

## Curriculum Vitae

Name: Hong Li  
Address: 51 shore Avenue  
Oyster Bay  
NY 11771  
Email: hong.li@yale.edu  
Mobile phone: 516-851-8564

### Educational background:

- 2001-2007 Ph.D degree, Neuroscience, University of Helsinki. Finland
- 1997-2000 Master degree in Science, studying human exercise physiology, Capital Physical College, Beijing, China.
- 1992-1997 Medical Doctor degree equivalent. Anhui Medical University, China.

### Awards:

- 2003: Outstanding international student in University of Helsinki.

### Research Interests:

My research interest is to study the mechanisms of brain development, synapse formation and experience-dependent neuronal plasticity. To reveal the mechanisms of brain development could not only enable us to cure neuronal developmental disorders but also pave a way to understand the mechanisms of learning and memory. I am very interested in combining molecular biology, imaging and electrophysiology methods to study how brain cells are generated and how neuronal circuitry is constructed. The questions about what genes are involved in brain development, the interactive talks between genes (proteins) and experience modified neuronal activity should be great of interest to explore.

### Research background:

My thesis work involved in studying the role of KCC2, a neuronal isoform of cation-chloride cotransporter in experience dependent neuronal plasticity and brain development. KCC2 is required for hyperpolarizing the GABA<sub>A</sub> responses. In our study we found that KCC2 expression is regulated by neuronal activity and neurotrophic factor (BDNF), indicating that KCC2 is significantly involved in the complex mechanisms of neuronal plasticity during development and pathophysiological conditions. Furthermore, I have analyzed a detail expression pattern of cation-chloride cotranporters during earlier brain development *in vivo* and *in vitro*. We found that the expression of KCC2 strictly follows neuronal maturation and correlated with synaptogenesis. The most interestingly, KCC2 is highly expressed in dendritic filopodia and spines. To understand the role of KCC2 in

the spine development, I analyzed KCC2 transgenic mice by confocal and two-photon neuronal imaging techniques. Surprisingly, we found that neurons lacking KCC2 display abnormal spine morphology and have reduced number of functional excitatory synapses. Subsequently, we discovered that the role of KCC2 in spine formation is not based on its chloride transport activity but on its intracellular protein and protein interaction. After fishing KCC2 interaction proteins by yeast-two hybrid and immunoprecipitations, we found out a cytoskeleton associated protein, 4.1N, interacts with KCC2 that underlies the mechanism of KCC2 in spine formation. The morphogenic role of KCC2 in excitatory synapses formation and its key role in GABA transmissions suggest KCC2 as a synchronizing factor in functional development of Glutamatergic and GABAergic synapses in cortical neurons and networks.

## Skills

- 1) Neuronal imaging, two photon and confocal microscopy imaging, time-lapse imaging. Familiar with imaging analysis software (Imaris), 3D reconstructing.
- 2) Neuronal dissociated and organotypic cultures.
- 3) Molecular biology: genetic methods, protein expression, purification and interaction, *in situ* hybridization, immunostaining.
- 4) Electrophysiology: Whole cell recording on cultured neurons and acute slices, extracellular recording,

## Publications:

1. **Li H.**, Khirug S, Cai C, Ludwig A, Blaesse P, Kolikova J, Afzalov R, Coleman SK, Lauri S, Airaksinen MS, Keinänen K, Khiroug L, Saarma M, Kaila K, Rivera C, KCC2 interacts with cytoskeleton to promote spine formation. *Neuron* 2007 Dec; 56(6):1019-33.
2. Cai C, **Li H**, Rivera C, Keinanen K. Interaction between SAP97 and PSD-95, two Maguk proteins involved in synaptic trafficking of AMPA receptors. *J Biol Chem.* 2006 Feb 17;281(7):4267-73.
3. Rivera C, Voipio J, Thomas-Crusells J, **Li H**, Emri Z, Sipila S, Payne JA, Minichiello L, Saarma M, Kaila K. Mechanism of activity-dependent downregulation of the neuron-specific K-Cl cotransporter KCC2. *J Neurosci.* 2004 May 12;24(19):4683-91.
4. Ludwig A, **Li H**, Saarma M, Kaila K, Rivera C. Developmental up-regulation of KCC2 in the absence of GABAergic and glutamatergic transmission. *Eur J Neurosci.* 2003 Dec;18(12):3199-206.
5. **Li H**, Tornberg J, Kaila K, Airaksinen MS, Rivera C. Patterns of cation-chloride cotransporter expression during embryonic rodent CNS development. *Eur J Neurosci.* 2002 Dec;16(12):2358-70.
6. Rivera C, **Li H**, Thomas-Crusells J, Lahtinen H, Viitanen T, Nanobashvili A, Kokaia Z, Airaksinen MS, Voipio J, Kaila K, Saarma M. BDNF-induced TrkB activation down-

regulates the K<sup>+</sup>-Cl<sup>-</sup> cotransporter KCC2 and impairs neuronal Cl<sup>-</sup> extrusion. *J Cell Biol.*  
2002 Dec 9;159(5):747-52.