BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Gerry S. Oxford, Ph.D	Director, Stark Neurosciences Research Institute Professor, Pharmacology & Toxicology
eRA COMMONS USER NAME (credential, e.g., agency login) goxford	

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
Elon College (Elon, NC)	B.A.	1969	Biology & Chemistry
Emory University (Atlanta, GA)	Ph.D.	1974	Physiology
Duke University (Durham, NC)	Postdoc	1974-76	Pharmacology

A. Personal Statement

I have over 40 years of experience in electrophysiological investigations of ion channels and receptors and have been supported for most of that time via NIH grants, both R01 and P01 support. In particular, my laboratory has worked and published studies on isolated sensory neurons since 1985, contributing to our understanding of bradykinin and capsaicin responses in nociceptors. Specifically, my lab has contributed significantly to our understanding of the structure-function parameters and modulation of TRPV1 and TRPA1 by various signaling cascades. On another front, my lab is one of the foremost in the study of "functional selectivity" of dopamine receptors, a phenomenon by which a single receptor isoform can selectively direct signaling to downstream pathways dependent upon the agonist. On the technical front, I have personal experience not only in electrophysiology, but also in live cell imaging and microscopy. I have a proven track record of managing research projects in the general area of ion channel biology and executing the aims and design of associated NIH funding.

In addition, I have extensive experience in mentoring and graduate education having directed the UNC Neurobiology Curriculum for 15 years, served as President of the Association of Neuroscience Departments and Programs, and been the PD or 6 NIH training grants.

B. Positions and Honors

Positions and Employment

1976-1982	Assistant Professor of Physiology	University of North Carolina at Chapel Hill
1982-1988	Associate Professor of Physiology	University of North Carolina at Chapel Hill
1988-2003	Professor of Physiology	University of North Carolina at Chapel Hill
2003-present	Professor of Pharmacology & Toxicology	Indiana University School of Medicine
2003-present	Executive Director, SNRI	Indiana University School of Medicine

Honors and Awards, Advisory, Editorial, and Review Activities:

1984-1988	Member, Physiology Study Section (IRG)
1985-1999	Editorial Board, Journal of General Physiology
1991-1995	Editorial Board, Endocrine Reviews
1993-1999	Editorial Board, Molecular Pharmacology
1996	President, Society of General Physiologists
1998 – 2001	Distinguished University Professor of Teaching, UNC-Chapel Hill
2003 - present	Executive Director, Stark Neurosciences Research Institute
2005	President, Association of Neuroscience Departments and Programs

C. Selected peer-Reviewed publications or manuscripts in press (chronological order)

McGehee, D.S. and **Oxford, G.S.** Bradykinin modulates the electrophysiology of cultured rat sensory neurons through a Pertussis toxin-insensitive G protein. *Molecular and Cellular Neurosciences* 2:21-30, 1991.

Naruse, K., McGehee, D.S., and **Oxford, G.S.** Differential responses of Ca-activated K channels to bradykinin in sensory neurons and a neuronal cell line. *American Journal of Physiology* 262:C453-C460, 1992.

McGehee, D.S., Goy, M.F., and **Oxford, G.S.** Involvement of the nitric oxide - cyclic GMP pathway in the desensitization of bradykinin responses of cultured rat sensory neurons. *Neuron* 9:315-324, 1992.

Koplas, P.A., Rosenberg, R.L., and **Oxford, G.S**. The role of calcium in the desensitization of capsaicin responses in rat dorsal root ganglion neurons. *J. Neuroscience* 17:, 3525-3537 1997.

Kuzhikandathil, E.V., Wang, H., Morozova, N., Szabo, T., Blumberg, P., and **Oxford, G.S**. Functional analysis of capsaicin receptor (VR1) multimerization and agonist responsiveness using a dominant negative mutation. *J. Neuroscience* 21:8697-8706, 2001.

Bhave, G., Zhu W., Wang, H., Brasier, D.J., **Oxford, G.S**., and Gereau, RW. CAMP-dependent protein kinase regulates desensitization of the capsaicin receptor (VR1) by direct phosphorylation. *Neuron* 35:721-731, 2002

Bhave, G., Hu H.J., Glauner, K.S., Zhu, W., Wang, H., Brasier, D.J., **Oxford, G.S**., and Gereau, R.W. Protein kinase C phosphorylation sensitizes but does not activate the capsaicin receptor TRPV1. *P.N.A.S*. 100:12480-12485, 2003.

Liu, L., Zhu, W., Zhang, Z-S, Yang, T., **Oxford, G**, Grant, A., and Simon, S. A. Nicotine inhibits voltage-dependent sodium and potassium channels and sensitizes vanilloid receptors. *J. Neurophysiology* 91:1482-1491, 2004.

Zhu, W., Galoyan, S.M., Petruska, J.C., **Oxford, G.S**. and Mendell, L.M. A developmental switch in acute sensitization of small dorsal root ganglion (DRG) neurons to capsaicin or noxious heating by NGF. *J. Neurophysiology* 92:3148-3152, 2004.

Zhu, W. and **Oxford, G.S**. Phosphoinositide-3-kinase and mitogen activated protein kinase signaling pathways mediate acute NGF sensitization of TRPV1. *Mol. Cell. Neuroscience* 34:689-700, 2007.

Zhu, W., Xu, P., Cuascut, F.X.**, Oxford, G.S**. and Hall, A.K. Activin acutely sensitizes dorsal root ganglion neurons and induces hyperalgesia via PKC-mediated potentiation of TRPV1. *J. Neuroscience* 27:13770-13780, 2007.

Butterworth J, **Oxford GS**. Local anesthetics: a new hydrophilic pathway for the drug-receptor reaction. *Anesthesiology*. 111(1):12-14, 2009.

Kunkler PE, Ballard CJ, **Oxford GS**, Hurley JH. TRPA1 receptors mediate environmental irritant-induced meningeal vasodilatation. *Pain* 152(1):38-44, 2011.

Brittain JM, Duarte DB, Wilson SM, Zhu W, Ballard C, Johnson PL, Liu N, Xiong W, Ripsch MS, Wang Y, Fehrenbacher JC, Fitz SD, Khanna M, Park CK, Schmutzler BS, Cheon BM, Due MR, Brustovetsky T, Ashpole NM, Hudmon A, Meroueh SO, Hingtgen CM, Brustovetsky N, Ji RR, Hurley JH, Jin X, Shekhar A, Xu XM, **Oxford GS**, Vasko MR, White FA, Khanna R. Suppression of inflammatory and neuropathic pain by uncoupling CRMP-2 from the presynaptic Ca^{2+} channel complex. *Nature Medicine* 17(7):822-829, 2011.

Zhu W, **Oxford GS**. Differential gene expression of neonatal and adult DRG neurons correlates with the differential sensitization of TRPV1 responses to nerve growth factor. *Neurosci Lett*. 500(3):192-196, 2011.

Molosh AI, Sajdyk TJ, Truitt WA, Zhu W, **Oxford GS**, Shekhar A. NPY Y1 receptors enhance overall inhibition of basolateral amygdala neurons by differentially modulating GABAA and NMDA receptors through divergent signal transduction pathways. *Neuropsychopharmacology* 38(7):1352-1364, 2013.

Oxford GS and Hurley JH. The role of TRP channels in migraine. *Open Pain Journal* 6:37-49, 2013.

Kunkler PE, Ballard CJ, Pellman JJ, Zhang LJ, **Oxford GS**, Hurley JH. Intraganglionic signaling as a novel nasal-meningeal pathway for TRPA1-dependent trigeminovascular activation by inhaled environmental irritants. *PLoS ONE* 9:e103086, 2014.

Zhu W, Vuppalanchi D, Oxford GS. Artemin sensitizes TRPV1 through a Ret-PLC-PKCδ signaling pathway. *Molecular Pharmacology* (under revision)

D. Research Support

Active

R01 GM086544-01A1 10/01/09 – 09/30/14 PI: Oxford

“Ligand Directed Functional Selectivity of G-Protein Coupled Receptor Signaling”

The proposed research addresses the molecular level mechanisms underlying the phenomenon of functional selectivity whereby different signaling pathways from a single GPCR isoform are preferentially activated by different agonist ligands of the receptor. The study employs electrophysiology and biochemical measurements of signaling between dopamine receptors and ion channels and enzymes in AtT20 neuroendocrine cells and neurons from the nucleus accumbens. Molecular events will be examined using mutant receptors and G-protein alpha subunits to distinguish functional selectivity.

R01 ES017430-01A1 04/01/10 – 3/31/14 PI: Oxford / Hurley (MPI)

“Role of TRP Channels in Environmental Irritant-Induced Headache”

The goals of this research program are to test the involvement of TRPA1 and TRPV1 receptors in the acute induction of environmental irritant-induced headache due to air pollutants. The project combines Laser Doppler flowmetry measurements of cerebral blood flow as a surrogate for headache, electrophysiology of native and cloned TRP receptors, and assessment of CGRP release from cerebral vascular beds. The central goal is to determine whether TRPA1 is involved in the phenomenon.