OMB No. 0925-0001 and 0925-0002 (Rev. 09/17 Approved Through 03/31/2020)

BIOGRAPHICAL SKETCH

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

NAME: Shamloo, Mehrdad

eRA COMMONS USER NAME (credential, e.g., agency login): **SHAMLOO.MEHRDAD**

POSITION TITLE: Professor

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

| INSTITUTION AND LOCATION | DEGREE  (if applicable) | Completion Date  MM/YYYY | FIELD OF STUDY |
| --- | --- | --- | --- |
|  |  |  |  |
| Malmo College of Health and Science, Sweden | B.S. | 06/1994 | Health and Science |
| University of Lund, Sweden | M.S. | 12/1998 | Medicine |
| University of Lund, Sweden | Ph.D. | 06/1994 –04/1999 | Neuroscience Research |

**A. Personal Statement**

# I have been working in the field of neuropharmacology, neurobehavior, and brain disorders since the inception of my doctoral studies in 1999. I have directed, as PI and Co-PI, numerous industrial and NIH-funded research projects in this field and have published this work extensively. I have maintained my own laboratory since 2001 in both industry and academic environments. In 2007 I started, managed, and directed the Stanford Behavioral and Functional Neuroscience Laboratory at Stanford University, a platform to conduct experimental behavioral pharmacology studies from start to end with assessment of the neuropathological and biochemical endpoints at the molecular and cellular levels. In addition, my laboratory has focused in last 10 year to understand the underlying mechanism behind the pathologies in neurodegenerative and psychiatric disorders with aim to develop therapeutic interventions to improve the quality of the life of people. Finally, I have extensive experience in animal modeling, drug discovery, neuro and behavior pharmacology, and have spent much time studying and publishing this work. Thus, I have the training, expertise, and strong leadership skills necessary to support the studies suggested in this proposal.

**B. Positions and Honors**

**Positions and Employment**

1. – 1994 Research Associate, Experimental Brain Research, University of Lund, Sweden

1999 – 2001 Associate Scientist, AGY Therapeutics, Inc. South San Francisco, CA

1. – 2003 Research Scientist, AGY Therapeutics, Inc. South San Francisco, CA

2003 – 2005 Senior Scientist**,** Program leaderAGY Therapeutics, Inc. South San Francisco, CA

2005 – 2007 Associate Director of Preclinical Development**,** Affymax. Palo Alto, CA

2007 – Present Director, Behavioral and Functional Neuroscience Laboratory. Stanford, CA

2008 – 2013 Program Director, Stanford Institute for Neuro-Innovation and Translational Neurosciences (SINTN). Stanford, CA

2012 – Present Professor of Neurosurgery, Comparative Medicine & Neurology and Neurological Sciences, School of Medicine. Stanford, CA

**Other Experience and Professional Memberships**

2014 – Present Member, American Neurological Association (ANA)

2008 – Present Member, International Behavioral Neuroscience Society (IBNS)

2008 – Present Member, International Brain Organization (IBRO)

1997 – Present Member, Society for Neuroscience (SfN)

**Honors**

2004 AGY Award for contribution, dedication and high quality preclinical studies to be used for

AGY-94806 IND filing

**C. Contribution to Science**

1. My scientific interests are focused to better understand normal and pathological brain function with the aim to support the discovery of novel therapeutic approaches for neurologic disorders such as stroke, Alzheimer’s disease (AD), and autism. While working in industry, I was responsible for the discovery and development of novel neuroprotective and regenerative small molecule and peptide therapeutics for multiple diseases. As the program leader for neuroprotection and regeneration programs at AGY Therapeutics, my work enabled several patent applications (see below), scientific publications, and an IND application and subsequent clinical trials. These years of experience in industry built on my extensive background in CNS drug discovery and preclinical development.

2. In 2007, I joined Stanford University and Neuroscience Institute to establish a new behavioral neuropharmacology center within neuroscience institute, the Stanford Behavioral and Functional Neuroscience Laboratory (SBFNL). As part of the Stanford Neurosciences Institute, BFNL provides a preclinical discovery platform for CNS target validation in preclinical in vivo and in vitro models. Our well validated neurobehavioral assessment tools in CNS disease models provide a unique platform for the screening and profiling of experimental therapeutics and genetic rodent models. We aim to accelerate the progress in both fundamental and applied studies of nervous system function. We collaborate with academic laboratories, non-profit foundations, and biopharmaceutical companies worldwide to support the understanding of human CNS disorders and develop therapeutic interventions. This center was created with the goal to lead and enable translation of scientific discoveries into novel therapeutic approaches that improve the quality of life for patients with disorders of the brain and spinal cord. I believe I have significantly contributed to progressing translational and basic science by building and maintaining this center. This center has increased the efficiency and productivity of many individual research programs by increasing the throughput and the replicability of results. This support given to academic and industry research has resulted in an acceleration of translational and preclinical neuroscience research and many high-impact publications (see below) as well as IND-filing and clinical studies worldwide.

1. Bader PL, Faizi M, Kim LH, Owen SF, Tadross MR, Alfa RW, Bett GC, Tsien RW, Rasmusson RL, Shamloo M. Mouse model of Timothy syndrome recapitulates triad of autistic traits. Proc Natl Acad Sci U S A. 2011 Sep 13;108(37):15432-7. PMID:21878566 | PMCID:PMC3174658
2. Guo X, Disatnik MH, Monbureau M, Shamloo M, Mochly-Rosen D, Qi X. Inhibition of mitochondrial fragmentation diminishes Huntington's disease-associated neurodegeneration. J Clin Invest. 2013 Dec 2;123(12):5371-88. doi: 10.1172/JCI70911. Epub 2013 Nov 15. PMID: 24231356 | PMCID: PMC3859413
3. Kallop DY, Meilandt WJ, Gogineni A, Easley-Neal C, Wu T, Jubb AM, Yaylaoglu M, Shamloo M, Tessier-Lavigne M, Scearce-Levie K, Weimer RM. A death receptor 6-amyloid precursor protein pathway regulates synapse density in the mature CNS but does not contribute to Alzheimer's disease-related pathophysiology in murine models. J Neurosci. 2014 May 7;34(19):6425-37. doi: 10.1523/JNEUROSCI.4963-13.2014. PMCID: in progress.
4. Portmann T, Yang M, Mao R, Panagiotakos G, Ellegood J, Dolen G, Bader PL, Grueter BA, Goold C, Fisher E, Clifford K, Rengarajan P, Kalikhman D, Loureiro D, Saw NL, Zhengqui Z, Miller MA, Lerch JP, Henkelman RM, Shamloo M, Malenka RC, Crawley JN, et al. Behavioral abnormalities and circuit defects in the basal ganglia of a mouse model of 16p11.2 deletion syndrome. Cell Rep. 2014 May 22;7(4):1077-92. PMID:24794428 | PMCID:PMC4251471

3. In addition to the work described above, in my laboratory in Stanford I have discovered multiple therapeutic targets for neurological diseases. As an example my research led to discovery selective partial agonists of β1-ADR as therapeutic approach for enhancing of the cognitive function in AD and Down’s syndrome. My group along with some other investigators were among the first to show an impairment of this signaling cascade in AD and its contribution to pathogenesis and cognitive deficits. This work has not lead to initiation of 5 clinical studies targeting the ADR system for treatment of the PD and AD. See below for published work in this area

1. Salehi A, Faizi M, Colas D, Valletta J, Laguna J, Takimoto-Kimura R, Kleschevnikov A, Wagnr SL, Aisen P, Shamloo M, Mobley WC. Restoration of norepinephrine-modulated contextual memory in a mouse model of Down syndrome. Sci Transl Med 2009. Nov 15:1(7): 7ra17.
2. Faizi M, Bader PL, Tun C, Encarnacion A, Kleschevnikov A, Belichenko P, Saw N, Priestley M, Tsien RW, Mobley WC, Shamloo M. Comprehensive behavioral phenotyping of Ts65Dn mouse model of Down syndrome: activation of β1-adrenergic receptor by xamoterol as a potential cognitive enhancer. Neurobiol Dis. 2011 Aug 43(2):397-413.
3. Coutellier L, Ardestani PM, Shamloo M. β1-adrenergic receptor activation enhances memory in Alzheimer's disease model. Ann Clin Transl Neurol. 2014 May 1;1(5):348-360.

**Complete List of Published Work in MyBibliography:**

[**http://www.ncbi.nlm.nih.gov/pubmed/?term=Shamloo+M**](http://www.ncbi.nlm.nih.gov/pubmed/?term=Shamloo+M)

**D. Research Support**

**Ongoing Research Support**

**ACTIVE**

1R56AG06839401 (Becker, Shamloo MPI’s) 05/16/2020 – 05/15/2021 0.00 calendar

National Institutes of Health

Altered ENS Neuroimmune Interactions Disrupt Gastrointestinal Motility in Alzheimers Disease.

Major goals:

1U18DA05241501 (Shamloo, PI) 09/30/2020 – 09/29/2021 1.20 calendar

National Institutes of Health $150,000

Validation and pharmacological profiling of a non-psychoactive THC analog, a novel and selective CB2 receptor agoinist, in proof of concept studies using rodent models of heroin addiction.

Major goals: The aim is to develop and test the efficacy of a cannabinoid 2 receptor agonist in in vitro and in vivo studies of heroin addiction.

N/A (Shamloo, PI) 04/01/20 – 03/31/21 0.6 calendar

Stanford - SPARK $40,000

Development of novel and selective death-associated protein kinase 1 inhibitors for the treatment of neurodegenerative diseases.

Major goals: Develop novel and selective death-associated protein kinase.

N/A (Shamloo, PI) 04/01/20 – 03/31/21 0.6 calendar

Stanford - SPARK $30,000

A small-Molecule Activator of AMPK for Treatment of Mitochondrial Disorders.

Major goals: The aim of this proposal is to develop a small molecule activator of AMPK for Treatment of Mitochondrial Disorders.

5 R01 AG054533 04 (Shamloo, PI) 08/01/17 – 05/31/22 1.80 calendar

National Institutes of Health $479,495

Role of beta adrenergic receptors in modulation of cognition, pathology and neuro inflammation in Alzheimer's disease.

Major goals: The aim of this proposal is to investigate the mechanistic basis of adrenergic receptor subtype tone on cognitive function, CNS resident microglia and infiltrating macrophage function, and recruitment of peripheral immune cells to the brain, to determine mechanisms through which modulation of these receptors can mitigate disease progression and reverse cognitive deficits in AD.

5 P30 NSO69375 09 (Steinberg & Shamloo, MPI’s) 03/01/11 – 11/30/20 2.40 calendar

National Institutes of Health $119,162

Stanford Neuroscience Research Cores for Gene Vectors, Microscopy and Behavior. Establish Neuroscience Research Cores: Behavior, Imaging and Gene-Vector CORES.

Major goals: This award provides supporting funds to three research cores within the Stanford Neuroscience Institute to establish a Gene Vector and Virus Core, advanced Microscopy Data Acquisition and Analysis Core and an automated Behavioral Core.

5 R01 DK101674 06A1 (Sonnenburg & Shamloo, MPI’s) 04/15/20 - 03/31/25 0.48 calendar

National Institutes of Health $10,196

Dietary and Microbial Reprogramming of Intestinal Microbiota-Produced Metabolites

Major goals: Define the bacterial species and genes that make toxic compounds and determine how the gut microbiota can be rationally altered to reduce the production of toxic substances.

N/A (Barron & Shamloo, MPI’s) 05/16/20 – 05/15/21 0.0 calendar

Stanford - Coulter Endowment Program $100,000

Opioid addiction: a non-psychoactive tetrahydrocannabinol (THC) analog

Major goals: Develop and test novel small molecule THC biomimetics as treatments for opioid addiction.

**Completed Research Support**

139082 2018 – 2019

Janssen Research & Development, LLC

Title: Development of phagocytosis assay in microglia and astrocyte with image and FACS analysis.

Role: Principal Investigator

Denali Therapeutics, Inc. 2016 – 2017

Title: The role of inflammation in neurological disorders.

Role: Principal Investigator

NIH R21 2016 – 2018

Title: Role of beta adrenergic receptors in modulation of cognition, pathology and neuroinflammation in Alzheimer's Disease.

Role: Principal Investigator

SIP 2016 – 2017

Title: Discovery and development of novel beta-adrenergic receptor ligands as potential therapeutic agents for Alzheimer’s disease.

Role: Principal Investigator

SPECTRUM/SPARK 2014 – 2017

Title: Develop a therapeutic for Alzheimer disease.

Role: Principal Investigator

BioTime, Inc. 2015 – 2017

Title: Establishing In-vivo efficacy of exogenous BDNF in the permanent distal Middle Cerebral Artery Occlusion (dMCAO) rat stroke model and measure post-treatment functional recovery

Role: Principal Investigator

Adamas Pharmaceuticals, Inc. 2015 – 2017

Title: Effect of Amantadine on Functional Recovery after Middle Cerebral Artery

Occlusion in Rats

Role: Principal Investigator