REGULATION OF AMPAR INTRACELLULAR TRANSPORT BY 4.1N PROTEIN

Bonnet, C.[1,2], Charpentier, J.[1,2], Mondin, M.[1,3], Choquet, D.[1,2,3] & Coussen, F.[1,2]

[1]Interdisciplinary Institute for Neuroscience and [2]Bordeaux Imaging Center
University of Bordeaux, CNRS
Bordeaux, France. E-mail : fcoussen@u-bordeaux.fr

KEY WORDS: Living cells, videomicroscopy, receptor dynamic, intracellular transport, synaptic plasticity

1. INTRODUCTION

The efficiency of excitatory synaptic transmission is determined by the number of AMPA receptors (AMPAR) localized at the post-synaptic plasma membrane. We are studying the role of post-Golgi intracellular transport of newly-synthetized AMPAR in the establishment of synaptic transmission[1].
The cytoplasmic C-terminal domain of GluA1 binds 4.1N through an interaction domain dependent on two serine phosphorylations. Using our new molecular tool together with video microscopy, we have studied the role of GluA1 / 4.1N interaction in the regulation of AMPAR transport and determined how synaptic plasticity regulates this interaction and thus GluA1 intracellular transport.

2. RESULTS

We have generated different mutants and shRNA interfering on the interaction GluA1 / 4.1N. 4.1N protein is necessary for the intracellular transport and exocytosis of GluA1. In basal conditions, the interaction GluA1 / 4.1N is only necessary for externalization of the receptor. However, during LTP this interaction regulates the exit of GluA1 from the Golgi and the speed of the vesicles containing GluA1.

3. REFERENCES

[1] Hangen, E., Cordelières, F.P., Petersen, J., Choquet, D. and Coussen, F. (2018). Neuronal activity and intracellular calcium levels regulate intracellular transport of newly synthetized AMPAR. Cell Reports, 24, 1001-1012.