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***In vitro* monosynaptic circuits of *Helix* neurons as an experimental model to study synapsin knock-down**

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Synapsins (Syn) are a conserved family of presynaptic proteins, involved in the fine-tuning of synaptic transmission. Syn mutations have been related with epileptogenesis and the study of mammalian knock-out models has clarified in some degree their role in this process. But the presence of different genes and isoforms, and the possible development of compensatory mechanisms hinder an accurate interpretation of results. Consequently, we are developing an *in vitro* monosynaptic connection, as a reliable Syn knock-down model, in order to over-express and analyze epilepsy-related forms of Syn.

We cloned two antisense RNAs (asRNA) against *Helix* synapsin (helSyn) into a plasmid able to constitutively overexpress them upon intra-nuclear injection. To verify their efficacy, expressing cells were immunolabeled against helSyn. In control group we observed that helSyn levels increased with time, whereas asRNAs expressing cells showed a significant decrease of immunostaining levels, starting from 48h of expression, confirming protein loss. Therefore, we investigated the effect of Syn knock-down on cellular morphology. asRNA expressing cells displayed a significant reduction of neurite linear outgrowth and branching generation from newly sprouting neurites, in addition to a markedly decrease in the number and mean size of varicosities, compared to controls. Now, we are functionally characterizing the asRNA expressing cells in order to proceed to the constitutive expression of mutated Syn.