BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
McLaughlin, BethAnn	Assistant Professor
eRA COMMONS USER NAME (credential, e.g., agency login) MCLAUGBM	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
Skidmore College, Saratoga Springs, NY	BA	1990	Biopsychology
University of Pennsylvania, Philadelphia, PA	PhD	1997	Neurological Sciences

A. Positions and Honors

Positions and Employment

1990-1992 Research Technician, Children's Seashore House, Division of Neuroscience, Philadelphia, PA
1997-1998 Postdoctoral Fellow, Department of Neurobiology, University of Pittsburgh
1998-2002 Research Assistant Fellow, Department of Neurobiology, University of Pittsburgh
2002-2005 Research Assistant Professor, Department of Pharmacology, Vanderbilt University School of Medicine, Nashville, TN
2003-pres Investigator, Vanderbilt Kennedy Center for Research on Human Development, Nashville, TN
2005-pres Assistant Professor, Department of Neurology, Vanderbilt University School of Medicine, Nashville, TN

Honors

1986-1990 Dean's List Scholar, Skidmore College
1997-1998 National Research Service Award Postdoctoral Training Fellowship in Neurodegeneration (NINDS)

Other Experience and Professional Memberships

Member: American Heart Association, New York Academy of Science, Society of Neuroscience and American Society of Cell Biology
1999 Chairman, Society for Neuroscience annual meeting Neurotoxicity Social: *Role of Protein Aggregates in Neurodegenerative Disease*
2003 Research into Aging Research Grant Advisory Review Panel
2005-pres Ad hoc reviewer: Neuron, Cell, Journal of Neuroscience, Journal of Neurochemistry, Molecular Pharmacology, Neurochemistry International, Molecular Toxicology, Epilepsy
2005-2008 Faculty Director of Community and Special Projects, Vanderbilt Kennedy Center
2006-2007 *Most Frequent Reviewers* Journal of Neuroscience
2007-2010 American Heart Association Scientific Council Member (Functional Genomics and Translational Biology)
2008 Autism Speaks Ad Hoc Grant Review Committee: Environmental Science in Autism
2008 Chairman, Marino Autism Research Institute, Scientific Symposium *Environment and Autism Etiology* Vanderbilt University
2008 Chairman, Vanderbilt Kennedy Center Strategic Planning Membership Committee
2008 Scientific Advisory Board, Burke Cornell Rehabilitation Hospital
2008 Ad hoc Reviewer, American Institute of Biological Sciences Study Section

B. Selected peer-reviewed publications (in chronological order)

Batshaw, M. L., Yudkoff, M., McLaughlin, B. A., Gorry, E., Anegawa, N. J. Smith, L. A. S., Hyman, S. L., & Robinson, M. B. (1995). The sparse fur mouse as a model for gene therapy in ornithine carbamoyltransferase deficiency. *Gene Therapy, 2*, 1-7.

Eberwine, J. W., & McLaughlin, B. A. (1995). Striatal RNA-binding proteins interact with huntingtin mRNA. In M. A. Ariano & D. J. Surmeier (Eds.), *Molecular and cellular mechanisms of neostriatal function*, pp. 143-149.

Robinson, M. B., Hopkins, K., Batshaw, M. L., McLaughlin, B. A., Heyes, M. P., & Oster-Granite, M. L. (1995). Evidence for excitoxicity in the brain of the ornithine carbamoyltransferase deficent sparse for mouse. *Developmental Brain Research, 90*(1-2), 35-44.

McLaughlin, B. A., Spencer, C., & Eberwine, J. (1996). CAG trinucleotide repeats interact with RNA binding proteins. *American Journal of Human Genetics, 59*(3), 561-569.

McLaughlin, B. A., Nelson, D., Silver, I. A., Erecinska, M., & Chesselet, M. F. (1998). Methylmalonate toxicity in primary neuronal cultures. *Neuroscience, 86*(1), 279-290.

McLaughlin, B. A., Nelson, D., Erecinska, M., & Chesselet, M. F. (1998). Toxicity of dopamine to striatal neurons in vitro and potentiation of cell death by a mitochondrial inhibitor. *Journal of Neurochemistry, 70*(6), 2406-2415.

McLaughlin, B. A., & Levitt, P. (1999). Molecular basis of neurological disease. In M. T. T. Wong-Riley (Ed.), *Neuroscience secrets*, pp. 349-356. Philadelphia: Hanley & Belfus, Inc.

Hoyt, K. R., McLaughlin, B.A., Higgins, D. S., Jr., & Reynolds, I. J. (2000). Inhibition of glutamate-induced mitochondrial depolarization by tamoxifen in cultured neurons. *Journal of Pharmacology and Experimental Therapeutics, 293*(2), 480-486.

Aizenman, E., Stout, A. K., Hartnett, K. A., Dineley, K. E., McLaughlin, B. A., & Reynolds, I. J. (2000). Induction of neuronal apoptosis by thiol oxidation: Putative role of intracellular zinc release. *Journal of Neurochemistry*, 75 (5), 1878-1888.

McLaughlin, B.A. (2001). Dopamine neurotoxicity and neurodegeneration. In M. F. Chesselet (Ed.), *Molecular mechanisms of cell death*, pp. 195-231. Towowa, NJ: Humana Press.

Nutall, M. E., Lee D., McLaughlin B. A., Erhardt J. A. (2001) Selective inhibitors of apoptotic caspases: Implications for novel therapeutic approaches. *Drug Discovery Today*, 6, 85-91.

McLaughlin, B. A., Pal, S., Tran, M. P., Parsons, A. A., Barone, F., Erhardt, J. A., & Aizenman, E. (2001). p38 activation is required upstream of potassium current enhancement and caspase cleavage in oxidant-induced neuronal apoptosis. *Journal of Neuroscience*, 21, 3303-3311.

Leszkiewicz, D. N., McLaughlin, B. A., & Aizenman, E. (2002). Protein kinases and light: Unlikely partners in a receptor localization puzzle. *Physiology & Behavior*, 77, 533-536.

Legos, J. J., McLaughlin, B. A., Skaper, S. D., Strijbos, P. J. M. L., Herin, G. A., Parsons, A. A., Aizenman, E., Barone, F. C., & Erhardt, J. A. (2002). The selective p38 MAP kinase inhibitor SB239063 protects rat hippocampal and primary cortical neurons from excitotoxic death. *European Journal of Pharmacology* 447, 37-42.

Du, S., McLaughlin, B. A., Pal, S., & Aizenman, E. (2002). In vitro neurotoxicity of methylisothiazolinone, a commonly used industrial and household biocide, proceeds via a zinc and mitogen-activated protein kinase-dependent pathway. *Journal of Neuroscience*, 22, 7408-7416.

McLaughlin, B. A., Erhardt, J. A., Legos, J. J., White, R., Barone, F. C., Parsons, A. A., & Aizenman, E. (2003). Caspase activation is required for neuroprotection in ischemic preconditioning. *Proceedings in the National Academy of Sciences USA*, 100, 715-720.

McLaughlin, B. A. (2004). The kinder side of killer proteases: Caspase activation contributes to neuroprotection and CNS remodeling. *Apoptosis*, 9, 111-121.

DiNapoli, M. & McLaughlin B. A. (2005). Role of the ubiquitin proteasome pathway in stroke and transient ischemic attacks - therapeutic opportunities for providing neuroprotection. *Current Opinions in Investigational Drugs*. 6, 686-699.

Milne, G. L., Musiek, E. S., Zanoni, G., Vidari, G., McLaughlin, B., Morrow, J. D. (2005) Development of a high performance liquid chromatography-electrospray ionization tandem mass spectrometric (LC/ESI/MS/MS) assay to quantify highly reactive cyclopentenone eicosanoids in vivo. *Free Radical Biology and Medicine* 39, 113.

Musiek, E., Milne, G. I., McLaughlin, B. A. & Morrow, J.D. (2005). Cyclopentenone eicosanoids as mediators of neurodegeneration: A pathogenic mechanism of oxidative stress and cyclooxyanese-mediated neurotoxicity. *Brain Pathology* 15: 149-158.

Musiek, E.M., Breeding, R.A., Milne G., Morrow, J. and McLaughlin B.A. (2006) Cyclopentenone Isoprostanes Are Novel Bioactive Products Of Lipid Oxidation Which Enhance Neurodegeneration. *Journal of Neurochemistry* 97, 1301-1313.

Milne, G. L., Musiek, E. S., Zanoni, G., Vidari, G., McLaughlin, B., Morrow, J. D.(2006) Development of a novel liquid chromatography-mass spectrometric assay to measure formation of highly reactive cyclopentenone isoprostanes in vivo *Clinical Pharmacology & Therapeutics* 79, 36.

Musiek, E.M, McLaughlin, B.A., and Morrow, J.D. (2007) Electrophilic cyclopentenone isoprostanes in neurodegeneration. *Journal of Molecular Neuroscience* 33:80–86.

O'Duffy, A.E., Bordelon, Y.M. and McLaughlin, B.A. (2007) Killer proteases and little strokes—how the things that do not kill you make you stronger. *Journal of Cerebral Blood Flow and Metabolism* 1-14.

Altay, T, McLaughlin, B.A., Wu, J.Y., Park, T.S. and Gidday, J. (2007) Slit modulates cerebrovascular inflammation and mediates neuroprotection against global cerebral ischemia. *Experimental Neurology* 207 186–194.

Musiek, E.M, Brooks, J.D., Brunoldi, E., Porta, A. Joo, M., Han, W., McLaughlin, B.A., Zanoni, G., Vidari, G., Blackwell, T.S., Milne, G.L., McLaughlin, B.A. and Morrow, J.D. Electrophilic Cyclopentenone Neuroprostanes are Anti-inflammatory Mediators Formed from the Peroxidation of the Omega-3 Polyunsaturated Fatty Acid Docosahexaenoic Acid. *Journal of Biological Chemistry*. (*In press).*

Kirshner, H. and McLaughlin, B.A. Wagging the dog- Moving closer to features defined by basic scientists, the protection of prodromal transient ischemic attacks reveals itself. *European Journal of Neurology*. *(In press).*

C. Research Support

Ongoing Research Support

R01 NS 050396-01 McLaughlin (PI) 12/01/04 - 11/30/09
NIH/NINDS
Cellular Mechanisms of Preconditioning Neuroprotection
The goal of this translational research program is to develop an in-depth understanding of the events that protect neurons from stroke in order to improve neuroprotective therapies.
Role: PI

R01 DK 48831-10 Morrow (PI) 04/01/04 - 03/31/09
NIH/NIDDK
Biochemistry and Pharmacology of D_2/E_2 Isoprostanes
The goal of this research program is to identify the cellular targets of prostaglandin-like compounds, termed isoprostanes, produced in vivo by the free radical-mediated peroxidation of arachidonic acid. These compounds possess potent biological activity and thus may serve as mediators of oxidant injury.
Role: Investigator

R01 GM051366 Wadzinski (PI) 09/1/08-08/31/13
NIH/NIGMS
Structure and function of protein kinase/PP2A complexes
This program is designed to study the composition and function of the protein phosphatase 2A signaling complex which is essential for neural cell growth, survival and signaling. The components that alter complex formation allowing for discreet signaling will be evaluated in this grant particularly as they relate to pathophysiologically relevant neurological injury.
Role: Investigator

Completed Research Support

None