Bruce Lyeth, Ph.D.

Clinical Interests

Dr. Lyeth's research interests focus on the pathophysiology of traumatic brain injury, excitotoxic cascades affecting signal transduction and behavior, and the development of therapeutic interventions for brain injury. Dr. Lyeth's laboratory uses a variety of pharmacological, behavioral and neuroanatomical methods to investigate the neural mechanisms involved in traumatic brain injury pathology. The ultimate goal of this research program is to develop novel clinical therapeutic strategies targeted at reducing the debilitating consequences of traumatic brain injury in patients. Pre-clinical testing of novel therapeutic strategies and compounds are routinely performed in this laboratory. Previous efforts have led to ongoing clinical trials evaluating hypothermia and glutamate receptor antagonists as therapeutic interventions for traumatic brain injury patients. Dr. Lyeth is investigating the role of early astrocyte damage following TBI and the consequences on neuronal cell death, the involvement of the neuropeptide NAAG in brain injury , and the role of HDAC inhibitors in modulating excitotoxicity.

Title

Professor IR

Specialty

Neurological Surgery

Department

Neurological Surgery

Division

Neurological Surgery

Education

Ph.D., Virginia Commonwealth University, Richmond, Virginia, 1986
B.A., Christopher Newport College, Newport News, Virginia, 1974
M.S., Radford College, Radford, Virginia, 1976

Fellowships

Medical College of Virginia, Richmond, Virginia, 1986-1987

Professional Memberships

Editorial Board, Journal of Neurotrauma
Member, Executive Council National Neurotrauma Society
UCLA Brain Injury Research Center Advisory Board

Honors and Awards

Outstanding Experimental Psychology Graduate Student Award, 1996

Select Recent Publications

Feng JF, Zhao X, Gurkoff GG, Van KC, Shahlaie K, Lyeth BG. Post-traumatic hypoxia exacerbates neuronal cell death in the hippocampus. *J Neurotrauma.* 2012 Apr 10;29(6):1167-79. Epub 2012 Jan 30.

Gurkoff GG, Shahlaie K, Lyeth BG. In vitro mechanical strain trauma alters neuronal calcium

responses: Implications for posttraumatic epilepsy. *Epilepsia*. 2012 Jun;53 Suppl 1:53-60. doi: 10.1111/j.1528-1167.2012.03475.x.

Beller JA, Gurkoff GG, Berman RF, Lyeth BG. Pharmacological Enhancement of Glutamate Transport Reduces Excitotoxicity in vitro. Restorative Neurology Neuroscience 29:331-346, 2011.

Feng J-F, Van KC, Gurkoff GG, Kopriva C, Olszewski RT, Song M, Sun S, Xu, M, Neale, JH, Yuen P-W, Lowe DA, Zhou J, Lyeth BG. Post-injury administration of NAAG Peptidase Inhibitor prodrug, PGI-02776, in experimental TBI. Brain Research 1395: 62-73, 2011.

Harley, W., Floyd, C., Dunn, T., Zhang, X.D., Chen, T.Y., Hegde, M., Palandoken, H., Nantz, M. H., Leon, L., Carraway, K.L., 3rd, Lyeth, B., and Gorin, F.A. Dual inhibition of sodium-mediated proton and calcium efflux triggers non-apoptotic cell death in malignant gliomas. Brain Res. 1363: 159-169, 2010.

Schwartzkroin PA, Wenzel HJ, Lyeth BG, Poon CC, DeLance A, Van KC, Campos L, Nguyen DV. Does ketogenic diet alter seizure sensitivity and cell loss following fluid percussion injury? Epilepsy Research 92(1): 74-84, 2010.

Shahlaie K, Lyeth BG, Gurkoff GG, Muizelaar JP, Berman RF. Neuroprotective Effects of Selective N-Type VGCC Blockade on Stretch-Injury Induced Calcium Dynamics in Cortical Neurons. J Neurotrauma 25(1):175-187, 2010.

Fedor M, Berman RF, Muizelaar JP, Lyeth BG. Hippocampal Theta Dysfunction after Lateral Fluid Percussion Injury. J Neurotrauma 27(9):1605-1615, 2010.

Zhang B, West EJ, Van KC, Gurkoff GG, Zhou, J, Zhang X-M, Kozikowski AP, Lyeth BG.. HDAC inhibitor increases histone H3 acetylation and reduces microglia inflammatory response following traumatic brain injury in rats. Brain Research, 1226: 181-191, 2008.

Zhao X, Gorin FA, Berman RF, Lyeth BG. Differential hippocampal protection when blocking intracellular sodium and calcium entry during traumatic brain injury in rats. J Neurotrauma, 25 (10): 1195-205, 2008.

Cox CD, West EJ, Liu MC, Wang KK, Hayes RL, Lyeth BG. Dicyclomine, an M1 muscarinic antagonist, reduces biomarker levels, but not neuronal degeneration, in fluid percussion brain injury. J Neurotrauma, 25(11): 1355-65, 2008.

Di Giorgio AM, Hou Y, Zhao X, Zhang B, Lyeth BG, Russell MJ. Dimethyl sulfoxide provides neuroprotection in a traumatic brain injury model. Restor Neurol Neurosci, 26(6): 501-7, 2008.

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