ELSC-ICNC Seminar: Edwin Rubel

March 7, 2013

On the topic of: "Regulation of Dendritic Patterning By Synaptic Activity In Binaural Brainstem Auditory Neurons"

ELSC & ICNC cordially invite you
to the lecture given by:

Edwin Rubel
University of Washington Physiology & Biophysics Department
Virginia Merrill Bloedel Hearing Research Center

On the topic of:

"Regulation of Dendritic Patterning By Synaptic Activity In Binaural Brainstem Auditory Neurons"

The lecture will be held on Thursday, March 7, 2013
at 17:00, at ELSC-ICNC: Silverman Bldg., 3rd Wing, 6th Floor, Edmond J. Safra Campus

Light refreshments at 16:45

Abstract:

The avian nucleus laminaris (NL) neurons receive segregated binaural excitatory input and compute interaural time differences essential for binaural hearing. Their unique structural and biophysical properties are conserved from reptiles to man. These properties provide a useful model to study compartment-specific dendritic regulation. Dendrites of NL neurons segregate into dorsal and ventral arborizations, receiving excitatory input from the ipsilateral and contralateral ears, respectively. Transection of NM axons to the contralateral NL leads to rapid retraction of ventral, but not dorsal, dendrites of NL neurons. To explore the extrinsic signals underlying this dendritic reorganization, we manipulated afferent inputs to NL neurons that were individually dye-filled. Blocking action potentials from one ear by either cochlea removal or temporary treatment of tetrodotoxin (TTX) led to significant retraction of affected NL dendrites within 8h, comparable in extent and time course to that induced by transection of NM axons. When the inner ear activity and hearing was allowed to recover from TTX treatments, retracted NL dendrites regrew to their normal size within 48h. Analyses of dendritic structure suggest that NL dendritic reorganization involves modification of the balance between rejections and extensions of terminal branches. These results indicate that early changes in NL dendritic structure in response to alternations in afferent inputs are not cellular events towards degeneration, but activity-dependent dynamic modulation. In vitro studies, employing dynamic imaging of dendritic branches during differential afferent stimulation conditions and multiphoton microscopy, further support the above findings and suggest that the rapid regulation of branch extension and retraction depends on differential stimulation of dendritic arbors as opposed to the absolute
afferent spike rates. I will conclude with suggestions of the intracellular pathways underlying the plasticity of dendritic arbors and observations in related parts of the normal and abnormal human brain.

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