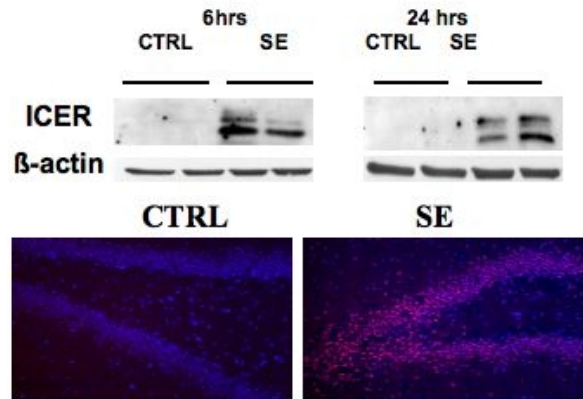


The Brooks - Kayal Lab

Research in the Brooks-Kayal Lab focuses on understanding the molecular and cellular mechanisms that result in development of epilepsy in order to develop new ways to prevent and treat this disorder. The lab uses a variety of molecular, neurophysiological and histological techniques and animal models of epilepsy. Current studies focus on the role of signal transduction pathways, including CREB, JAK/STAT, BDNF and Egr on GABA(A) receptor expression and trafficking. The lab also studies the effects of early-life seizure activity and seizure treatment on brain development.

Dr. Amy Brooks-Kayal is the Chief of Child Neurology and the Ponzio Family Chair in Pediatric Neurology. Dr. Brooks-Kayal came to the University of Colorado Denver in July 2008 from The Children's Hospital of Philadelphia (CHOP) and University of Pennsylvania (Penn) School of Medicine. She is internationally recognized for clinical care and research in epilepsy, and is a member of the Board of Directors of the American Epilepsy Society and a previous editor of *Epilepsia*, the journal of the International League against Epilepsy.



Lund et al, *Science Signaling* 2008

Lab Personnel

Amy Brooks-Kayal, M.D. (Professor)
Yogendra Raol, Ph.D. (Assistant Professor)
Marco Gonzalez, PhD. (Instructor)
Heidi Grabenstatter (Postdoctoral Fellow)
Yasmin Cruz (PRA)
Lauren Goldstein (PRA)
Dusty Christian, (Staff Assistant)
James Simpson (Finance Administrator)

Location

University of Colorado Denver- Anschutz Medical Campus
Research Complex II, Room 4122
12700 E 19th Avenue, MS 8605
Aurora, CO 80045

Contact Us

Phone 303-724-4253
Fax 303-724-4254

Amy.Brooks-Kayal@ucdenver.edu

Current NIH Funded Studies

Research Studies Molecular Determinants of GABAR Gene Regulation

Co-PI: Amy Brooks-Kayal, MD
Source: NIH R01; NINDS
Amount: \$242,578
Term 04/06-03/11

GABA(A) Receptor Subunit Regulation in Epileptogenesis

PI, Amy Brooks-Kayal, MD
Source: NIH R01; NINDS
Amount: \$235,350
Term: 12/05-12/09

GABA(A) Receptors in Developmental Epileptogenesis

PI, Amy Brooks-Kayal, MD
Source: NIH R01; NINDS
Amount: \$180,653
Term: 12/04-11/09

Recent Publications

2006

Frye CA, Rhodes ME, Raol YH, Brooks-Kayal AR. Early postnatal stimulation alters pregnane neurosteroids in the hippocampus. *Psychopharmacology (Berl)*. 2006 Jun; 186(3):343-50. [PMID: 16491430]

Porter BE, Cui XN, Brooks-Kayal AR. Status epilepticus differentially alters AMPA and kainate receptor subunit expression in mature and immature dentate granule neurons. *Eur J Neurosci*. 2006 Jun;23(11):2857-63. [PMID: 16819974]

Raol YH, Lund IV, Bandyopadhyay S, Zhang G, Roberts DS, Wolfe JH, Russek SJ, Brooks-Kayal AR. Enhancing GABA(A) receptor alpha 1 subunit levels in hippocampal dentate gyrus inhibits epilepsy development in an animal model of temporal lobe epilepsy. *J Neurosci*. 2006 Nov 1;26(44):11342-6. [PMID: 17079662]

Raol YH, Zhang G, Lund IV, Porter BE, Maronski MA, Brooks-Kayal AR. Increased GABA(A)-receptor alpha1-subunit expression in hippocampal dentate gyrus after early-life status epilepticus. *Epilepsia*. 2006 Oct;47(10):1665-73. [PMID: 17054689]

Roberts DS, Hu Y, Lund IV, Brooks-Kayal AR, Russek SJ. Brain-derived neurotrophic factor (BDNF)-induced synthesis of early growth response factor 3 (Egr3) controls the levels of type A GABA receptor alpha 4 subunits in hippocampal neurons. *J Biol Chem*. 2006 Oct 6;281(40):29431-5. [PMID: 16901909]

Marsh E, Brooks-Kayal AR, Porter B: Seizures and Antiepileptic Drugs: Does Exposure Alter Normal Brain Development? *Epilepsia* 47(12): 1999-2010, 2006.

2008

Marsh ED, Minarcik J, Campbell K, Brooks-Kayal AR, Golden JA: FACS-array gene expression analysis during early development of mouse telencephalic interneurons. [Dev Neurobiol](#). 68(4):434-45, 2008.

Hu Y, Lund IV, Gravielle M, Farb DH, Brooks-Kayal AR, Russek SJ: Surface Expression of GABA-A Receptors is transcriptionally controlled by the interplay of CREB and its Binding

Partner ICER. *J. Biol Chem*, 283(14):9328-9340, 2008.

Lund IV, Hu Y, Raol YSH, Benham RS, Faris R, Russek SJ, Brooks-Kayal AR: BDNF selectively regulates GABA-A receptor transcription by activation of the JAK/STAT pathway, *Science Signaling*, 1(41ra9), 2008.

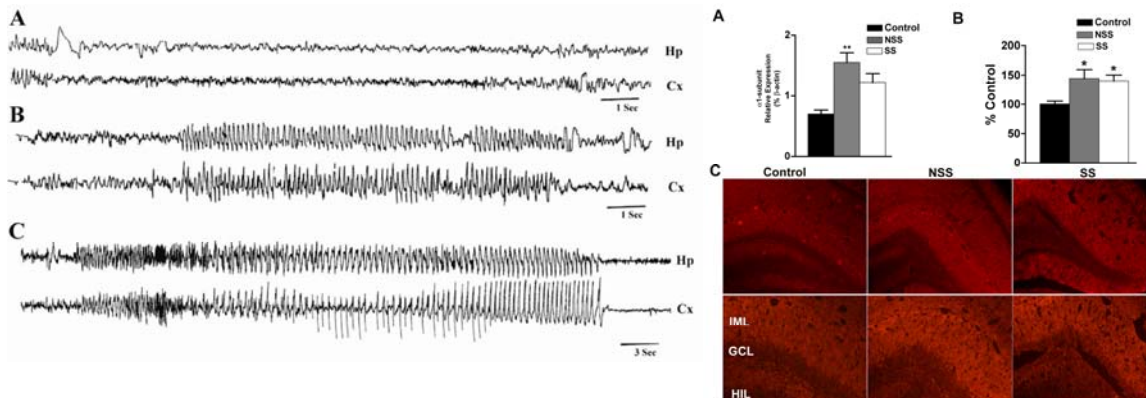
2009

Raol YSH, Lapides DA, Keating J, Brooks-Kayal AR, Cooper EC: A KCNQ channel opener for neonatal seizures and status epilepticus, *Ann Neurol*, 2009 Mar;65(3):326-36.

Jacobs MP, Leblanc GG, Brooks-Kayal AR, Jensen FE, Joebels JL, Spencer DD, Swann JW: Curing Epilepsy: Progress and Future Directions. *Epilepsy and Behav.*, 2009 Mar;14(3):438-45.

Brooks-Kayal AR., Raol Y., Russek SJ: Alteration of Epileptogenesis Genes, *Neurotherapeutics*, 2009 Apr;6(2):312.

Marsh E, Fulp C, Gomez E, Nasrallah I, Minarcik J, Sudi J, Christian SL, Mancini G, Labosky P, Dobyns W, Brooks-Kayal AR, Golden JA. Targeted loss of Arx results in a developmental epilepsy mouse model and recapitulates the human phenotype in heterozygous females. *Brain*. 2009 Jun;132(Pt 6):1563-76.



Translational Epilepsy Research Program

The Translational Epilepsy Research Program formed jointly between the School of Medicine and the School of Pharmacy brings together expertise of researchers in molecular biology, pharmacology, neurochemistry, cellular and in vivo physiology and animal models of epilepsy. This research program includes members from the School of Pharmacy and members of the Departments of Pediatrics and Neurology in the School of Medicine including Drs. Manisha Patel, Amy Brooks-Kayal, Tim Benke, Andy White, Lauren Frey, Yogendra Raol, Marco Gonzalez and Audrey Yee. This state of the art trans-school program provides shared research infrastructure needed by multiple investigators (ie., core facilities for rodent EEG monitoring, cell and tissue culture, microscopy/imaging, electrophysiology, cognitive and behavioral testing, and molecular biology), and provides an essential bridge between the University's strengths in basic neuroscience, medicinal chemistry and targeted therapeutic compound development and its strong clinical programs to facilitate more rapid development and translation of new therapies for neurological disease from target identification to pre-clinical development to clinical trials. The Rodent Neurophysiology Core also benefits all members of the University research community working in rodent models of neurological disease, providing state-of-the-art neurophysiological (EEG, Sleep) monitoring for models of CNS disorders, including head trauma, stroke, neurodevelopmental and neurodegenerative diseases as well epilepsy.