

BOEHM, Jannic

Téléphone 1: (514) 343-6370

Téléphone 2:

Télécopieur: (514) 343-7972

Courriel: jannic.boehm@umontreal.ca

Site Web: <http://neurosciences.umontreal.ca/recherche/les-chercheurs/jannic-boehm/>

Département de neurosciences
Université de Montréal
C.P. 6128, Succ. Centre-ville
Montréal, QC, H3C 3J7 Canada

Statut universitaire / University status

Professeur sous octroi agrégé, Département de neurosciences, Faculté de médecine, Université de Montréal

Appartenance à d'autres groupes / Affiliation with other groups

Membre régulier, Groupe de recherche sur le système nerveux central (GRSNC) du FRQS

Formation / Training

Diploma in Biology, University of Würzburg, Würzburg, Allemagne, 1996

Ph.D., University of Ulm, Ulm, Allemagne, 2001

Stage postdoctoral, Neuroscience, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY, États-Unis, 2002-2007

Orientations de la recherche

- Les changements dans la plasticité synaptique associés à la maladie d'Alzheimer
- La régulation du transport des ARNm et de la traduction de protéines pendant la plasticité synaptique

Principaux projets en cours

- Effets de la protéine précurseur de l'amyloïde sur la transmission synaptique.
- Participation des protéines argonaute dans la plasticité synaptique.

Research orientations

- Changes in synaptic plasticity during Alzheimer's disease
- Regulation of mRNA transport and protein translation during synaptic plasticity

Current research projects

- Effects of amyloid precursor protein on synaptic transmission.
- Involvement of argonaute proteins in synaptic plasticity.

Publications choisies / Selected publications

Trillaud-Doppia, E., Paradis-Isler, N. and Boehm, J. (2016). A single amino acid difference between the intracellular domains of amyloid precursor protein and amyloid-like precursor protein 2 enables induction of synaptic depression and block of long-term potentiation. *Neurobiol Dis*, 91: 94-104.

Boehm, J. (2013). A 'danse macabre': tau and Fyn in STEP with amyloid beta to facilitate induction of synaptic depression and excitotoxicity. *Eur J Neurosci*, 37 (12): 1925-30.

Tamburri, A., Dudilot, A., Licea, S., Bourgeois, C. and Boehm, J. (2013). NMDA-receptor activation but not ion flux is required for amyloid-beta induced synaptic depression. *PLoS One*, 8: e65350.

Mondragon-Rodriguez, S., Trillaud-Doppia, E., Dudilot, A., Bourgeois, C., Lauzon, M., Leclerc, N. and Boehm, J. (2012). Interaction of endogenous tau protein with synaptic proteins is regulated by N-methyl-D-aspartate receptor-dependent tau phosphorylation. *J Biol Chem*, 287: 32040-53.

Boehm, J., Kang, M. G., Johnson, R. C., Esteban, J., Huganir, R. L. and Malinow, R. (2006). Synaptic incorporation of AMPA receptors during LTP is controlled by a PKC phosphorylation site on GluR1. *Neuron*, 51: 213-25.

Hsieh, H., Boehm, J., Sato, C., Iwatsubo, T., Tomita, T., Sisodia, S. and Malinow, R. (2006). AMPAR removal underlies Abeta-induced synaptic depression and dendritic spine loss. *Neuron*, 52: 831-43.