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

Centre de Neurosciences Psychiatriques

CNP SEMINAR

ANNOUNCEMENT

Thursday, October 30, 2014, 1 p.m.

"Ca^{2+} signaling to survival and death in the CNS"

Prof Giles Hardingham

Centre for Integrative Physiology
The University of Edinburgh
Edinburgh, United Kingdom
e-mail: gharding@staffmail.ed.ac.uk

Invited by Kim Do Cuénod
(Kim.Do@chuv.ch)

Salle Escale, Hôpital Psychiatrique de Cery
Site de Cery, CH-1008 Prilly-Lausanne

NMDA receptors are a subtype of ionotropic glutamate receptor with an important role in the physiology and pathophysiology of central neurons. Inappropriate levels of Ca^{2+} influx through the NMDA receptor can contribute to neuronal loss in acute trauma such as ischemia and traumatic brain injury, as well as certain neurodegenerative disorders such as Huntington's disease. However, normal physiological patterns of synaptic NMDA receptor activity can promote neuroprotection against both apoptotic, oxidative and excitotoxic insults. As a result, NMDA receptor blockade can promote neuronal death outright or render them vulnerable to secondary trauma, depending on the nature of the activity.

Our laboratory contributes to a growing knowledge of the molecular mechanisms underlying both the neuroprotective and neurodestructive effects of Ca^{2+}, as well as the factors that determine whether signals are harmful or beneficial. The coordinated transcriptional changes that underlie Ca^{2+}-dependent neuroprotective effects will be discussed, including new investigations into Ca^{2+}-dependent transcriptional control in human stem cell-derived neurons. In addition, recent research into molecular mechanisms of Ca^{2+}-mediated excitotoxicity involving channel subunit composition and mitochondrial dysfunction will be outlined. Increased understanding in these areas of NMDAR Ca^{2+} signalling is leading to new potential therapeutic targets and strategies for excitotoxic disorders, as well as an appreciation of the harmful consequences of NMDA receptor blockade.

Selected Publications:

1. Hardingham & Bading (2010). *Nat. Rev. Neurosci.* 11, 682-696.

2. Papadia et al (2008). *Nat. Neurosci* 11, 476-487.

3. Martel et al (2012). *Neuron* 74(3):543-56.

4. Qiu et al (2013). *Nat. Comm* 4:2034.

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