



Mehrdad Shamloo

Professor (Research) of Neurosurgery and, by courtesy, of Neurology

 NIH Biosketch available Online

CONTACT INFORMATION

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Bio

BIO

Professor Mehrdad Shamloo has held several positions at various biopharmaceutical companies in the San Francisco Bay Area, with demonstrated extensive focus on both CNS drug discovery and pre-clinical development. In 2008, Dr. Shamloo joined Stanford University and established the Behavioral and Functional Neuroscience Core Laboratory (BFNL), as well as his own research laboratory which focused on the furtherment of understanding of normal and pathological brain functions in neurological disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), stroke and autism.

Dr. Shamloo's efforts and research are currently directed towards the neurodegenerative pathways responsible for Selective Neuronal Vulnerability of sensitive brain nuclei in the aforementioned disorders. Through these investigations, his aim is to understand the processes leading to the functional and behavioral malfunction in these neurological disorders, and to subsequently develop novel therapeutics to treat them. Long term, Dr. Shamloo seeks to accelerate the translation of experimental discoveries to novel therapeutic approaches, and to ultimately improve the quality of life for patients with brain disorders.

Dr. Shamloo received his doctoral degree from the Wallenberg Neuroscience Center of Lund University in Sweden.

ACADEMIC APPOINTMENTS

- Professor (Research), Neurosurgery
- Professor (Research) (By courtesy), Neurology & Neurological Sciences
- Member, Bio-X
- Member, Wu Tsai Neurosciences Institute

ADMINISTRATIVE APPOINTMENTS

- Program Director, Institute of Neuro-Innovation and Translational Neurosciences, (2008-2013)
- Director, Behavioral and Functional Neuroscience Laboratory, (2007- present)

PROFESSIONAL EDUCATION

- MS, Medical School, University of Lund, Sweden , BioMedical (1998)

- Ph.D., Faculty of Medicine, University of Lund, Sweden (Wallenberg Neuroscience Research Center) , Medical Science (1999)

LINKS

- Shamloo Laboratory: <http://neurosurgery.stanford.edu/research/shamloo/>
- Behavioral and Functional Neuroscience Laboratory: <http://sbfnl.stanford.edu/>

Research & Scholarship

CURRENT RESEARCH AND SCHOLARLY INTERESTS

My laboratory aims to better understand normal and pathological brain function so that we can contribute to the discovery of novel therapeutic approaches for neurologic disorders such as Alzheimer's disease (AD), Parkinson's disease, stroke and autism. We have focused our efforts on targets involved in neurodegenerative pathways responsible for Selective Neuronal Vulnerability of sensitive nuclei in brain in these disorders.

We have identified Beta adrenergic system as one of key player in brain function in these disorders. Beta adrenergic modulation of the inflammation, cognition and pathological progression of CNS diseases are key projects in the lab. We are studying the mechanistic basis for modulation of inflammation, pathology and cognition in these disorders.

Teaching

STANFORD ADVISEES

Postdoctoral Faculty Sponsor

Matteo Santoro

Publications

PUBLICATIONS

- **Role of Endoplasmic Reticulum Stress in Learning and Memory Impairment and Alzheimer's Disease-Like Neuropathology in the PS19 and APP(Swe) Mouse Models of Tauopathy and Amyloidosis.** *eNeuro*
Briggs, D. I., Defensor, E., Memar Ardestani, P., Yi, B., Halpain, M., Seabrook, G., Shamloo, M.
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- **Beta-adrenergic receptor antagonism is proinflammatory and exacerbates neuroinflammation in a mouse model of Alzheimer's Disease.** *Neurobiology of disease*
Evans, A. K., Ardestani, P., Yi, B., Park, H. H., Lam, R., Shamloo, M.
2020: 105089
- **Reactions to Multiple Ascending Doses of the Microtubule Stabilizer TPI-287 in Patients With Alzheimer Disease, Progressive Supranuclear Palsy, and Corticobasal Syndrome: A Randomized Clinical Trial.** *JAMA neurology*
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2019
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- **LRRK2 modifies alpha-syn pathology and spread in mouse models and human neurons** *ACTA NEUROPATHOLOGICA*
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2019; 137 (6): 961–80
- **PGE(2) signaling via the neuronal EP2 receptor increases injury in a model of cerebral ischemia** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*

- Liu, Q., Liang, X., Wang, Q., Wilson, E. N., Lam, R., Wang, J., Kong, W., Tsai, C., Pan, T., Larkin, P. B., Shamloo, M., Andreasson, K. I.
2019; 116 (20): 10019–24
- **LRRK2 modifies alpha-syn pathology and spread in mouse models and human neurons.** *Acta neuropathologica*
Bieri, G., Brahic, M., Bousset, L., Couthouis, J., Kramer, N. J., Ma, R., Nakayama, L., Monbureau, M., Defensor, E., Schule, B., Shamloo, M., Melki, R., Gitler, et al
2019
 - **Peripheral TREM1 responses to brain and intestinal immunogens amplify stroke severity** *Nature Immunology*
Liu, Q., Johnson, E., et al
2019
 - **PGE2 signaling via the neuronal EP2 receptor increases injury in a model of cerebral ischemia.** *Proceedings of the National Academy of Sciences of the United States of America*
Liu, Q., Liang, X., Wang, Q., Wilson, E. N., Lam, R., Wang, J., Kong, W., Tsai, C., Pan, T., Larkin, P. B., Shamloo, M., Andreasson, K. I.
2019
 - **Intracerebral Delivery of Brain-Derived Neurotrophic Factor Using HyStem (R)-C Hydrogel Implants Improves Functional Recovery and Reduces Neuroinflammation in a Rat Model of Ischemic Stroke** *INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES*
Ravina, K., Briggs, D. I., Kislat, S., Warraich, Z., Nguyen, T., Lam, R. K., Zarembinski, T. I., Shamloo, M.
2018; 19 (12)
 - **Early adolescent Rai1 reactivation reverses transcriptional and social interaction deficits in a mouse model of Smith-Magenis syndrome** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
Huang, W., Wang, D. C., Allen, W. E., Klope, M., Hu, H., Shamloo, M., Luo, L.
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 - **Interaction of mitochondria fission factor with dynamin related protein 1 governs physiological mitochondria function in vivo** *SCIENTIFIC REPORTS*
Kornfeld, O. S., Qvit, N., Haileselassie, B., Shamloo, M., Bernardi, P., Mochly-Rosen, D.
2018; 8
 - **Activity-dependent modulation of hippocampal synaptic plasticity via PirB and endocannabinoids.** *Molecular psychiatry*
Djurisic, M., Brott, B. K., Saw, N. L., Shamloo, M., Shatz, C. J.
2018
 - **Inhibition of Drp1/Fis1 interaction slows progression of amyotrophic lateral sclerosis.** *EMBO molecular medicine*
Joshi, A. U., Saw, N. L., Vogel, H., Cunningham, A. D., Shamloo, M., Mochly-Rosen, D.
2018
 - **Drp1/Fis1 interaction mediates mitochondrial dysfunction, bioenergetic failure and cognitive decline in Alzheimer's disease.** *Oncotarget*
Joshi, A. U., Saw, N. L., Shamloo, M., Mochly-Rosen, D.
2018; 9 (5): 6128–43
 - **Small molecule modulator of sigma 2 receptor is neuroprotective and reduces cognitive deficits and neuroinflammation in experimental models of Alzheimer's disease.** *Journal of neurochemistry*
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2017; 140 (4): 561-575
 - **Modulation of neuroinflammation and pathology in the 5XFAD mouse model of Alzheimer's disease using a biased and selective beta-1 adrenergic receptor partial agonist.** *Neuropharmacology*
Ardestani, P. M., Evans, A. K., Yi, B., Nguyen, T., Coutellier, L., Shamloo, M.
2017; 116: 371-386
 - **Discovery of novel brain permeable and G protein-biased beta-1 adrenergic receptor partial agonists for the treatment of neurocognitive disorders.** *PLoS one*
Yi, B., Jahangir, A., Evans, A. K., Briggs, D., Ravina, K., Ernest, J., Farimani, A. B., Sun, W., Rajadas, J., Green, M., Feinberg, E. N., Pande, V. S., Shamloo, et al
2017; 12 (7): e0180319
 - **A novel pharmacological tool blocks physiological mitochondrial fission through specifically inhibiting the Mff-Drp1 protein-protein interaction.**
Kornfeld, O. S., Qvit, N., Monbureau, M., Halpain, M., Shamloo, M., Mochly-Rosen, D.
AMER SOC CELL BIOLOGY.2017

- **The Golgi Outpost Protein TPPP Mediates Uniform Microtubule Polarity and Branching in Oligodendrocytes.**
Fu, M., Oses-Prieto, J. A., Lee, C., Saw, N. L., Shi, R., Nori, M., Shamloo, M., Burlingame, A., Barres, B. A.
AMER SOC CELL BIOLOGY.2017
- **A small molecule p75NTR ligand normalizes signalling and reduces Huntington's disease phenotypes in R6/2 and BACHD mice.** *Human molecular genetics*
Simmons, D. A., Belichenko, N. P., Ford, E. C., Semaan, S., Monbureau, M., Aiyaswamy, S., Holman, C. M., Condon, C., Shamloo, M., Massa, S. M., Longo, F. M.
2016; 25 (22): 4920-4938
- **Potential biomarkers to follow the progression and treatment response of Huntington's disease.** *journal of experimental medicine*
Disatnik, M., Joshi, A. U., Saw, N. L., Shamloo, M., Leavitt, B. R., Qi, X., Mochly-Rosen, D.
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- **Novel, selective EPO receptor ligands lacking erythropoietic activity reduce infarct size in acute myocardial infarction in rats** *PHARMACOLOGICAL RESEARCH*
Kiss, K., Csonka, C., Paloczi, J., Pipis, J., Goerbe, A., Kocsis, G. F., Murlasits, Z., Sarkoezy, M., Szucs, G., Holmes, C. P., Pan, Y., Bhandari, A., Csont, et al
2016; 113: 62-70
- **Molecular and Neural Functions of Rai1, the Causal Gene for Smith-Magenis Syndrome.** *Neuron*
Huang, W., Guenther, C. J., Xu, J., Nguyen, T., Schwarz, L. A., Wilkinson, A. W., Gozani, O., Chang, H. Y., Shamloo, M., Luo, L.
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- **A small molecule p75NTR ligand normalizes signalling and reduces Huntington's disease phenotypes in R6/2 and BACHD mice.** *Human molecular genetics*
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- **An 8-week, open-label, dose-finding study of Nimodipine for the treatment of progranulin insufficiency from GRN gene mutations**
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- **Drug-controllable protein tags for the selective visualization or selective shutoff of newly synthesized proteins of interest in mammalian cells and in vivo**
Jacobs, C., Geng, Y., Badiie, R., Nguyen, T., Evans, A., Chung, H., Yang, Y., Shamloo, M., Tsien, R. Y., Lin, M. Z.
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- **Utilizing a novel peptide Inhibitor to modulate mitochondrial dynamics and investigate Drp1's physiological roles.**
Kornfeld, O. S., Qvit, N., Disatnik, M., Monbureau, M., Halpain, M., Evans, A., Shamloo, M., Rosen, D.
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- **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.** *journal of neuroscience*
Kallop, D. Y., Meilandt, W. J., Gogineni, A., Easley-Neal, C., Wu, T., Jubb, A. M., Yaylaoglu, M., Shamloo, M., Tessier-Lavigne, M., Scearce-Levie, K., Weimer, R. M.
2014; 34 (19): 6425-6437
- **β1-adrenergic receptor activation enhances memory in Alzheimer's disease model.** *Annals of clinical and translational neurology*
Coutellier, L., Ardestani, P. M., Shamloo, M.
2014; 1 (5): 348-360
- **the Basal Ganglia of a Mouse Model of 16p11.2 Deletion Syndrome** *CELL REPORTS*
Portmann, T., Yang, M., Mao, R., Panagiotakos, G., Ellegood, J., Dolen, G., Bader, P. L., Grueter, B. A., Goold, C., Fisher, E., Clifford, K., Rengarajan, P., Kalikhman, et al
2014; 7 (4): 1077-1092
- **Small molecule p75NTR ligands reduce pathological phosphorylation and misfolding of tau, inflammatory changes, cholinergic degeneration, and cognitive deficits in ABPP(L/S) transgenic mice.** *Journal of Alzheimer's disease : JAD*
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2014; 42 (2): 459-483

- **Behavioral abnormalities and circuit defects in the Basal Ganglia of a mouse model of 16p11.2 deletion syndrome.** *Cell reports*
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- **Alzheimer's therapeutics targeting amyloid beta 1-42 oligomers I: Abeta 42 oligomer binding to specific neuronal receptors is displaced by drug candidates that improve cognitive deficits.** *PLoS one*
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- **Small Molecule p75(NTR) Ligands Reduce Pathological Phosphorylation and Misfolding of Tau, Inflammatory Changes, Cholinergic Degeneration, and Cognitive Deficits in A beta PPL/S Transgenic Mice** *JOURNAL OF ALZHEIMERS DISEASE*
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- **Inhibition of mitochondrial fragmentation diminishes Huntington's disease-associated neurodegeneration** *JOURNAL OF CLINICAL INVESTIGATION*
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- **A Small Molecule TrkB Ligand Reduces Motor Impairment and Neuropathology in R6/2 and BACHD Mouse Models of Huntington's Disease.** *journal of neuroscience*
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- **A Dramatic Increase of C1q Protein in the CNS during Normal Aging** *JOURNAL OF NEUROSCIENCE*
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2013; 33 (33): 13460-13474
- **A dramatic increase of C1q protein in the CNS during normal aging.** *journal of neuroscience*
Stephan, A. H., Madison, D. V., Mateos, J. M., Fraser, D. A., Lovelett, E. A., Coutellier, L., Kim, L., Tsai, H., Huang, E. J., Rowitch, D. H., Berns, D. S., Tenner, A. J., Shamloo, et al
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- **GluN2B Antagonism Affects Interneurons and Leads to Immediate and Persistent Changes in Synaptic Plasticity, Oscillations, and Behavior.** *Neuropsychopharmacology*
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Beraki, S., Litrus, L., Soriano, L., Monbureau, M., To, L. K., Braithwaite, S. P., Nikolich, K., Urfer, R., Oksenberg, D., Shamloo, M.
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- **A role for C1q in normal brain aging**
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- **Npas4: A Neuronal Transcription Factor with a Key Role in Social and Cognitive Functions Relevant to Developmental Disorders** *PLOS ONE*
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- **Deficits in Cognition and Synaptic Plasticity in a Mouse Model of Down Syndrome Ameliorated by GABA(B) Receptor Antagonists** *JOURNAL OF NEUROSCIENCE*
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2012; 2 (2): 142-154
- **Thy1-hAPP(Lond/Swe+) mouse model of Alzheimer's disease displays broad behavioral deficits in sensorimotor, cognitive and social function** *BRAIN AND BEHAVIOR*
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- **Identification of a central role for complement in osteoarthritis** *NATURE MEDICINE*
Wang, Q., Rozelle, A. L., Lepus, C. M., Scanzello, C. R., Song, J. J., Larsen, D. M., Crish, J. F., Bebek, G., Ritter, S. Y., Lindstrom, T. M., Hwang, I., Wong, H. H., Punzi, et al
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- **Mouse model of Timothy syndrome recapitulates triad of autistic traits** *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA*
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Horie, N., Pereira, M. P., Niizuma, K., Sun, G., Keren-Gill, H., Encarnacion, A., Shamloo, M., Hamilton, S. A., Jiang, K., Huhn, S., Palmer, T. D., Bliss, T. M., Steinberg, et al
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