An innovative amyloid-induced neurotoxicity paradigm: investigating *in vitro* the molecular mechanisms

Background:

A wide range of human neurodegenerative diseases (e.g., Alzheimer's, Parkinson's, Prion) is associated with the misfolding and following aggregation of the involved proteins.

- In the last decade we validated **salmon Calcitonin (sCT)** as a model to study amyloid neurotoxicity. sCT displays a slower aggregation dynamics with respect to the other famous amyloid's proteins (Amyloid Beta and Tau), allowing to identify the toxic species (prefibrillar oligomers) in native state and thus to look inside the real early amyloid toxicity mechanisms.
- We recently proposed a **novel neurotoxicity paradigm,** based on the formation of amyloid pores in neuronal membranes able, *per se,* to impair synaptic plasticity and to drive the activation postsynaptic NMDA receptors, leading to the unbalancing of intracellular calcium homeostasis.

Methodologies:

- We want to go deep into the molecular mechanisms of this "**amyloid induced excitotoxicity**" performing experiments of **cellular viability, time-laps calcium imaging and patch-clamp recordings** on **cell-lines and primary neurons from the hippocampus.**

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