BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Jian Luo	Instructor
eRA COMMONS USER NAME (credential, e.g., agency login)	
LUO.JIAN	

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
Hunan Medical University, Hunan, China	B.M./M.D	1985-1990	Medicine
Hunan Medical University, Hunan, China	M.S.	1990-1993	Medicine
Purdue University, West Lafayette, IN, USA	Ph.D.	2000-2003	Neuroscience
Stanford University	Postdotoral	2004-2010	Neurology and Neurological Sciences

A. Personal Statement

My career in biomedical research thus far has been comprised of a rich set of experiences attained from my involvement in diverse scientific environments spanning from China to the United States. My interest in pursuing a research career began when I started my medical training in China. When I was working in a teaching hospital as a physician, I was drawn to research and spent progressively more time in the laboratory. Concurrently, I received several research grants including one from the National Natural Science Foundation of China. These funding opportunities further stimulated my passion for research at which point I made the decision to dedicate my career entirely to research and immigrated to the United States to further pursue my research career in 1999. Since then, my main research interests have focused on central nervous system (CNS) injury and disease. While pursuing PhD at Purdue, my dissertation work was mainly on traumatic spinal cord injury, studying the mechanisms of secondary injury leading to cell death and functional deficits. At Stanford, my postdoctoral research on transforming growth factor β (TGF-β) and macrophage colony-stimulating factor (M-CSF) signaling includes a variety of animal models of brain injury and neurodegenerative diseases, such as excitotoxicity, traumatic brain injury, Alzheimer's disease, multiple sclerosis and Parkinson's disease.

B. Positions and Honors

Positions and Employment

2010- **Instructor**, Stanford University, Stanford, CA, USA.
2004-2010 **Postdoctoral Scholar**, Stanford University, Stanford, CA, USA.
2000-2003 **Graduate Student**, Purdue University, West Lafayette, IN, USA.
1996-1999 **Attending Doctor**, Xiangya Hospital of Hunan Medical University, Hunan, China.
1990-1996 **Resident Doctor**, Xiangya Hospital of Hunan Medical University, Hunan, China.
1990-1993 **Graduate Student,** Hunan Medical University, Hunan, China.

Other Experience and Professional Memberships

2009- Member, Institutional Animal Care and Use Committee (IACUC), VA Palo Alto Heath Care System, Palo Alto, CA.
2009 Program Committee Member, The 2nd International Congress on Image and Signal Processing (CISP'09) and The 2nd International Conference on BioMedical Engineering and Informatics (BMEI'09), October 2009, Tianjin, China.
2007- The Society for Molecular Imaging
2000- The Society for Neuroscience

Honors

2009 Alzheimer's Association Young Scientist Award.
2007 Stanford Immunology Postdoc Travel Fellowship.
2007 Academy of Molecular Imaging Travel Award.
2004 Purdue University Pfizer Graduate Student Research Award, IN, USA.

C. Peer-reviewed Publications

Luo J, Borgens RB and Shi R. Polyethylene glycol immediately repairs neuronal membranes and inhibits free radical production after acute spinal cord injury. *J Neurochem* 83: 471-80, 2002.

Shi R, **Luo J** and Peasley MA. Acrolein inflicts axonal membrane disruption and conduction loss in isolated guinea pig spinal cord. *Neuroscience* 115:337-40, 2002.

Luo J, Li N, Robinson JP and Shi R. Detection of reactive oxygen species by flow cytometry after spinal cord injury. *J Neurosci Methods* 120:105-112, 2002

Luo J, Li N, Robinson JP and Shi R. The increase of reactive oxygen species and their inhibition in an isolated guinea pig spinal cord compression model. *Spinal Cord* 40: 656-65, 2002.

Luo J, Borgens RB and Shi R. Polyethylene glycol improves function and reduces oxidative stress in synaptosomal preparations after spinal cord injury. *J Neurotrauma* 21:994-1007, 2004

Luo J and Shi R. Diffusive oxidative stress after spinal cord injury and its inhibition by polyethylene glycol. *Neurosci Lett* 359:167-70, 2004

Luo J and Shi R. Acrolein induces plasma membrane damage and oxidative stress in mammalian spinal cord tissue. *Neurochem Int* 44: 475-486, 2004

Luo J, Robinson JP, Shi R. Acrolein-induced cell death in PC12 cells: role of mitochondria-mediated oxidative stress. *Neurochem Int* 47:449-57, 2005.

Luo J, Uchida K, Shi R. Accumulation of acrolein-protein adducts after traumatic spinal cord injury. *Neurochem Res* 30:291-5, 2005.

Luo J and Shi R. Acrolein induces oxidative stress in brain mitochondria. *Neurochem Int* 46:243-52, 2005.

Lin AH, **Luo J**, Mondshein LH, ten Dijke P, Vivien D, Contag CH, Wyss-Coray T. Global analysis of Smad2/3-dependent TGF-β signaling in living mice reveals prominent tissue-specific responses to injury. *J Immunol* 175:547-54, 2005. (Lin and Luo: equal contributions)

Luo J, Lin AH, Masliah E, Wyss-Coray T. Bioluminescence imaging of Smad signaling in living mice shows correlation with excitotoxic neurodegeneration. *Proc Natl Acad Sci USA* 103:18326-31, 2006. (Luo and Lin: equal contributions)

Shi R and **Luo J.** The Role of Acrolein in Spinal Cord Injury. *Applied Neurology* 2: 22-27, 2006

Luo J, Ho PP, Buckwalter MS, Hus T, Lee LY, Zhang H, Kim D, Kim S, Gambhir SS, Steinman L and Wyss-Cory T. Glia-dependent TGF-beta signaling, acting independent of the TH17 pathway, is critical for initiation of autoimmune encephalomyelitis. *J Clin Invest*, 117: 3306-3315, 2007.

Chong AK, Satterwhite T, Pham HM, Costa MA, **Luo J**, Longaker MT, Wyss-Coray T, Chang J. Live imaging of Smad2/3 signaling in mouse skin wound healing. *Wound Repair Regen,* 15:762-766, 2007.

Ando T, Hatsushika K, Wako M, Ohba T, Koyama K, Ohnuma Y, Katoh R, Ogawa H, Okumura K, **Luo J**, Wyss-Coray T, Nakao A. Orally administered TGF-beta is biologically active in the intestinal mucosa and enhances oral tolerance. *J Allergy Clin Immunol* 120: 916-923, 2007.

Satterwhite TS, Chong AK, **Luo J**, Pham H, Costa M, Longaker MT, Wyss-Coray T, Chang J. In vitro analysis of transforming growth factor-β1 inhibition in novel transgenic SBE-luciferase mice. *Ann Plast Surg*, 59:207-213, 2007.

Luo J, Lin A and Wyss-Coray T. Bioluminescence imaging of excitotoxic and endotoxic brain injury in living mice, in *Advances in Alzheimer's and Parkinson's diseases: insights, progress and perspectives* (Editors: Fisher A, Memo M, Stocchi F and Hanin I), Springer, New York, NY. 2007.

Luo J and Shi R. Polyethylene glycol inhibits apoptotic cell death following traumatic spinal cord injury. *Brain Res*, 1155:10-6, 2007.

Luo J, Ho PP, Steinman L and Wyss-Cory T. Bioluminescence in vivo imaging of autoimmune encephalomyelitis predicts disease progression. *Journal of Neuroinflammation* 5: 6, 2008.

Luo J, and Wyss-Coray T. Bioluminescent analysis of Smad dependent TGF-β signaling in live mice, in *Bioluminescence: second edition. Methods in Molecular Biology* 574:193-202, 2009.

Lanz TV, Ding Z, Ho PP, **Luo J**, Agrawal AN, Srinagesh H, Axtell R, Zhang H, Platten M, Wyss-Coray T, Steinman L. Angiotensin II sustains brain inflammation in mice via TGF-beta. *J Clin Invest*, 120: 2782-94, 2010.

Villeda SA, **Luo J**, Mosher KI, Zou B, Britschgi M, Bieri G, Stan TM, Fainberg N, Ding Z, Eggel A, Lucin KM, Czirr E, Couillard-Després S, Aigner L, Li G, Peskind ER, Kaye JA, Quinn JF, Galasko DR, Xie X, Rando TA and Wyss-Coray T. The aging systemic milieu negatively regulates neurogenesis and cognitive function**.** *Nature* 2011 477(7362):90-4.

D. Research Support

Ongoing Research Support

Jian Luo (PI) **12/01/2011 – 11/30/2012**

The Michael J. Fox Foundation for Parkinson's Research

TGF-β signaling as novel therapeutic treatment for Parkinson's disease,

The major goal of this project is to investigate the potential benefits of increasing TGF-β signaling in dopaminergic neurons in a pre-clinical model of Parkinson's disease.

Role: PI