SEMINAR

Topic: **Altered Neuronal Oscillations in the NMDA Hypofunction Model of Schizophrenia**

Speaker: **Bernat Kocsis, Harvard Medical School**

Time: 15:30-17:00, 22 May 2014

Venue: Room 153, Geography Building, 3663 Zhongshan Road North, Shanghai
(华东师范大学中山北路校区，地理楼 153室)

ABSTRACT OF THE TALK

NMDA receptor (NMDA-R) hypofunction is a key component of the pathomechanism of cognitive impairment in schizophrenia. NMDA-R antagonists elicit psychotic symptoms in human and schizophrenia-relevant signs in rodents, including a strong increase in gamma activity in different cortical areas. NMDA-Rs are composed of several subunits and the major differences in the distribution and dynamical properties of NMDA-Rs containing the NR2A and NR2B subunits indicate that they may play different roles in cortical network activity, and that hypofunction of these receptors may differently affect gamma synchrony, critical for a number of cognitive functions. Non-selective NMDA-R blockade induces strong aberrant gamma activity and we have shown recently that it primarily depends on NMDA-Rs containing the NR2A subunit whereas blockade of NR2B/C/D subunit-containing receptors do not have such effect. We have also demonstrated, however, a second type of gamma enhancement elicited in a state-dependent fashion during REM sleep by selective blockade of NR2B subunit-containing NMDA-Rs, at short latency. Thus, pathologic neuronal synchronization due to NR2A receptor deficiency may contribute to cognitive deficits in schizophrenia where the number of interneurons co-expressing NR2A and parvalbumin is selectively reduced. In contrast, the characteristics of the oscillations induced by NR2B-dependent mechanisms are in agreement with its demonstrated higher tolerability and the possibility of minimizing psychotomimetic side effect.

BIOGRAPHY

Bernat Kocsis, is an associate professor at Harvard Medical School. After graduation as an MD and a with diploma in Engineering he started his career studying brainstem activity in an animal model of stroke but after arriving to the US in 1988, his interest shifted to cortical and hippocampal oscillations. His primary focus was on subcortical regulation of cortical activity and the action of neuromodulatory systems and developed and/or introduced several new techniques of signal analysis into the field. He joined the Harvard faculty in 1997 and established a laboratory in the Department of Psychiatry which uses the techniques of neuronal and system level electrophysiology and neuropharmacology in animal experiments to study the mechanisms of neuronal synchronization and the role of oscillations in normal function of neural networks and their alterations in psychiatric diseases. Current projects include investigations of 1. the activity of neuronal networks in animal models of schizophrenia, 2. the effect of psychoactive compounds effective in ADHD, depression, anxiety, and schizophrenia on the electrical activity of the hippocampus and cortex, and 3. the basic mechanisms of serotonergic modulation of rhythmic synchronized forebrain activity.