# Coincidence detection between apical and basal dendrites drives STDP in cerebellar Golgi cells

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# *Supplementary Information*

***List of abbreviations:***

$Ca^{2+}$, calcium;

LTP, long-term potentiation;

LTD, long-term depression;

mf, mossy fiber.

GrCs, granule cells;

GoCs, Golgi cells;

pf, parallel fiber;

aa, ascending axon;

EPSC, excitatory post-synaptic current;

EPSP, excitatory post-synaptic potential:

STDP, spike-timing dependent plasticity;

GL, granular layer.

***Supplementary note 1.***

***Modeling of long-term synaptic plasticity***

Plasticity was modeled following the approach described in [1], and [2]. The equations used below have been adapted mainly from [1]. According to the $Ca^{2+}$ control hypothesis [3], synaptic plasticity is a non-linear [BCM-shaped] function of the activity-dependent rise in postsynaptic $Ca^{2+}$ concentration: large $Ca^{2+}$ transient should lead to LTP, while a moderate increase in $Ca^{2+}$ concentration determines LTD. When the $Ca^{2+}$ concentration change is negligible or null, no synaptic plasticity of any kind takes place. The computational model proposed by Shouval and Bear [1] for the hippocampus has been adapted to the mf - GrC synapse as follows.

***The Ω function***

The Ω function expresses the relation between $Ca^{2+}$ concentration and changes in synaptic weight (W).

$$\Omega = 0.5 + sig(Ca - \alpha 2, \beta 2) - 0.5\ sig(Ca - \alpha 1, \beta 1)$$

Where *sig* is a sigmoidal function defined as:

$$sig(x, \beta) = exp(\beta x) / (1 + exp(\beta x))$$

with $\alpha 1 = 0.25$, $\alpha 2 = 0.77$, $\beta 1 = 80$ and $\beta 2 = 80$. These values allowed to fit the plasticity model to existing experimental data [4].

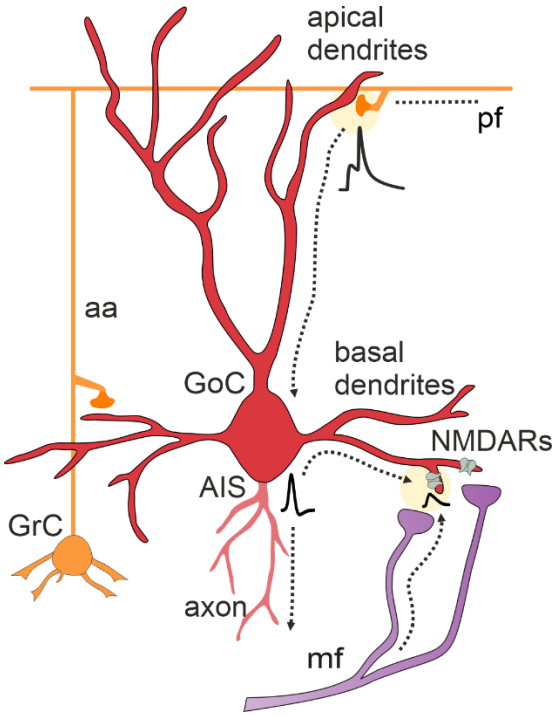
***The η function***

The $\eta$ function represents the calcium-dependent learning rate and is inversely related to the learning time constant $\tau$ such that $\eta = 1/\tau$, with:

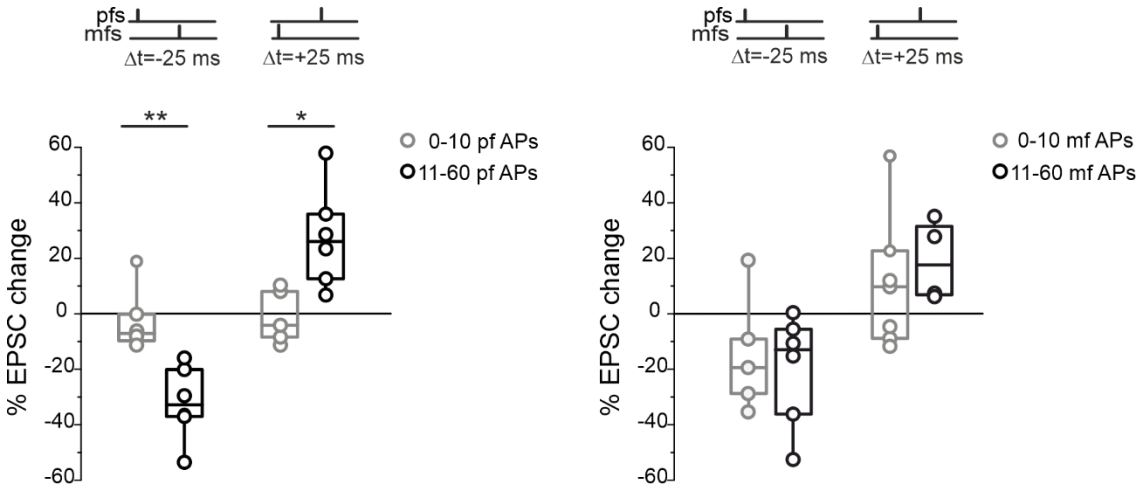
$$\tau = \frac{P_1}{P_2 + (Ca)^{P_3}} + P_4$$

The values of parameters have been taken from [2] : $P_1 = 100$ ms, $P_2 = 0.002$, $P_3 = 4$, $P_4 = 1000$ msec.

***Supplementary Tables and Figures***

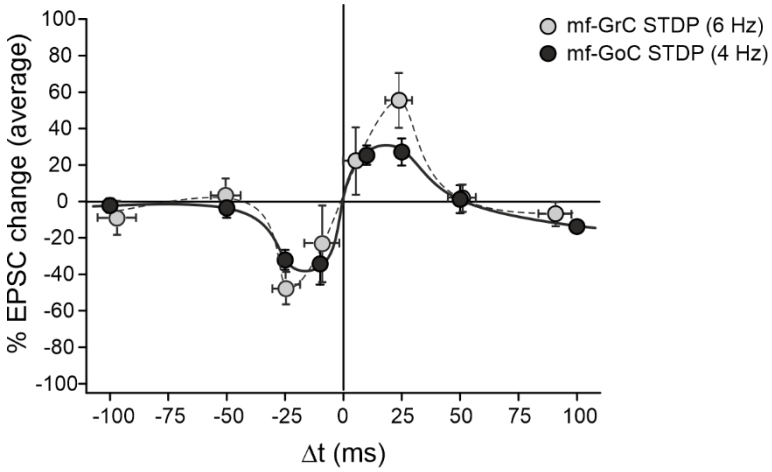



**Supplementary Figure 1**. ***Schematic representation of a GoC***. Enlargement of the GoC microcircuit, illustrating the afferent excitatory inputs from pfs/aas and mfs onto apical and basal dendrites, respectively, and the process of dendritic integration revealed by previous experimental and modelling works [5].



**Supplementary Figure 2.** ***STDP requires the action potential elicited by parallel fiber stimulation.***

Box-and-whisker plots show the changes in EPSC amplitude based on the number of APs evoked by pf stimulation (left; ≤ 10 out of 60 stimuli, gray circles; > 10 out of 60 stimuli, black circles) or by mf stimulation (right; ≤ 10 out of 60 stimuli, gray circles; > 10 out of 60 stimuli, black circles) during STDP induction. *, $p < 0.05$, **, $p < 0.01$, ***, $p < 0.001$; Student's unpaired *t*-test.




**Supplementary Figure 3**. ***STDP in the GL.*** mf-GoC STDP timing curve obtained following pf-EPSP → mf-EPSP and mf-EPSP → pf-EPSP pairing (Δt = ± 25 ms; black circles) compared with that obtained from [6] (gray circles). Data in the plot indicates mean±SEM.

## *Supplementary References*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