17th Annual Neuroscience Center of Excellence
2006 Retreat

Keynote Speaker and Chancellor’s Award Lecturer
in Neuroscience

Huda Y. Zoghbi, M.D.
Professor of Molecular and Human Genetics,
Pediatrics, Neurology, and Neuroscience;
Investigator, Howard Hughes Medical Institute;
Member of the National Academy of Sciences;
Baylor College of Medicine, Houston, TX

Rett Syndrome and MeCP2:
Gateway to Neuropsychiatric Disorders

Rett syndrome (RTT) is a neurological disorder that disrupts postnatal development. RTT patients appear normal for 6-18 months and then lose the ability to speak and walk, and develop incessant hand-wringing motions as well as autistic features. RTT is caused by mutations in the transcriptional repressor, methyl CpG binding protein 2 (MeCP2). MeCP2 is believed to alter chromatin structure and histone acetylation by binding methylated cytosine residues and recruiting histone deacetylases to promoter sequences. Mutations in MECP2 cause not only Rett syndrome, but a variety of other disorders ranging from mild learning disability or autism in females to severe mental retardation, psychoses, or encephalopathy in males. MeCP2 is highly abundant in the brain, is expressed in neurons, and its appearance coincides with neuronal maturation. Mice genetically engineered to carry a truncating mutation (Mecp2308) develop most of the features of the human disease including stereotypic forelimb motions, balance problems, seizures, anxiety-like behaviors, and altered social interactions.

This presentation will discuss how genetic, molecular, and electrophysiology studies are beginning to provide insight about the pathogenesis of Rett syndrome and about the role of MeCP2 in postnatal brain development and synaptic plasticity. In addition, I will discuss how studying Rett pathogenesis could provide insight into several neuropsychiatric disorders.

Deadline for abstract and registration submission is May 8, 2006
Contact Melissa Musacchia at 599-0831 or e-mail: mmusac1@lsuhsc.edu