Département de psychiatrie
Centre de neurosciences psychiatriques
Site de Cery
CH-1008 Prilly - Lausanne

Centre de Neurosciences Psychiatriques

CNP SEMINAR

ANNOUNCEMENT

Friday, September 18, 2015, 11:00 a.m.

"Social motivation deficits in ASDs : dissecting the role of reward system"

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Reduced function of *Shank*3, the gene encoding the excitatory synapse scaffolding protein SHANK3, is the most common monogenic cause of Autism Spectrum Disorders (ASDs). However, the mechanisms by which SHANK3 haploinsufficiency affects the neural circuits generating ASD-related behaviors such as social deficits remain elusive. Giving that activity of the Ventral Tegmental Area (VTA), could predict and encode social interactions, using *ex vivo*, *in vivo* electrophysiological recordings and behavioral experiments, we explore VTA-SHANK3 haploinsufficiency on dopamine (DA) neuron function and social behavior in mice. We show that Shank3 haploinsufficiency in VTA impairs DA neuron maturation and impacts on social motivation, while temporally precise mGluR1 modulation offers a potential ASD treatment strategy.

Selected Publications:

1) Yuan T., Mameli M., O' Connor E.C., Dey P.N., Verpelli C., Sala C., Perez-Otano I., Lüscher C. and Bellone C.: Expression of cocaine-evoked synaptic plasticity by GluN3A-containing NMDA receptors. *Neuron, 2013 Nov 20;80(4):1025-38*

2) De la Rossa A*, Bellone C*, Golding B, Vitali I, Moss J, Toni N, Lüscher C, Jabaudon D. In vivo reprogramming of circuit connectivity in postmitotic neocortical neurons. *Nat. Neurosci.2013 Feb;16(2):193-200*

3) Bellone C., Mameli M., Lüscher C. In utero exposure to cocaine delays postnatal synaptic maturation of glutamatergic transmission in the VTA. *Nat. Neurosci. 2011 Oct 2;14(11):1439-46.*

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