Impromptu Seminar
COLLOQUIA IN CELLULAR SIGNALLING

Venue: Medical University Vienna, Center for Physiology and Pharmacology, Institute of Pharmacology, Waehringerstrasse 13a, 1090 Vienna, "**Leseraum**".

(Stefan Böhm, Tel.: (01) 40160 31200, stefan.boehm@meduniwien.ac.at, Peter Koppensteiner, Tel.: (01)40160 31220, peter.koppensteiner@meduniwien.ac.at)

Thursday 23.4.2015 14:00 s.t. **Ottavio Arancio** (host: P. Koppensteiner)

Department of Pathology and
Cell Biology, Taub Institute
Columbia University
New York, USA

"Alzheimer's disease: defeating it at the synapse"

Ottavio Arancio (oa1@columbia.edu)

Abstract: The progressive increase in Aβ levels in the brain (the amyloid hypothesis of Alzheimer's Disease) has dominated the field of the etiopathogenesis of the disease in the last 20 years. This hypothesis has been modified to account for the finding that soluble Aβ oligomers underlie subtle synaptic changes responsible for the early amnesic symptoms in the disease. However, Aβ is not the only pathological agent involved in Alzheimer's Disease etiopathogenesis. Tau, a protein found primarily associated with vesicular compartments in differentiated neurons that regulates axonal transport and microtubule stability, is also likely to play a major role. Interestingly, a number of reports have suggested a possible involvement of diffusible tau forms in the cognitive decline observed in Alzheimer's Disease patients. In this seminar, I will discuss our recent discoveries that both small soluble tau and Aβ oligomers either alone or synergistically impair synaptic plasticity and memory through an APP-dependent mechanism.