

GABA-glycine co-release and its consequences on inhibition

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Numerous, if not all, neuropathies result from an excitation-inhibition imbalance leading to improper function. Aiming a better understanding of neurotransmission mechanisms is thus worth it. In the last decades, numerous evidences proved that synaptic transmission is far more complex than we thought with some neurons able to co-released several neurotransmitters. This presynaptic diversity of neurotransmitter echoes the one of available receptors in postsynaptic, presynaptic and extrasynaptic positions and let glimpses cooperatives mechanisms of neuronal activity regulation. It is surprising then that GABA-glycine co-release, despite it appearing redundancy, is one of the most common co-release system encounter in the brain. Indeed, functional GABA-glycine co-release have been identified in the thalamus, brainstem, spinal cord and cerebellum after the developmental GABA/glycine shift occurs. In those regions, GABA and glycine play complementary roles to regulate excitability *via* inhibitory currents kinetics and metabotropic effects. There is however, a puzzling case in the cerebellum where the 50 billions granule cells composing its cortex can only be inhibited by GABA mainly co-released with glycine by Golgi cells. Despite the absence of glycinergic receptor at that synapse, we found that a glycine application decrease the GABAergic inhibition of granule cells. Moreover, this effect is diminished with the increase of *de novo* GABA synthesis and completely reversed when neuronal supply of cytosolic glycine is blocked. As GABA and glycine interact with the same vesicular transporter, we hypothesized that changes in cytosolic concentration of glycine may dynamically influence vesicular filling of GABA by competition and thus granule cells inhibition strength.

Biography :

Passionate about Science from childhood, I developed a solid base of knowledge and skills during my 10 years journey in Life science. After a bachelor's degree in biology and a master of neuroscience at the Rouen University I pursued with a PhD at IBENS where I focused my research on the Golgi-granule cells inhibitory transmission in adult mice cerebellum. Presenting

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