a>
	AIS	0.01		
	Nodes	0.01		
	Collateral	0.02		
Kv4.3	Dendrites	0.001	-88	HH Diwakar et al., <a href="#">2009</a>
Kir2.x	Dendrites	0.00001	-88	HH Diwakar et al., <a href="#">2009</a>
	Soma	0.00003		

# Modelling PC: HH Compartmental models

Conductance/Location		Gmax (S/cm <sup>2</sup> )	Erev (mV)	Description of channel (H.H or Markovian)	References
<b>Ca DEPENDENT K CHANNELS</b>					
Kca1.1	Dendrites	$3.5 \times 10^{-2}$	-88	Markovian	Anwar et al., <a href="#">2010</a>
	Soma	0.01			
Kca2.2	Dendrites	$1 \times 10^{-3}$	-88	Markovian	Solinas et al., <a href="#">2007a,b</a>
	Soma	$1 \times 10^{-3}$			
Kca3.1	Dendrites	0.002	-88	HH	Rubin and Cleland, <a href="#">2006</a>
	Soma	0.01			
<b>Ca CHANNELS</b>					
Cav2.1	Dendrites	$1 \times 10^{-3}$	137.5	HH	Swensen and Bean, <a href="#">2005</a> ; Anwar et al., <a href="#">2010</a>
	Soma	$2.2 \times 10^{-4}$			
	AIS	$2.2 \times 10^{-4}$			
	Nodes	$2.2 \times 10^{-4}$			
	Collateral	$2.2 \times 10^{-4}$			

# Modelling PC: HH Compartmental models

Conductance/Location Ca CHANNELS		Gmax (S/cm <sup>2</sup> )	Erev (mV)	Description of channel (H.H or Markovian)	References
Cav2.1	Dendrites	$1*10^{-3}$	137.5	HH	Swensen and Bean, <a href="#">2005</a> ; Anwar et al., <a href="#">2010</a>
	Soma	$2.2*10^{-4}$			
	AIS	$2.2*10^{-4}$			
	Nodes	$2.2*10^{-4}$			
	Collateral	$2.2*10^{-4}$			
Cav3.1	Dendrites	$5*10^{-6}$	137.5	HH	Anwar et al., <a href="#">2010</a>
	Soma	$7*10^{-6}$			
	AIS	$1*10^{-5}$			
	Nodes	$1*10^{-5}$			
	Collateral	$1*10^{-5}$			
Cav3.2	Dendrites	0.0012	137.5	HH	Huguenard and McCormick, <a href="#">1992</a>
	Soma	0.0008			
Cav3.3	Dendrites	0.0001	137.5	HH	Xu and Clancy, <a href="#">2008</a>
	Soma	0.0001			

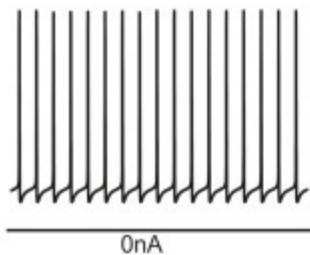
# Modelling PC: HH Compartmental models

Conductance/Location	Gmax (S/cm <sup>2</sup> )	Erev (mV)	Description of channel (H.H or Markovian)	References
<b>MIXED CATIONIC CHANNEL</b>				
HCN1	Dendrites	0.000004	-34.4	HH Angelo et al., <a href="#">2007</a> ; Larkum et al., <a href="#">2009</a>
	Soma	0.0004		
<b>CALCIUM BUFFER—PUMPS DENSITY</b>				
Ca Buffer	Dendrites	$2 \cdot 10^{-8}$	Markovian	Anwar et al., <a href="#">2010</a>
	Soma	$5 \cdot 10^{-8}$		
	AIS	$5 \cdot 10^{-8}$		
	Nodes	$5 \cdot 10^{-7}$		
	Collateral	$5 \cdot 10^{-8}$		

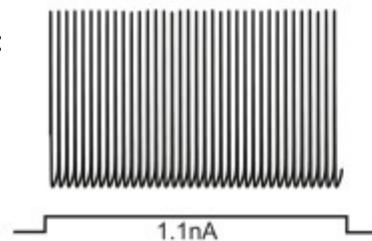
# Modelling PC: HH Compartmental models

A

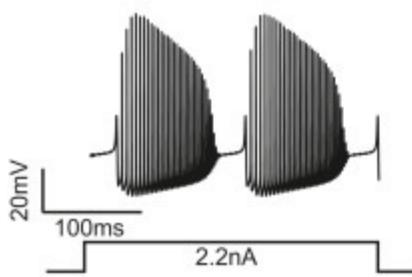
Auto-rhythmic properties no current injection



Simple spikes: low somatic current injection

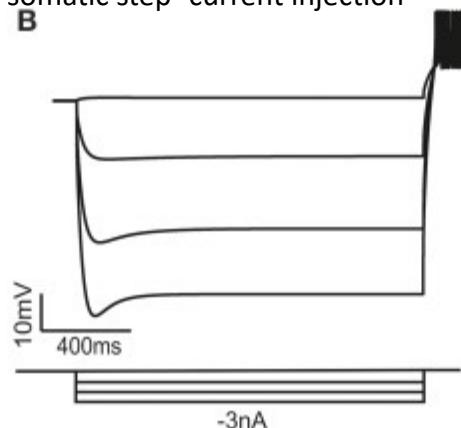


Complex spikes: high somatic current injection



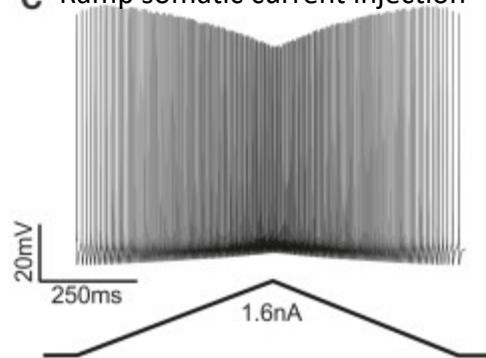
sag and rebound depolarization: Negative somatic step -current injection

B



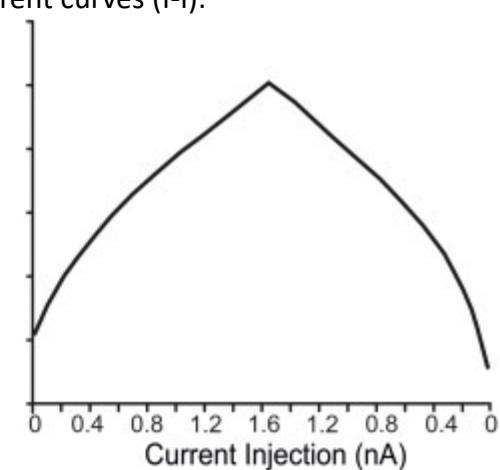
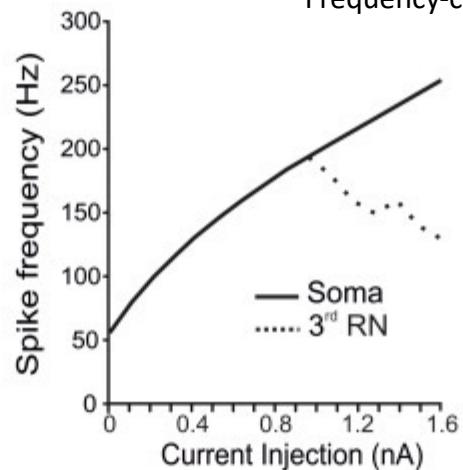
Frequency modulation:

C Ramp somatic current injection



D

Frequency-current curves (f-I):



ramp current induces a linear frequency modulation of Soma (up to about 300Hz while RNs are unable to sustain firing frequencies above 200 Hz)

up and down ramp current non perfectly symmetrical

# Modelling single neurons: the Leaky Integrate and Fire neuron

In the Leaky Integrate-and-Fire (LIF) neuron, the subthreshold dynamics of the membrane potential is modelled through a single passive term:

Membrane potential dynamics

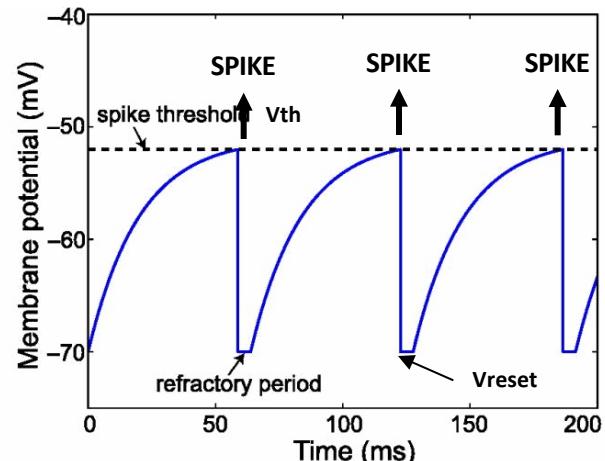
$$\rightarrow \tau_m \frac{dV_m(t)}{dt} = -(V_m(t) - E_L) + R_m \cdot I_{in}(t)$$

Spike condition

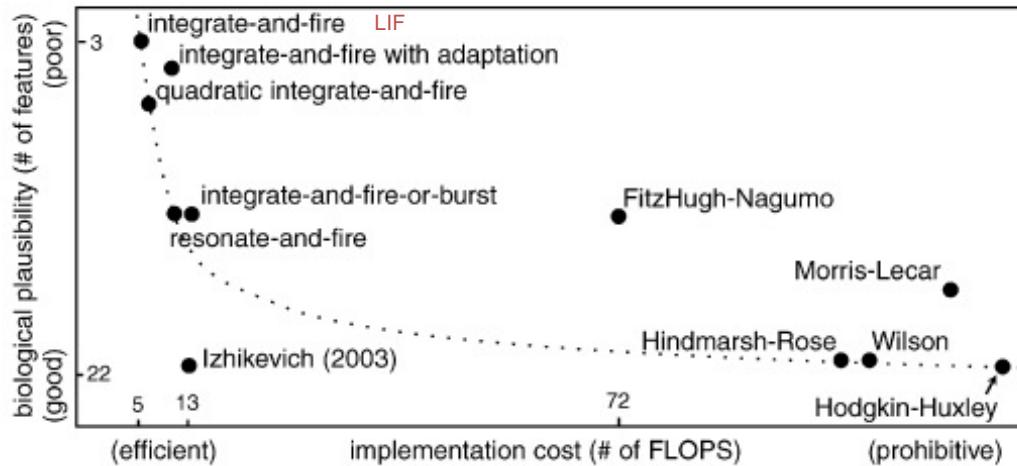
$$\rightarrow \text{If } V_m > V_{th}, \text{ then } V_m = V_{reset}$$

- $\tau_m$  is the membrane time constant ( $\tau_m = R_m \cdot C_m$ , where  $R_m$  and  $C_m$  are the membrane resistance and capacitance, respectively), and it accounts for how fast the  $V_m$  curve increases
- $E_L$  is the resting potential and it represents the steady-state value of  $V_m$  in absence of external input current
- $I_{in}$  is the input current.

Action potentials are approximated as **single spike instants**: whenever  $V_m$  reaches a firing threshold  $V_{th}$ , the membrane potential is reset to a fixed value  $V_{reset}$ . After the spike,  $V_m$  remains at  $V_{reset}$  value (constant) during the refractory period and it is not possible to emit spikes.



# Modelling single neurons: biological plausibility vs computational load



[Izhikevich, *IEEE Trans Neural Networks*, 2003]

Compromise between biological plausibility and implementation cost:

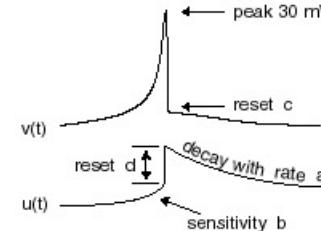
- HH multi-compartment models with morphology representation ✓ biological plausibility ✗ computational load
- LIF point neuron models ✗ biological plausibility ✓ computational load
- Multi-dimensional LIF models:
  - Izhikevich (non linear)
  - Adaptive Exponential Leaky Integrate and Fire (LIF) model (non linear)
  - Generalized LIF (linear)
- Fitting with experimental traces for OPTIMIZATION – also optimization has a cost!

# Multi-dimensional LIF models: Izhikevich neuron

$$\begin{cases} \frac{dV_m(t)}{dt} = 0.04 * V_m^2(t) + 5 * V_m] + 150 - u(t) + 1 \\ \frac{du(t)}{dt} = a * b(V_m(t) - u(t)) \end{cases}$$

**Membrane Potential**

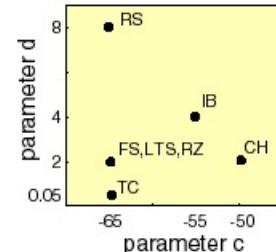
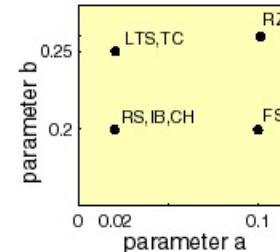
**Membrane Recovery variable  $u(t)$**



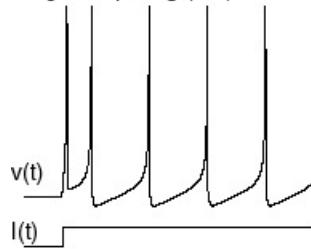
If  $V(t) > 30mV \rightarrow$  Spikes.

$$\begin{cases} V_m(t+1) = c \\ u(t+1) = u(t) + d \end{cases}$$

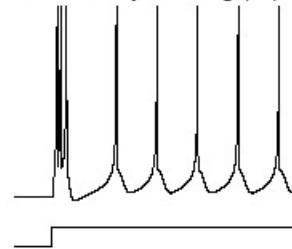
$v' = 0.04v^2 + 5v + 140 - u + 1$   
 $u' = a(bv - u)$   
**if**  $v = 30$  mV,  
**then**  $v \leftarrow c$ ,  $u \leftarrow u + d$



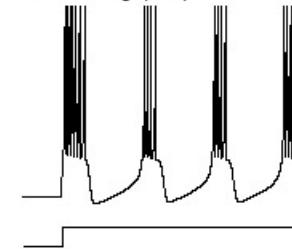
regular spiking (RS)



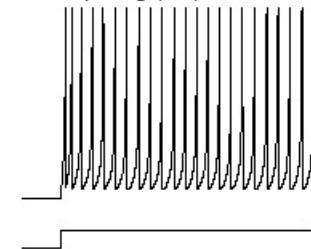
intrinsically bursting (IB)



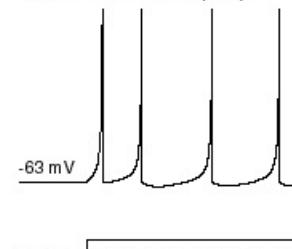
chattering (CH)



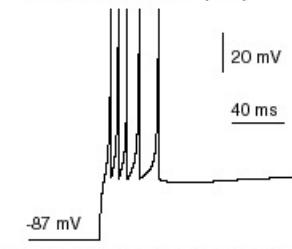
fast spiking (FS)



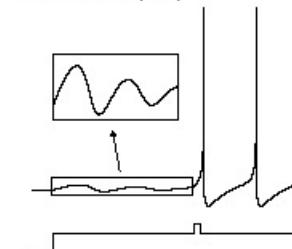
thalamo-cortical (TC)



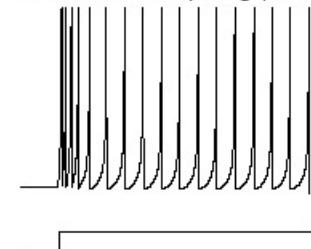
thalamo-cortical (TC)



resonator (RZ)



low-threshold spiking (LTS)



[Izhikevich, IEEE Trans Neural Networks, 2003]

# Multi-dimensional LIF models: Adaptive Exponential LIF neuron

The model:

$$\begin{cases} C_m \cdot V_m'(t) = -g_L \cdot (V_m(t) - E_L) + g_L \cdot \Delta_T \cdot e^{\frac{V_m(t)-V_{th}}{\Delta_T}} - w(t) + I_m(t) & V_m(t) \text{ Membrane Potential} \\ \tau_w \cdot w'(t) = a \cdot (V(t) - E_L) - w(t) & w(t) \text{ Adaptive current} \end{cases}$$

If  $V_m(t) \geq V_{th} \rightarrow SPIKE:$

$$\begin{cases} V_m(t+1) = V_r \\ w(t+1) = w(t) + b \end{cases}$$

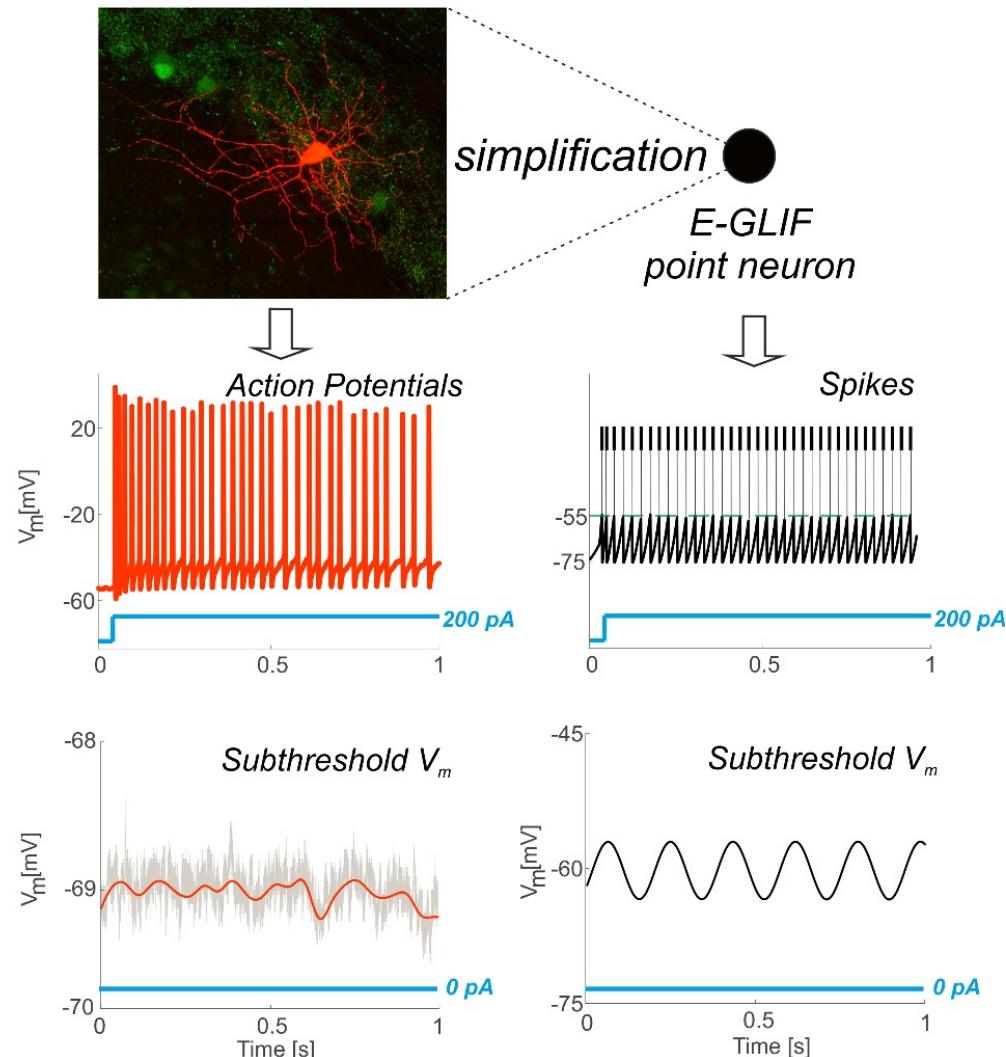
Properties:

- multiple electroresponsive properties based on parameter values
- replacement of the strict voltage threshold by a more realistic smooth spike initiation zone.
- subthreshold resonances or adaptation as in the Izhikevich model.

[Brette and Gerstner, *J Neurophysiol*, 2005]

# Towards a unified point neuron model for cerebellar neurons

- Aim: a model able to reproduce all the cerebellar electroresponsive mechanisms, while keeping:
  - **Neurophysiological realism** (elements in the model  $\leftrightarrow$  biophysical mechanisms)
  - **Low computational load** ( $\rightarrow$  linear and analytically solvable, to increase simulation step without loosing precision within large-scale Spiking Neural Networks - SNNs)
  - **Generalized features** (not fitting on single traces)
  - Different **sets of parameters** for different cells, reproducing **all** the electrophysiological properties of each population, i.e. **spike patterns** more than sub/supra-threshold mechanisms (since within SNN)



# Extended-Generalized LIF neuron model (E-GLIF)

Membrane potential  $V_m$

Adaptive current  $I_{adapt}$

Spike-triggered depolarizing current  $I_{dep}$

Biological quantities/parameters

Artificial parameter

$$\begin{cases} V'_m(t) = \frac{1}{C_m} \left( \frac{C_m}{\tau_m} (V_m(t) - E_L) + I_{stim} + I_e + I_{dep}(t) - I_{adapt}(t) \right) \\ I'_{adapt}(t) = k_{adapt} (V_m(t) - E_L) - k_2 I_{adapt}(t) \\ I'_{dep}(t) = -k_1 I_{dep}(t) \end{cases}$$

Refractory period  $T_{ref}$

Stochasticity in spike generation

$$\begin{cases} t_{spk} \notin \Delta t_{ref} \\ rng < (1 - e^{-\lambda(t_{spk})t_{spk}}) \end{cases} \quad \lambda(t) = \lambda_0 e^{\frac{V_m(t) - V_{th}}{\tau_V}}$$

Updates at spike event

$$\begin{cases} V_m(t_{spk}) \leftarrow V_r \\ I_{dep}(t_{spk}) \leftarrow A_1 \\ I_{adapt}(t_{spk}) \leftarrow I_{adapt}(t_{spk} - 1) + A_2 \end{cases}$$

$I_{stim}$  = external stimulation current;

$C_m$  = membrane capacitance;

$\tau_m$  = membrane time constant;

$E_L$  = resting potential;

$I_e$  = endogenous current;

$k_{adapt}, k_2$  = adaptation constants;

$k_1$  =  $I_{dep}$  decay rate;

$V_{th}$  = threshold potential;

$\lambda_0, \tau_V$  = escape rate parameters;

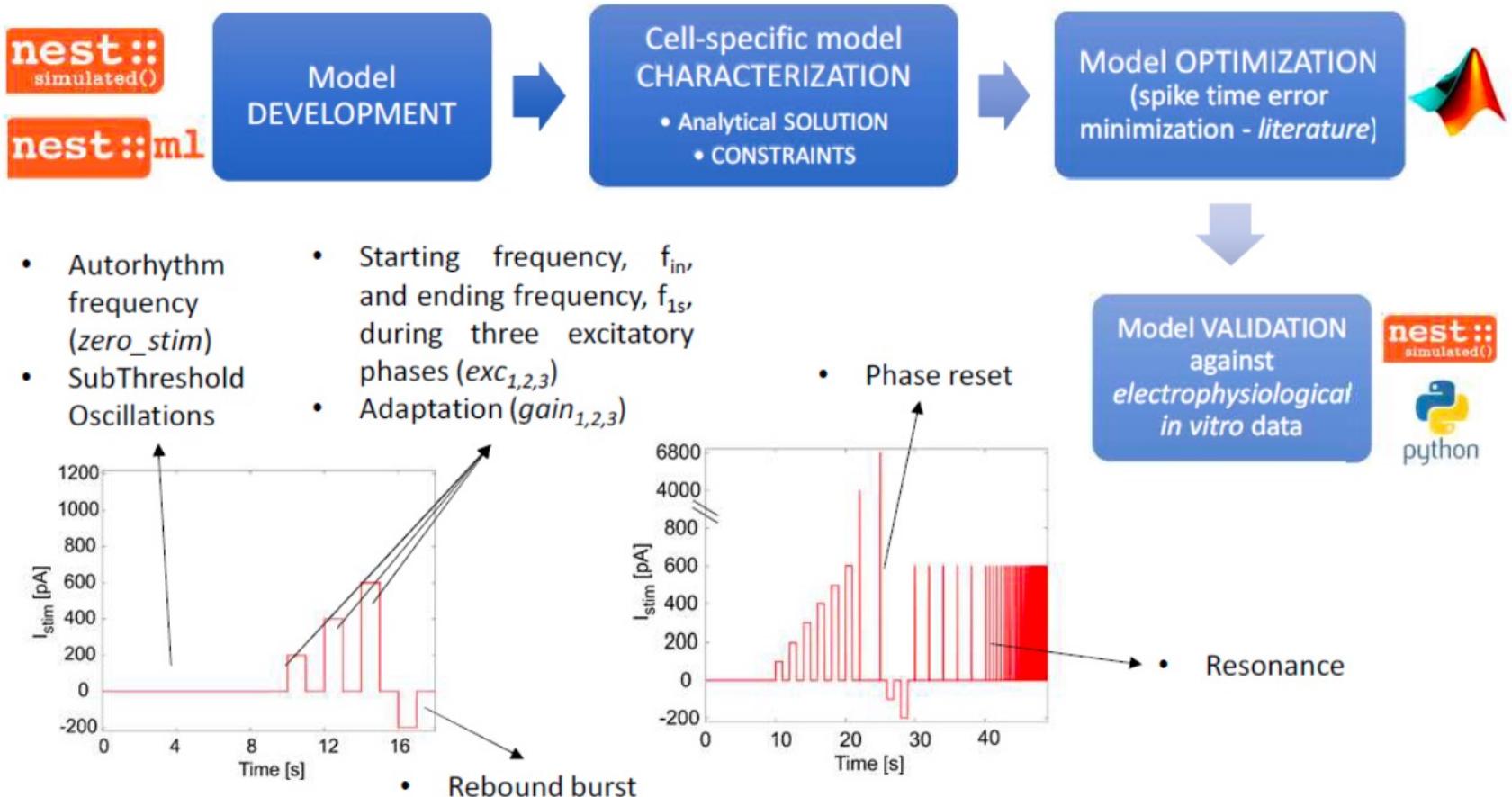
$t_{spk}^+$  = time instant immediately following the spike

$V_r$  = reset potential;

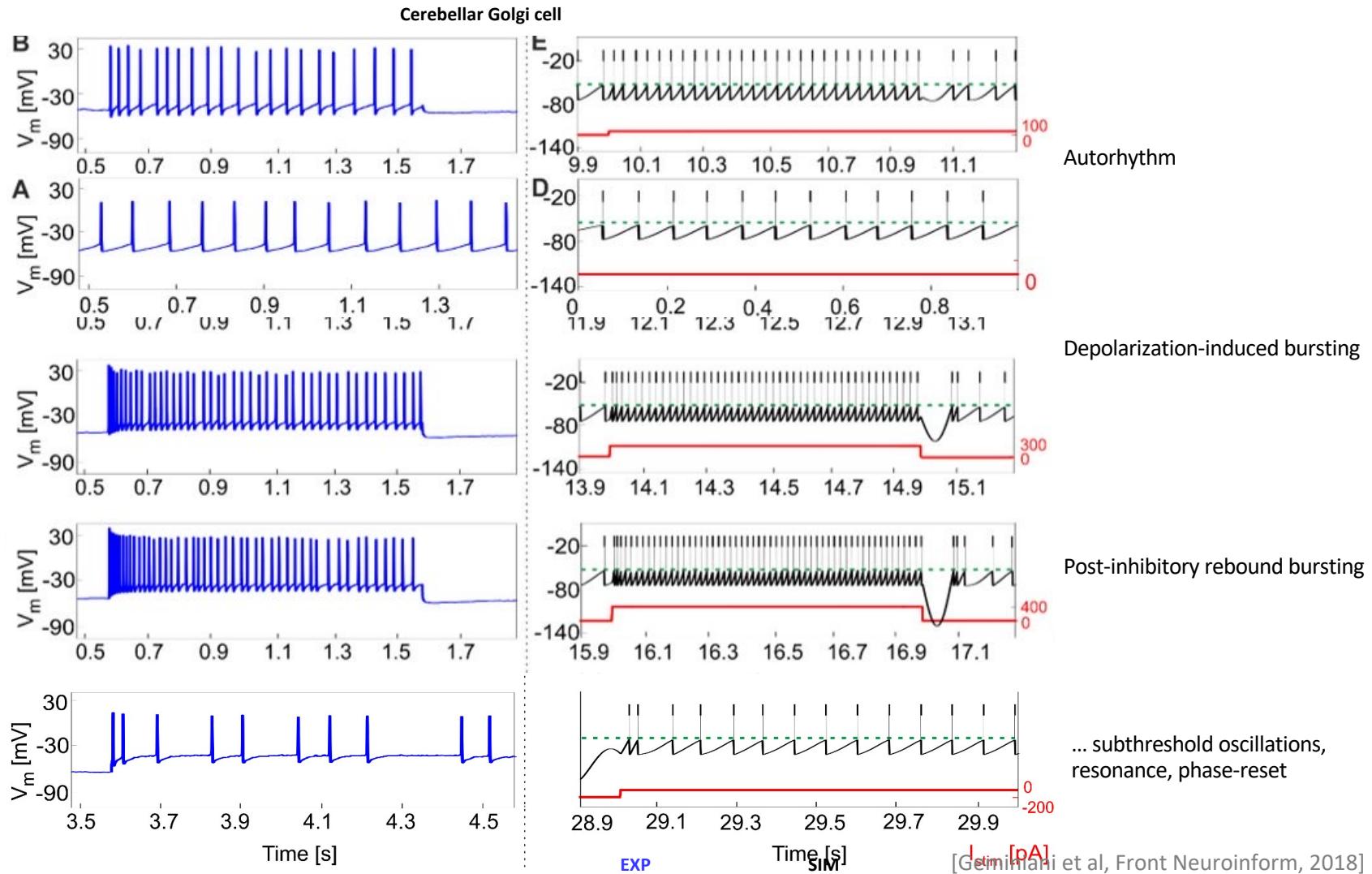
$A_2, A_1$  = model currents update constants.

[Geminiani et al, *Front Neuroinform*, 2018]

# E-GLIF – implementation, optimization and validation

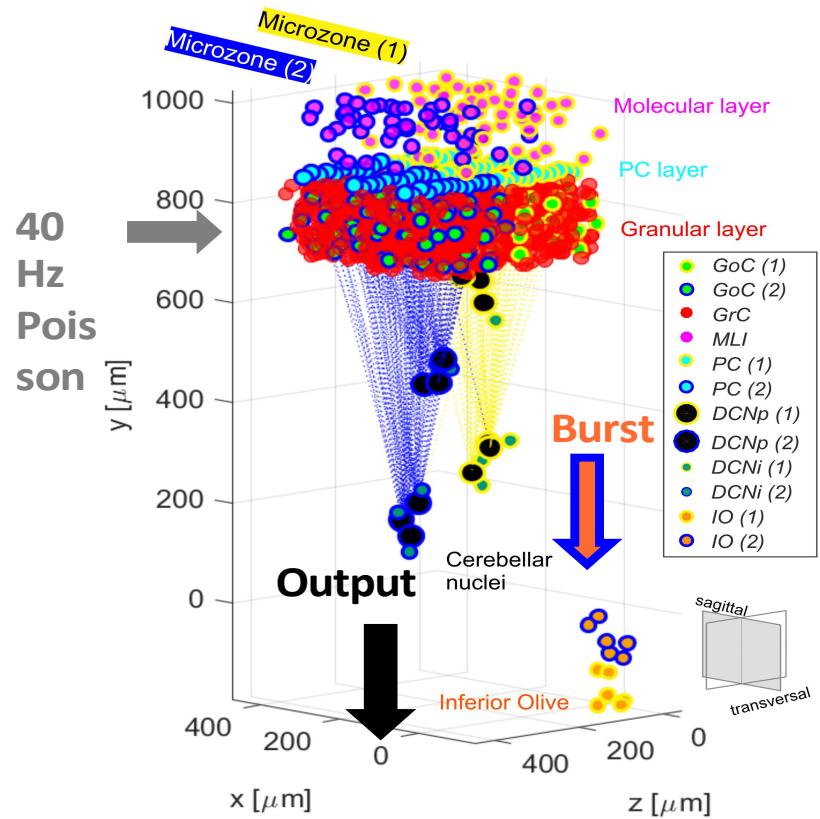
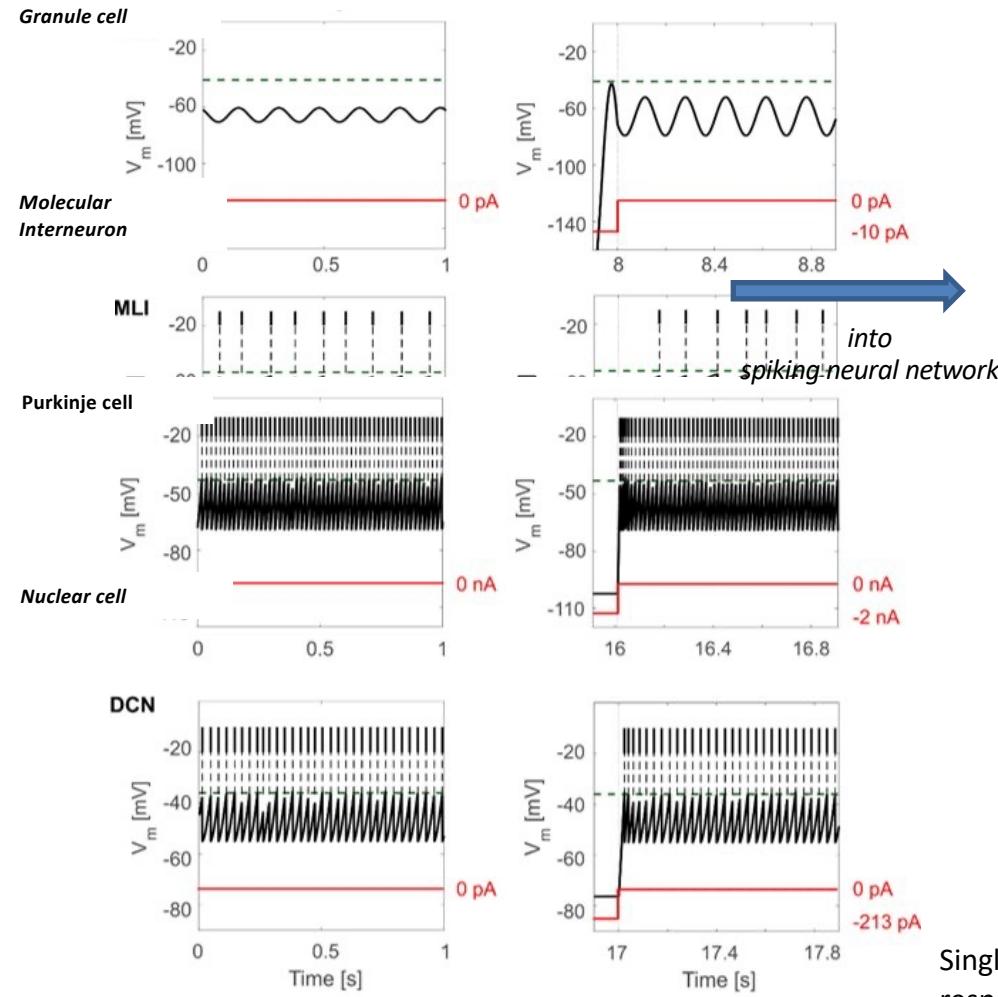


# E-GLIF for cerebellar Golgi cells: validation against experimental data



# E-GLIF for other cerebellar neurons

Tuned on experimental data for the **other cerebellar neurons**



Single neuron properties propagate to network dynamics: pause-burst responses enhance the time precision of the output

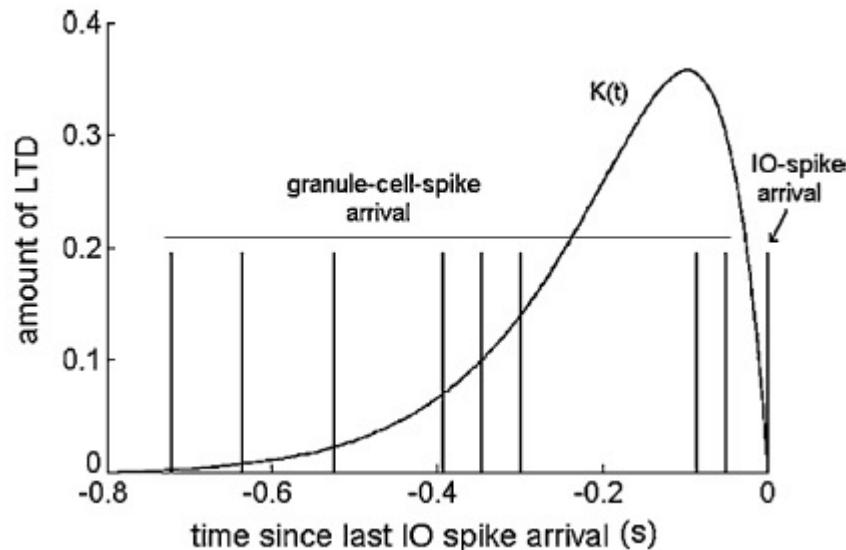
# Modelling plasticities (pf-PC)

LTD

$$\Delta w_i = \beta \int_{-\infty}^{t_{IOSPIKE}} k(t_{IOSPIKE} - t) h(t)_{PF_i} dt$$
$$k(t) = e^{-\left(\frac{t-t_0}{\tau}\right)} \sin\left(2\pi\left(\frac{t-t_0}{\tau}\right)\right)^{20}$$
$$h(t) \begin{cases} 1 & \text{if } PF_i \text{ is active at time } t \\ 0 & \text{otherwise} \end{cases}$$

LTP

$$\Delta w_i = \alpha$$



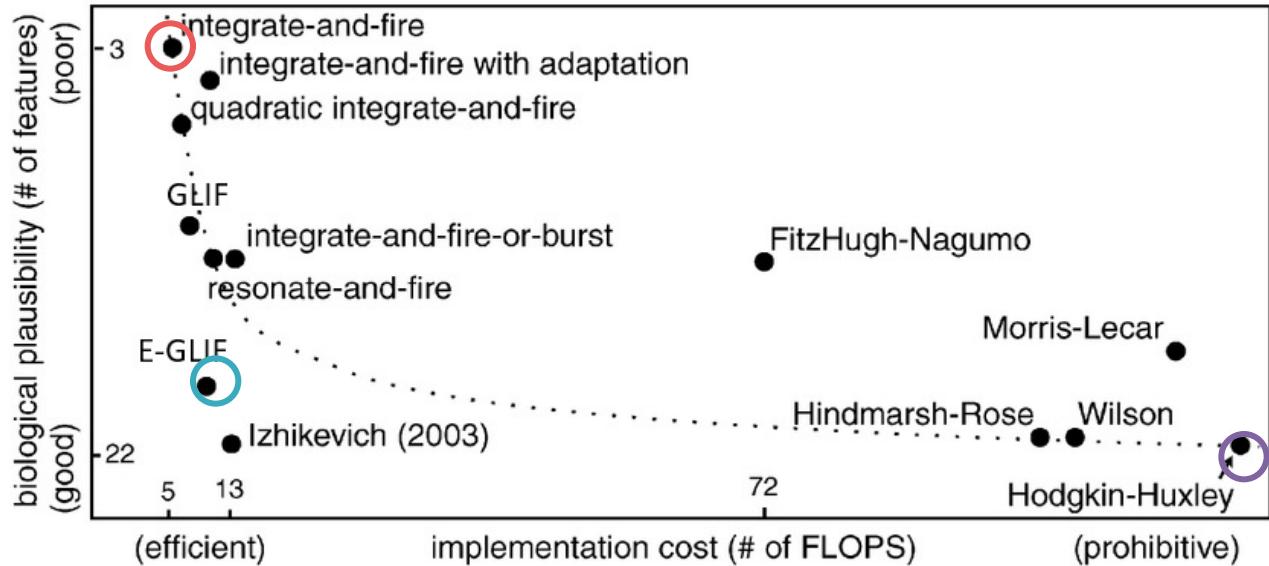


Figure 2.1 Trade-off between biological plausibility and computational load in single neuron models. Basic IF and HH models are at the two extreme cases. GLIF and E-GLIF approximate values have been added. The E-GLIF simplifies the action potential representation, but approaches the Izhikevich and HH precision in terms of firing patterns, outperforming the other multidimensional LIF models. In multi-compartment models, the HH representation remains the standard and amplifies by orders of magnitude both computational capability and biological plausibility. Modified from ([Izhikevich, 2004](#)).

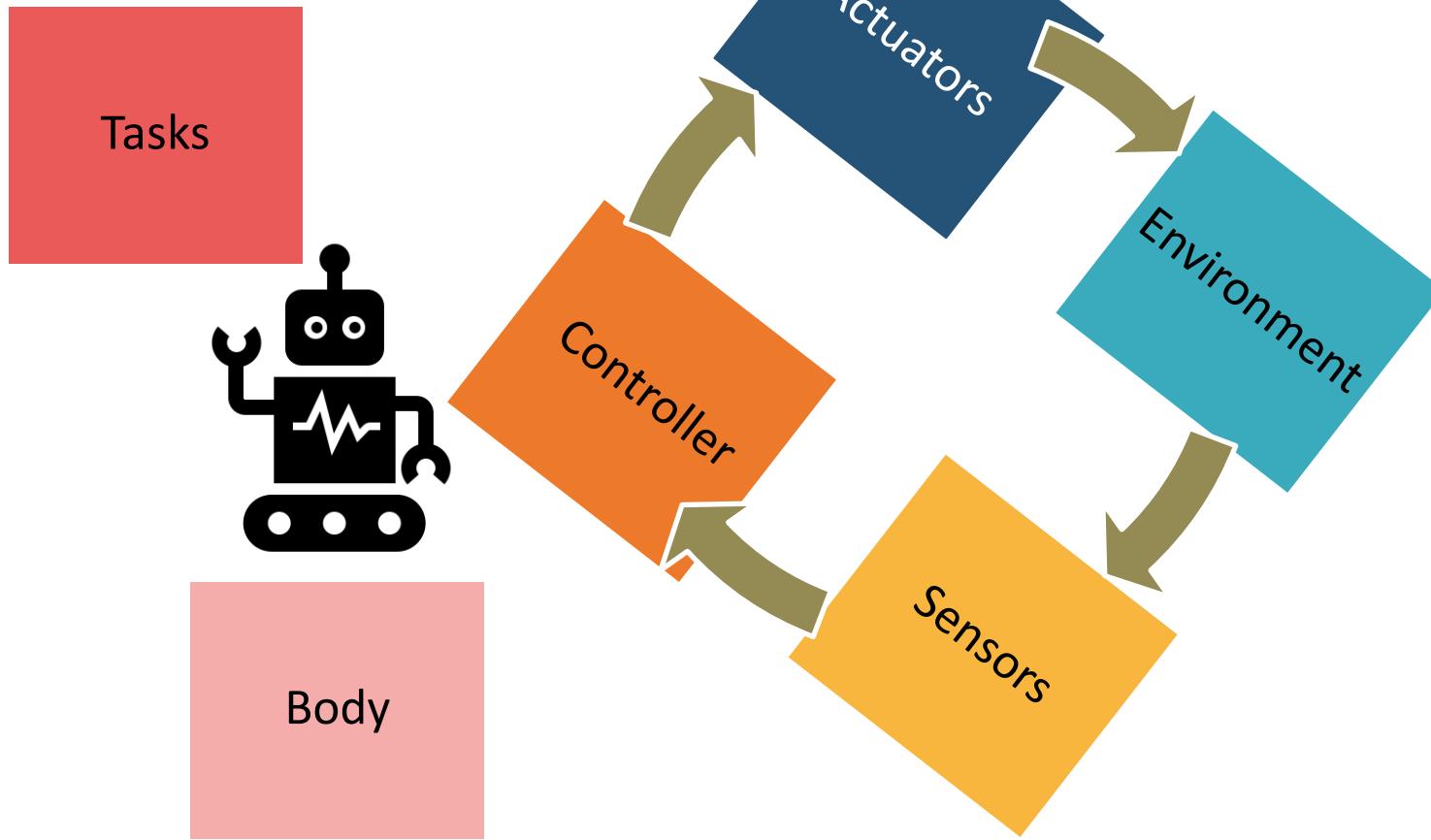


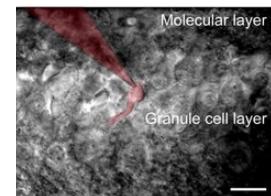
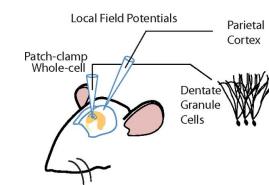
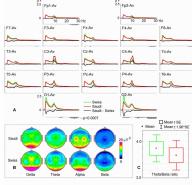
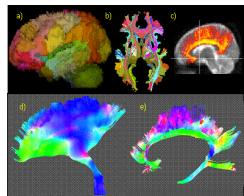
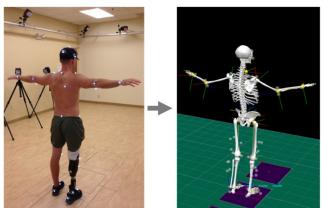
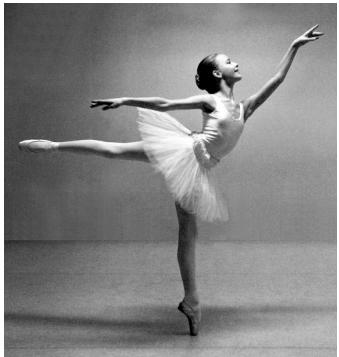
# Neuroengineering 2021/22

COMPUTATIONAL NEUROSCIENCE - Part 5 Neurorobotics

# Neurorobotics

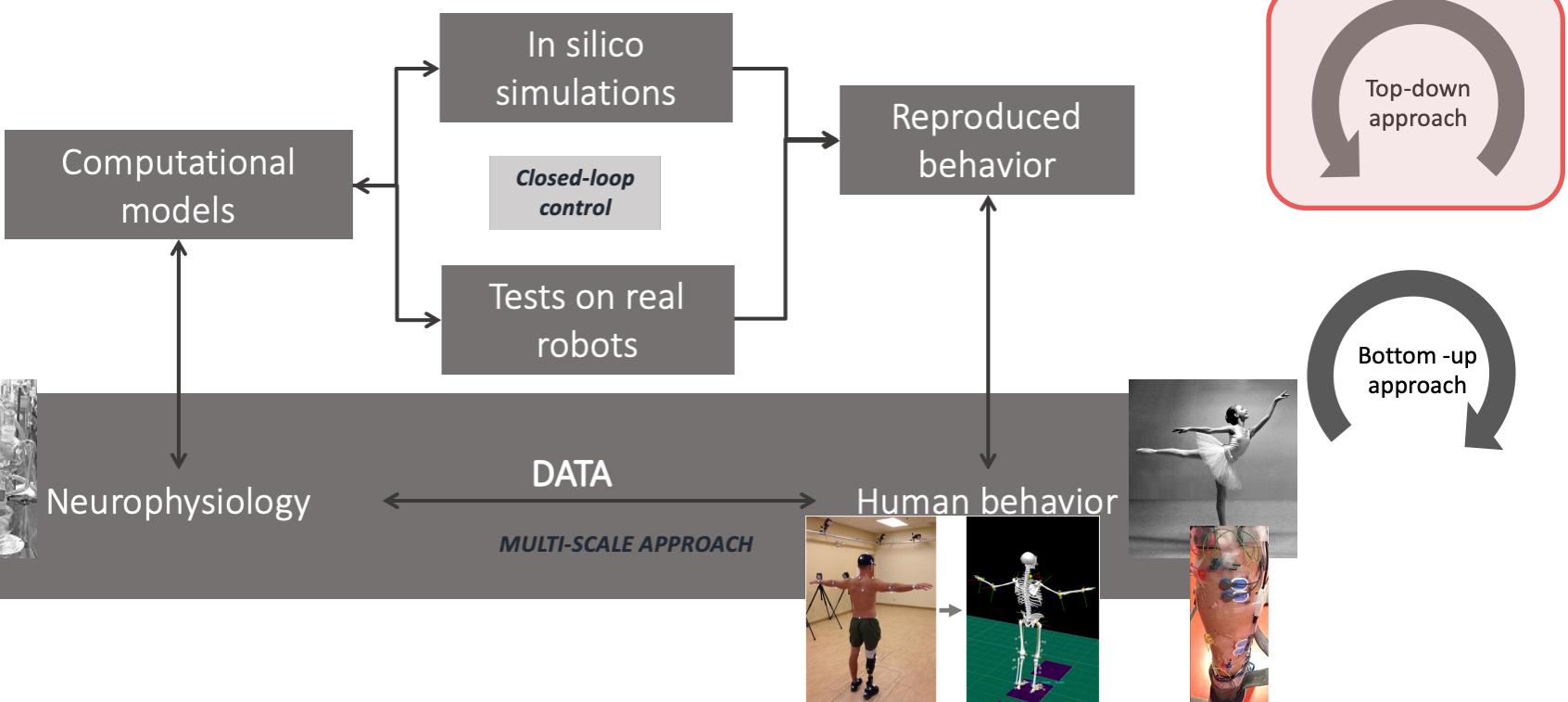
Human Brain Project





# Approaches to neurorobotics design

Human Brain Project



D'Angelo et al. Funct. Neurol 2013

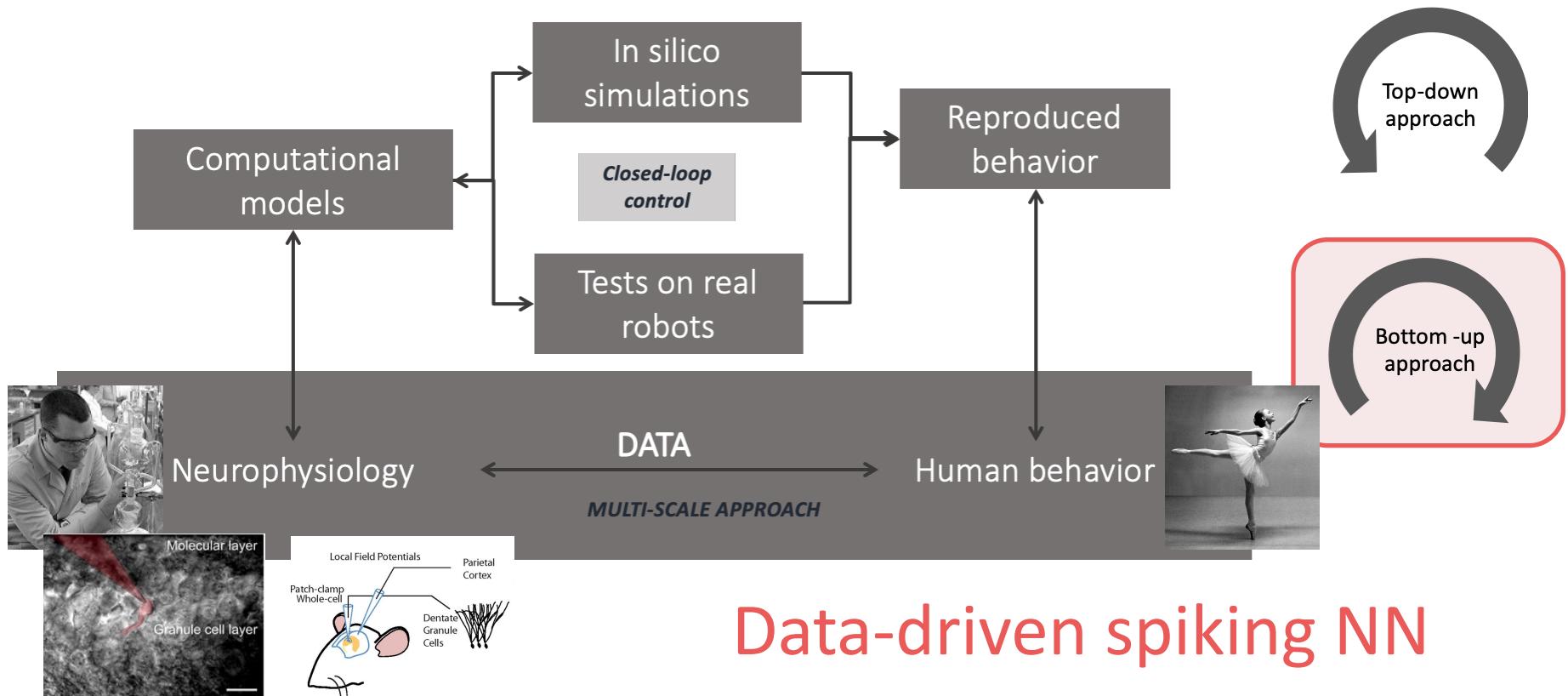
D'Angelo et al. Front. Cell. Neuroscience, 2016

# Approaches to neurorobotics design

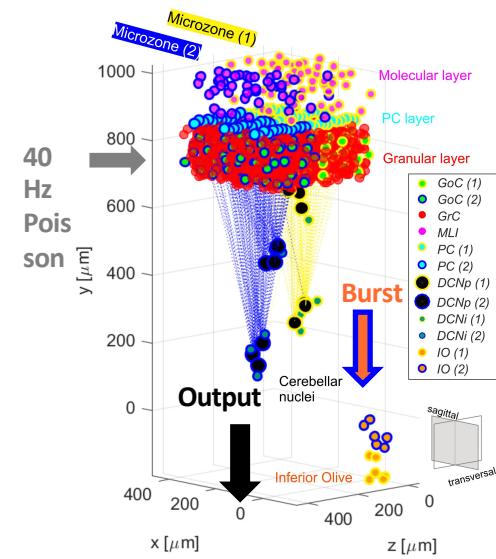
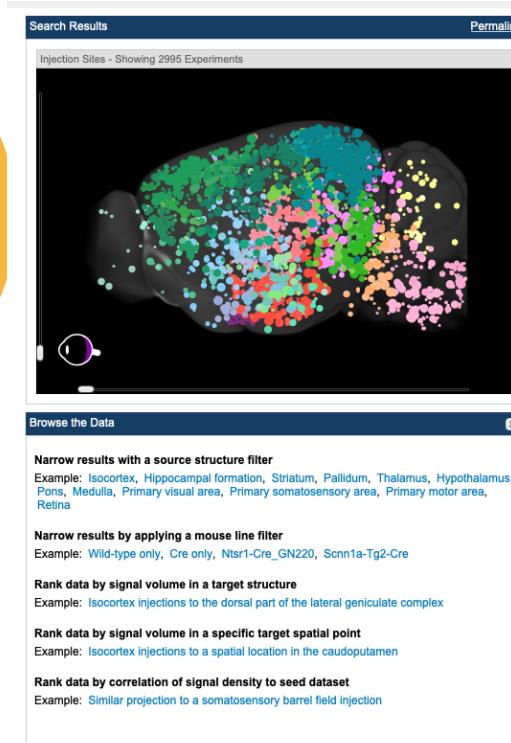
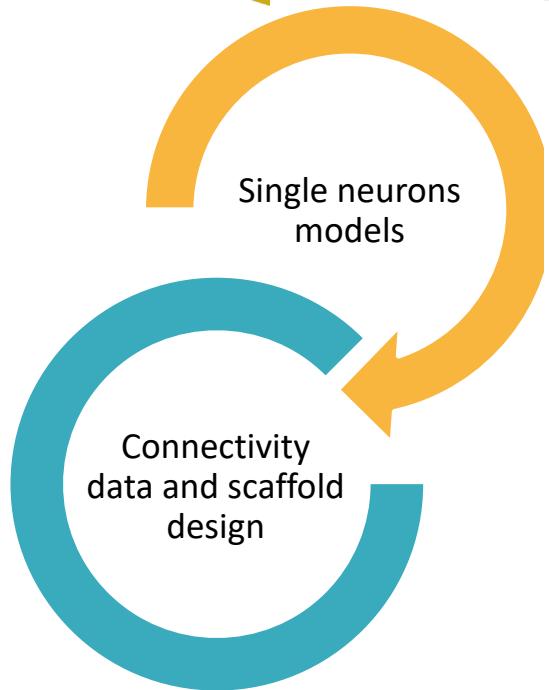
Human Brain Project

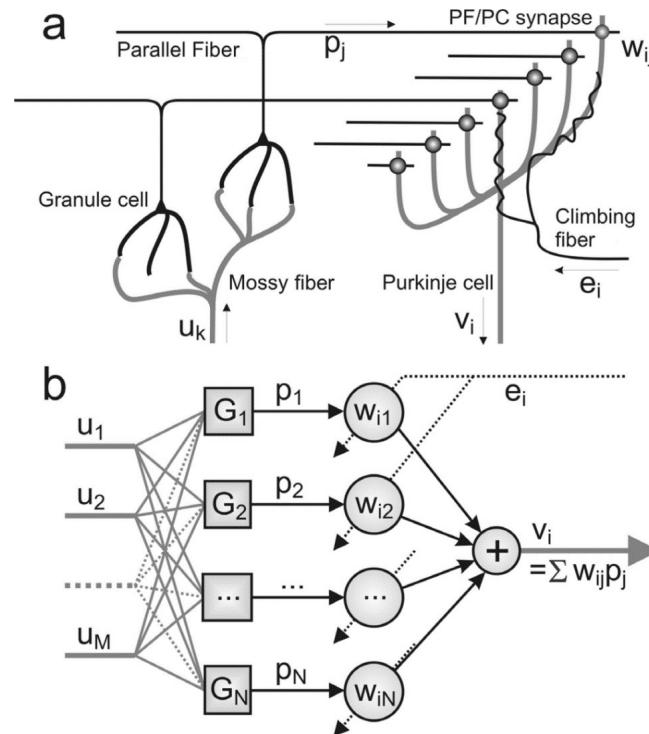
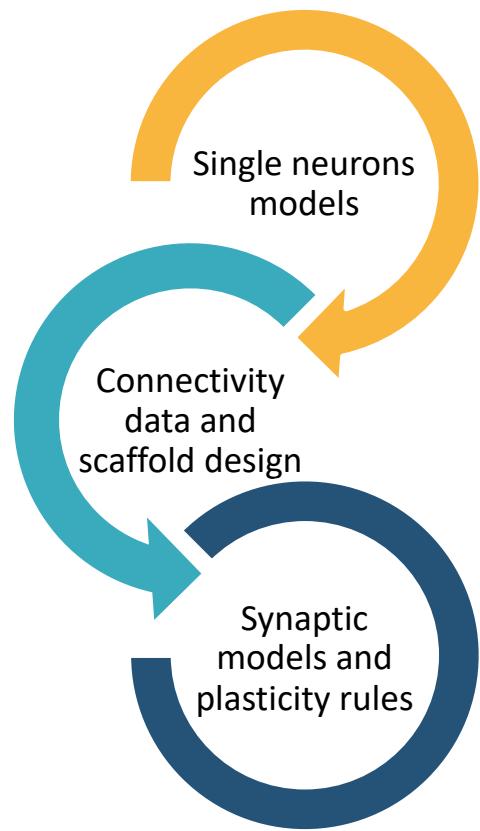


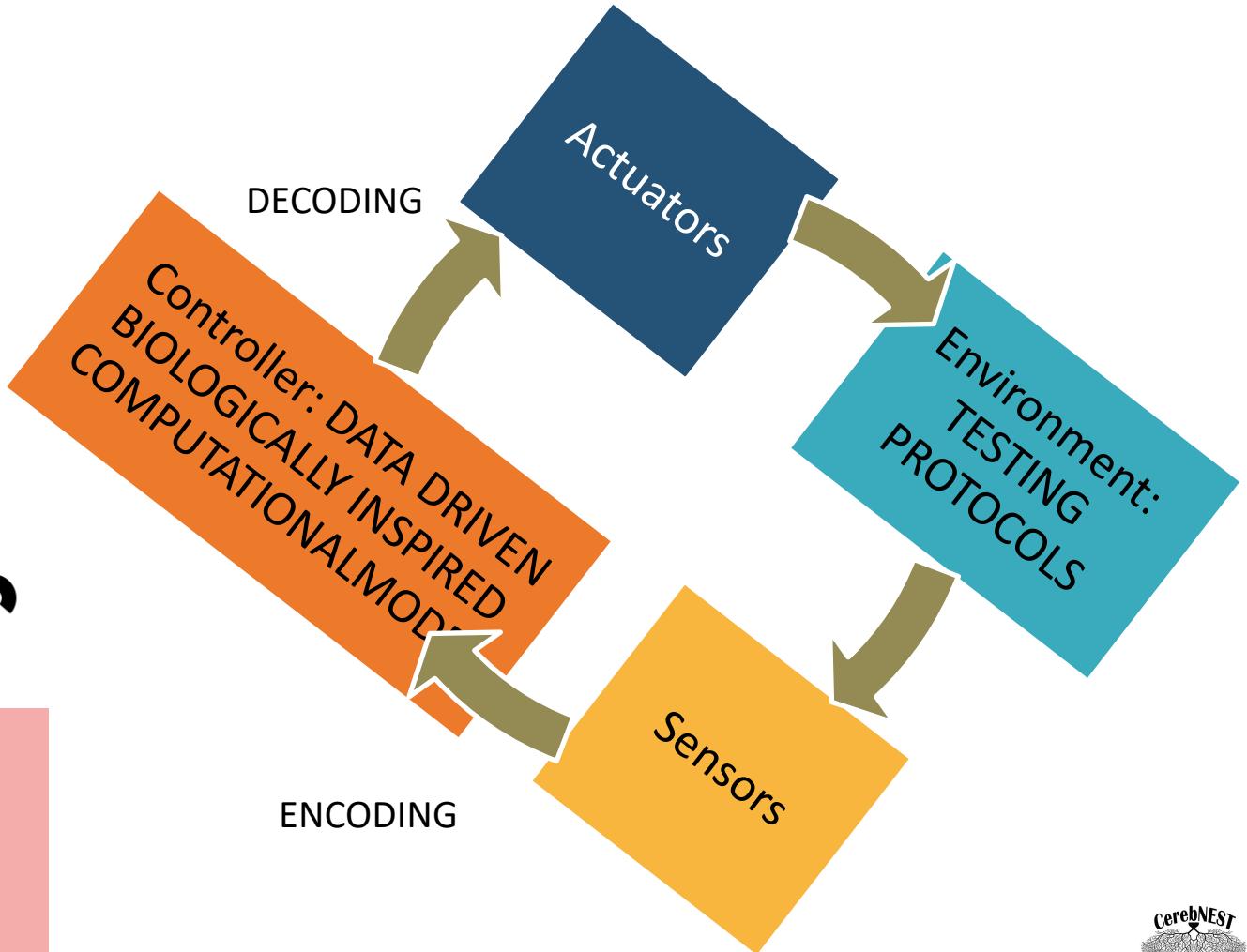
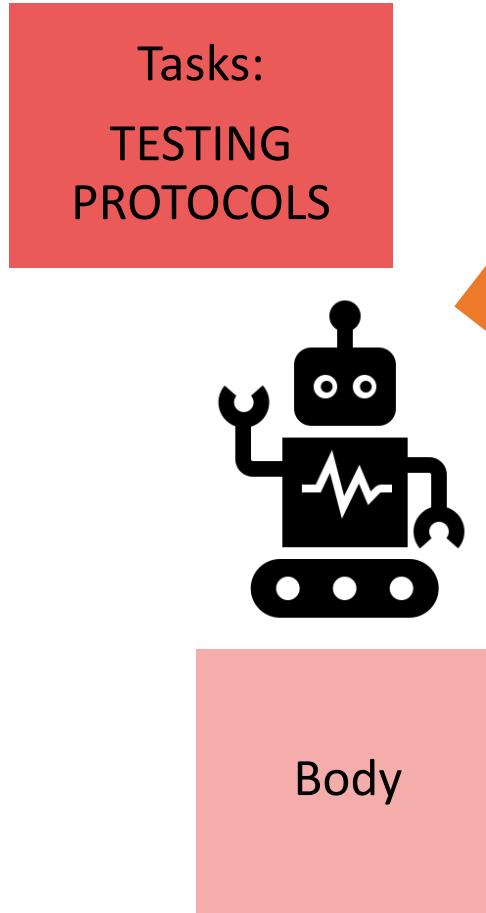
D'Angelo et al. Funct. Neurol 2013  
D'Angelo et al. Front. Cell. Neuroscience, 2016



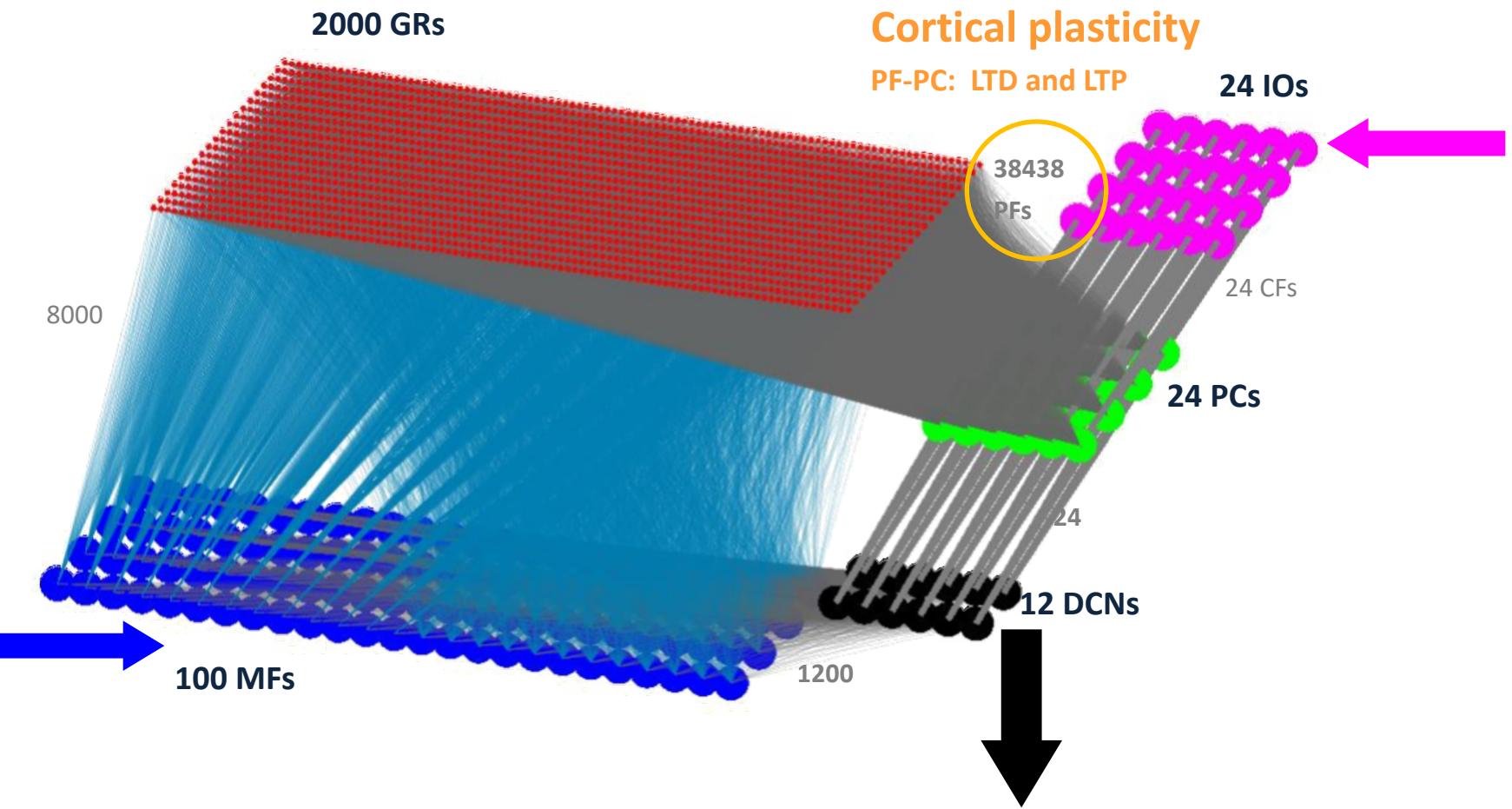
## Data-driven spiking NN







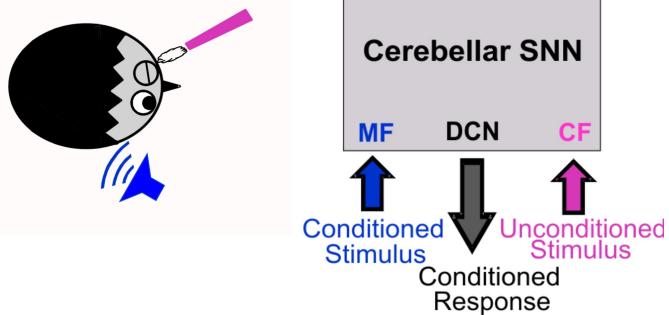
# Cerebellar spiking NN



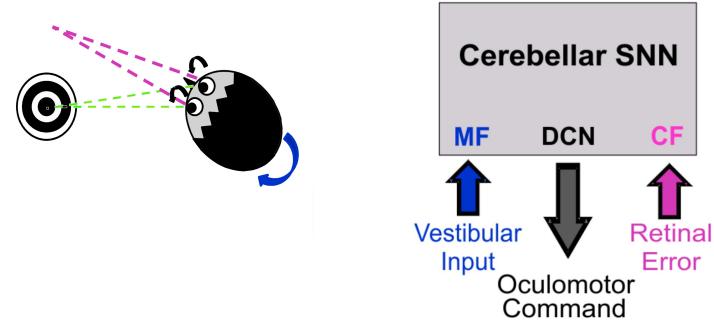
# Testing protocols of the Cerebellar Models

Casellato et al., PlosOne 2014;  
Antonietti et al., IEEE TMBE 2015

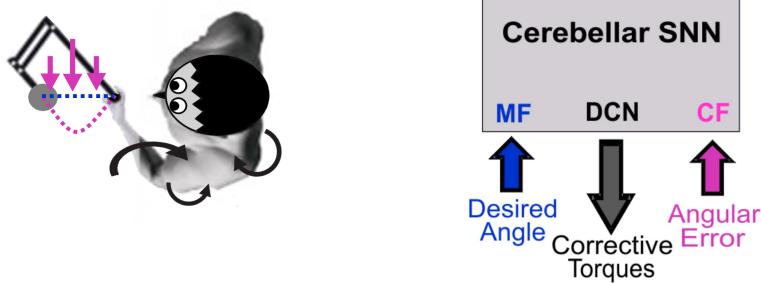
Eye Blink Classical Conditioning (EBCC)



Vestibulo-Ocular Reflex (VOR)



Movements perturbed by Force Fields (FF)



LEARNING FEATURES EMERGE  
FROM PLASTICITY CHANGES IN THE SNN

# Embodiment with real robots



Ros et al. Neural Computation 2006  
Luque et al. Int. J. Neural Syst. 2011  
Antonietti et al., IEEE TMBC 2015  
Antonietti et al. IEEE TNNLS 2018

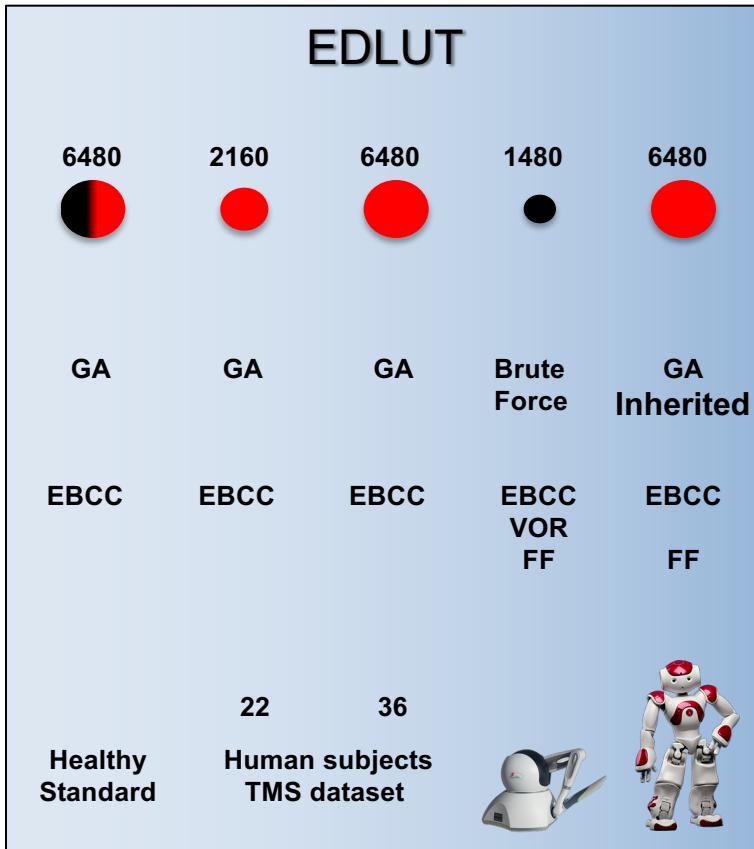
## Simulator

## Number of Neurons Plasticity (1 vs 3)

## Parameter Optimization

## Testing paradigms

## Embedding or Data Fitting



# Embodiment with real robots



Ros et al. Neural Computation 2006  
Luque et al. Int. J. Neural Syst. 2011  
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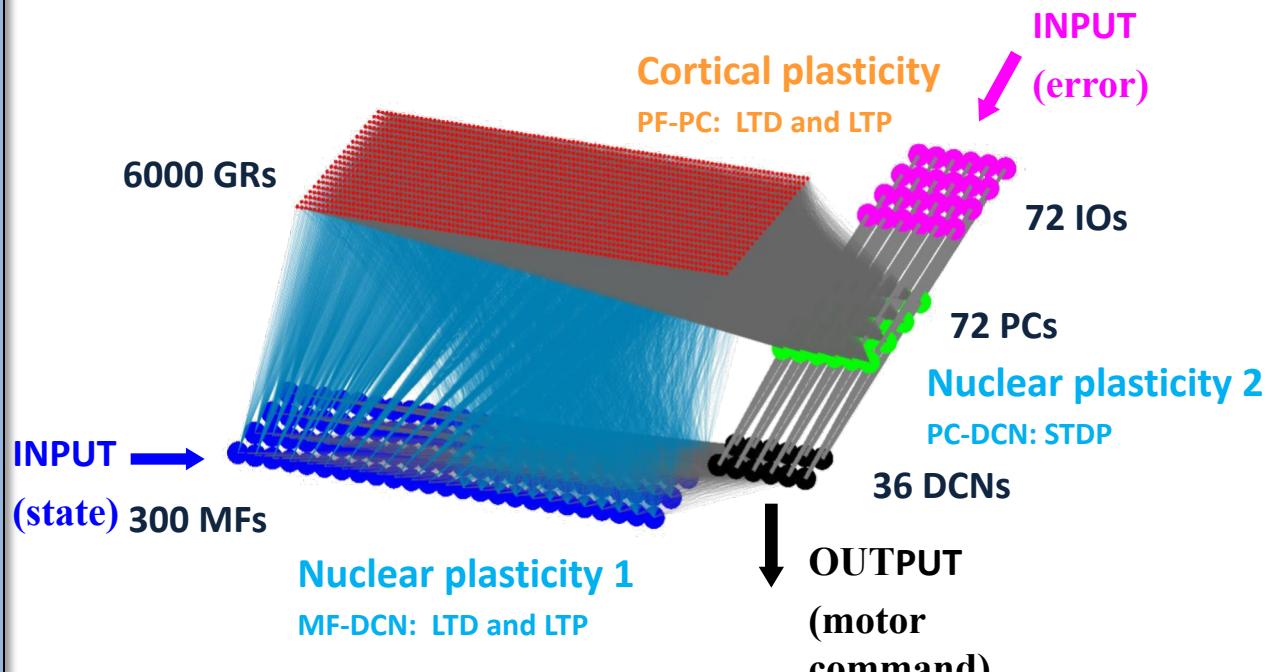
## Simulator

Number of Neurons  
Plasticity (1 vs 3)

Parameter  
Optimization

Testing paradigms

Embedding or  
Data Fitting

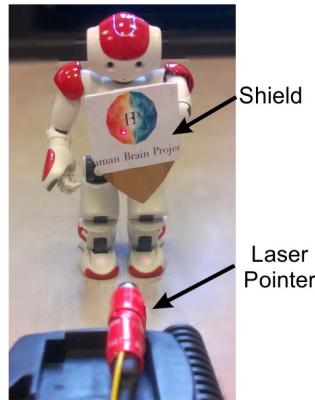


# EBCC with Nao robot



Antonietti et al., IEEE TMBE 2015;  
Antonietti et al., IEEE TNNLS 2018

NAO Robot

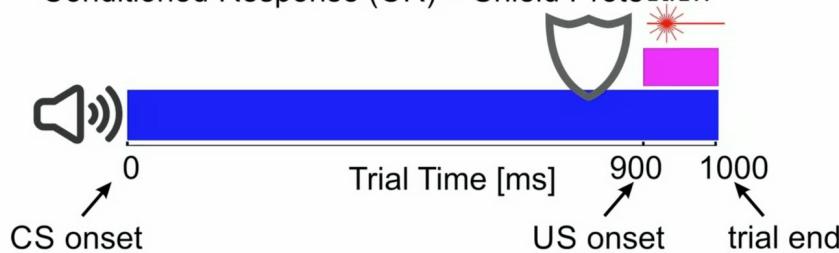


## During the Acquisition Phase (13 Trials)

Conditioned Stimulus (CS) = Tone

Unconditioned Stimulus (US) = Laser Beam

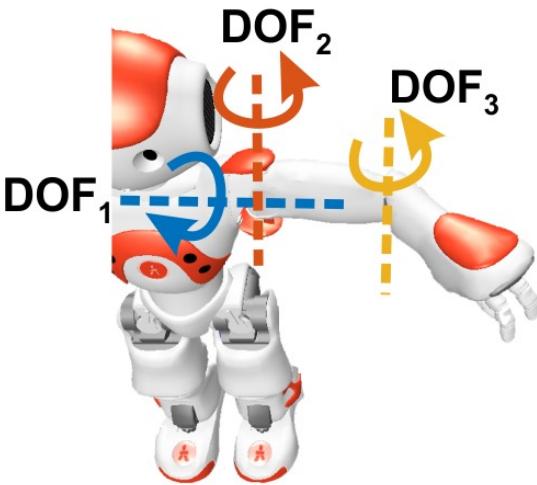
Conditioned Response (CR) = Shield Protection



NAO wants to protect himself from the US

# Robotic Embodiment of the Cerebellar Models: FF with NAO Robot

## Multi-joint Force Field with NAO Robot

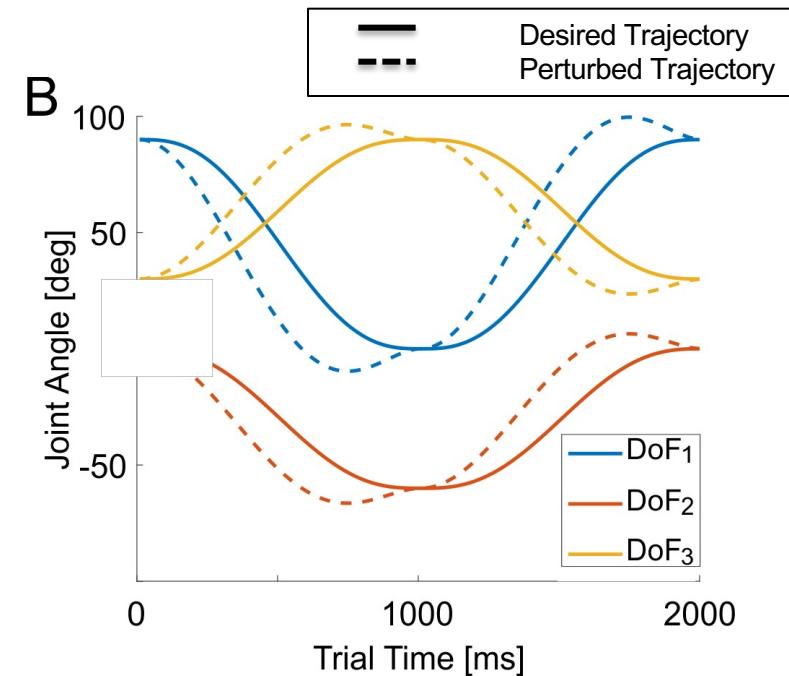


### Protocol

**5 trials of Baseline**  
(No perturbation)

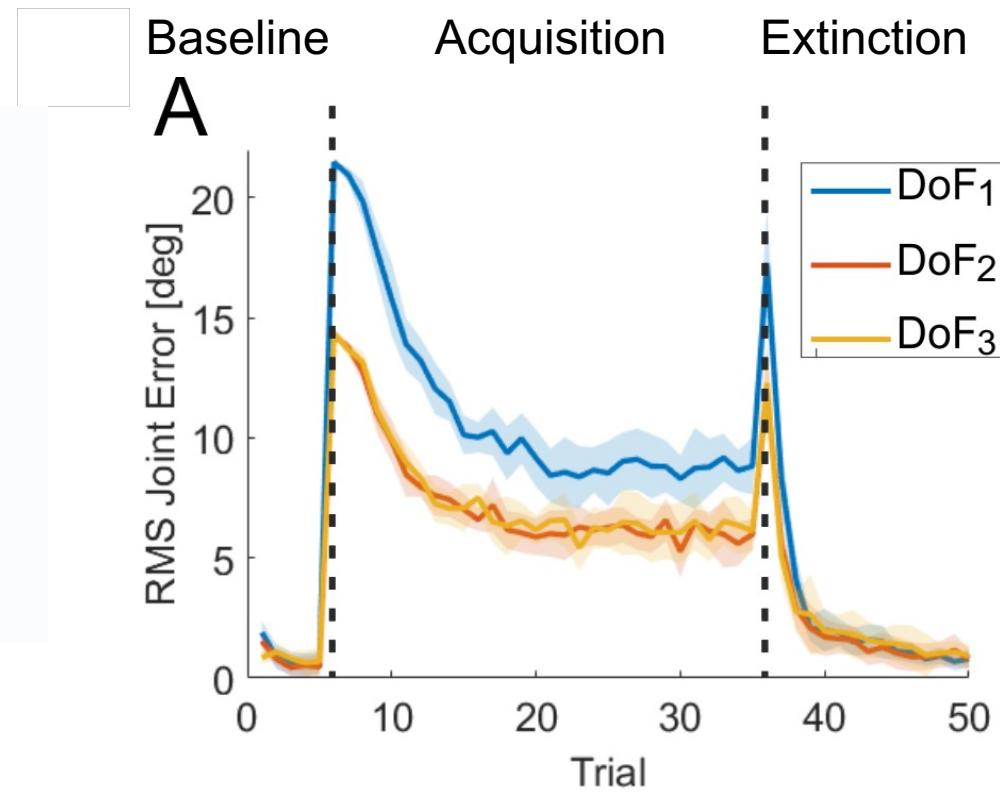
**30 trials of Acquisition**  
(Perturbation starts)

**15 trials of Extinction**  
(Perturbation ends)



# Robotic Embodiment of the Cerebellar Models: MC with NAO Robot

5 trials of Baseline  
(No perturbation)





Antonietti et al., Comp. Int. And Neurosc. 2019;

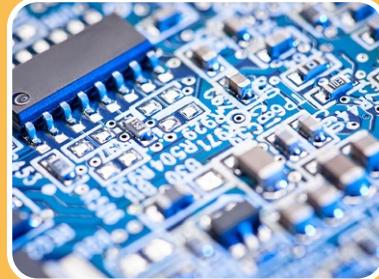
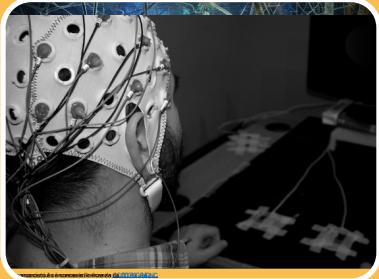


# Embodiment with virtual robots: NRP

- Brain simulators at increasing computation load (more features, more neurons, more plasticity,...)
- Physics simulator (virtual robots)
- Robot controller
- Experiment pipeline



# Impacts

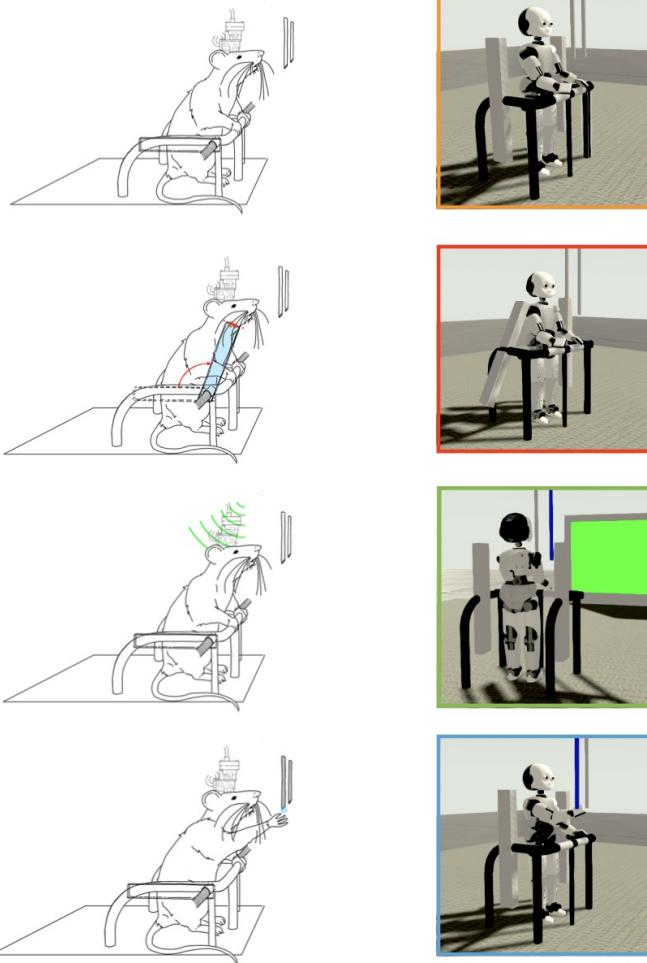


Impacts on  
Basic  
Neuroscience

Impacts on  
Applied  
Neuroscience

Impacts on  
Robotics and  
Computer  
Science

# Impacts on basic neuroscience



**Figure 9:** *In vivo* and *in silico* protocol co-execution. The robotic subject successfully performs the protocol as it was designed to be executed *in vivo*. The colored frames are used to mark a temporal reference on the spiking plot in Figure 8.

# Impacts on applied neuroscience



Geminiani et al., Int J Neural Syst 2018

- From physiological to pathological models

