Meet the Editorial Board Member

James R. Bamburg
Department of Biochemistry and Molecular Biology
Molecular and Radiological Bioscience Building, Room 235
Fort Collins, CO 80523-1870
Colorado State University
USA


James R. Bamburg

James R. Bamburg is an Emeritus Professor of Biochemistry and Molecular Biology at Colorado State University. He is also a member and former Director of the Molecular, Cellular and Integrative Neuroscience Program, a founder of the Cell and Molecular Biology Graduate Program and a member of the School of Biomedical Engineering at Colorado State University as well as a member of the Gates Center for Regenerative Medicine at the University of Colorado Anschutz Medical Campus Denver. He received his Bachelor's of Science in Chemistry from the University of Illinois and his Ph.D. in Biochemistry from the University of Wisconsin where he isolated and characterized several important hemorrhagic mycotoxins. He pursued postdoctoral training in Pharmacology and Neuroscience at Stanford University before joining Colorado State University as an assistant professor. During his 5 sabbatical leaves, he was a Guggenheim Fellow at the Laboratory of Molecular Biology, Cambridge, U.K., twice a Sr. International Fellow of the NIH Fogarty Center, once in London, U.K. at Kings College and again in Sydney, Australia at the Children's Medical Research Institute and the University of Sydney. He was twice a Visiting Professor, once at the University of California San Diego and again at The Scripps Research Institute, La Jolla, California. He was also a summer William Evans Visiting Scholar at Otago University, Dunedin, New Zealand. His research has been funded for over 42 years by the National Institutes of Health and by grants from many foundations and private agencies. He has served for many years on the editorial boards of *Biochemistry*, *Cytoskeleton,* and *Current Neuropharmacology*.

In a search for actin regulatory proteins in neuronal growth cones, he discovered the first member of the ADF/cofilin family of proteins and has continued to study their activity and regulation in many different biological processes. To study actin-based processes in cells, he developed the first fluorescence microscopy center at Colorado State University, which he helped develop into a university-wide foundational core of advanced microscopes accessible to all CSU researchers. His lab first demonstrated the phosphoregulation of ADF/cofilin, identified the phosphoregulatory site, their competition with muscle tropomyosin in binding F-actin, their cooperative binding to F-actin, their regulation of myosin II contractility in cells, and with many collaborators, their role in all aspects of neuronal differentiation, growth cone motility, pathfinding, synaptogenesis and synaptic function. His current research is an outgrowth of a discovery made by his wife and research partner, Laurie S. Minamide, of rod-shaped bundles of cofilin-actin filaments (rods) that occur within neurites of stressed neurons. Rods have a transient neuroprotective effect but cause the loss of synapses distal to them. Rods are found in the brains of humans with several different neurological and/or neurodegenerative disorders including Alzheimer's disease and in many mouse models used for studying these disorders. Their current research is focused on understanding the signaling pathways of rod formation and finding inhibitors of rod formation as potential therapeutics for dementia.

SELECTED PUBLICATIONS

[1] Meberg, P.J.; Bamburg, J,R. Increase in neurite outgrowth mediated by overexpression of actin depolymerizing factor. *J. Neurosci*., **2000**, *20*, 2459-2469.
[2] Minamide, L.S.;Striegl, A.M.; Boyle, JA.; Meberg, P.J.; Bamburg, J.R. Neurodegenerative stimuli induce persistent ADF/cofilin-actin rods that disrupt distal neurite function. *Nat. Cell Biol*., **2000,** *2*, 628-636.
[3] Bernstein, B.W.; Bamburg, J.R. Actin-ATP hydrolysis: a major energy drain for ischemically stressed neurons. *J. Neurosci*., **2003,** *23*, 1-6.
[4] Chen, H.; Bernstein, B.W.; Sneider, J.; Boyle, J.A.; Minamide, L.S.; Bamburg, J.R. *In vitro* activity differences between proteins of the ADF /cofilin family define two distinct subgroups. *Biochemistry,* **2004**, *43,* 7127-7142.
[5] Maloney, M.T.; Minamide, L.S.; Kinley, A.; Boyle, J.A.; Bamburg, J.R. β-Secretase-cleaved APP accumulates at actin inclusions induced in neurons by stress or amyloid beta: a feed-forward mechanism for Alzheimer's Disease. *J. Neurosci.,* **2005,** *25,* 11313-11321.
[6] Bernstein, B.W.; Chen, H.; Boyle, J.A.; Bamburg, J.R. Formation of actin-ADF/cofilin rods transiently retards decline of mitochondrial potential and ATP in stressed neurons. *Am. J. Physiol. Cell Physiol*., **2006,** *291,* C828-39.
[7] Minamide, L.S.; Maiti, S.; Boyle, J.A.; Davis, R.C.; Coppinger, J.A.; Bao, Y.; Huang, T.Y.; Yates, J.; Bokoch, G.M.; Bamburg, J.R. Isolation and characterization of cofilin-actin rods from stressed cells. *J. Biol. Chem.,* **2010,** *285*, 5450-5460.
[8] Wiggan, O.; Shaw, A.E.; DeLuca, J.G.; Bamburg, J.R. ADF/cofilin regulates actomyosin assembly through competitive inhibition of myosin II binding to F-actin. *Dev. Cell,* **2012**, *22,* 530-543.
[9] Bernstein, B.W.; Shaw, A.E.; Minamide, L.S., Pak, C.W.; Bamburg, J.R. Incorporation of cofilin into rods depends on disulfide intermolecular bonds: Implications for actin regulation and neurodegenerative disease. *J. Neurosci.,* **2012,** *32*, 6670-6681.
[10] Walsh, K.P.; Minamide, L.S.; Kane, S.J.; Shaw, A.E.; Brown, D.R.; Pulford, B.; Zabel, M.D.; Lambeth, J.D.; Kuhn, T.B.; Bamburg, J.R. Amyloid-β and proinflammatory cytokines utilize a prion-dependent pathway to activate NADPH oxidase and induce cofilin-actin rods in hippocampal neurons. *PLoS One,* **2014,** *9,* e95995.

1570-159X/22

[11] Bamburg, J.R.; Bernstein, B.W. Actin dynamics and cofilin-actin rods in Alzheimer disease. *Cytoskeleton (Hoboken),* **2016,** *73,* 477-497.
[12] Shaw, A.E.; Bamburg, J.R. Peptide regulation of cofilin activity in the CNS: a novel therapeutic approach for treatment of multiple neurological disorders. *Pharmacol. Ther.,* **2017,** *175*, 17-27.
[13] Fixman, B.B.; Babcock, I.W.; Minamide, L.S.; Shaw, A.E.; Oliveira da Silva, M.I.; Runyan, A.M.; Maloney, M.T.; Field, J.J.; Bamburg, J.R. Modified roller tube method for precisely localized and repetitive intermittent imaging during long-term culture of brain slices in an enclosed system. *JoVE,* **2017**, e56436. doi:10.3791/56436.
[14] Tahtamouni, L.H.; Nawasreh, M.M.; Al-Mazaydeh, Z.A.; Al-Khateeb, R.A.; Abdellatif, R.N.; Bawadi, R.M.; Bamburg, J.R.; Yasin, S.R. Cephalostatin 1 analogues activate apoptosis *via* the endoplasmic reticulum stress signaling pathway. *Eur. J. Pharmacol*., **2018,** *818*, 400-09.
[15] Won, S.J.; Minella, A.M.;,Wu, L.; Eun, C.H.; Rome, E.; Herson, P.S.; Shaw, A.E.; Bamburg, J.R.; Swanson, R.A. Cofilin-actin rod formation in neuronal processes after brain ischemia. *PLoS One,* **2018,** *13*(10), e0198709.
[16] Smith, L.K.; Kuhn, TB.; Chen, J.; Bamburg, J.R. HIV-1 associated neurodegenerative disorders: a new perspective on the role of lipid rafts in Gp-120-mediated neurotoxicity. *Curr. HIV Res.,* **2018**, *16*(4), 258-269.
[17] Wiggan, O.; DeLuca, J.G.; Stasevich, T.J.; Bamburg, J.R. Lamin A/C deficiency enables increased myosin-II bipolar filament ensembles that promote divergent actomyosin network anomalies through self-organization. *Mol. Biol. Cell,* **2020**, *31*(21), 2363-2378.
[18] Smith, L.K.; Babcock, I.W.; Minamide LS, Shaw, A.E.; Bamburg, J.R.; Kuhn, T.B. Direct interaction of HIV gp120 with neuronal CXCR4 and CCR5 receptors induces cofilin-actin rod pathology *via* a cellular prion protein- and NOX-dependent mechanism. *PLoS One*, **2021**, *16*(3), e0248309. DOI: 10.1371/journal.pone.0248309.
[19] Bamburg, J.R.; Minamide, L.S.; Wiggan, O.; Tahtamouni, L.H.; Kuhn, T.B. Cofilin and actin dynamics: Multiple modes of regulation and their impacts in neuronal development and degeneration. *Cells*., **2021**, *10*(10), 2726. DOI: 10.3390/cells10102726.