Ben A Bahr

List of Publications by Year
in descending order

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		136950	168389
65	2,966	32	53
papers	citations	h-index	g-index

66	66	66	3934
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Stimulation of autophagy and synaptic maintenance are commonalities induced by an exerciseâ€mimetic and diet supplement to avoid initiators of ageâ€related cognitive decline. FASEB Journal, 2022, 36, .	0.5	0
2	Distinct ginseng extracts produce disparate effects on proteostatic support through the autophagyâ€lysosomal pathway that is linked to synaptic resilience and cognitive health. FASEB Journal, 2022, 36, .	0.5	0
3	Distinct and dementiaâ€related synaptopathy in the hippocampus after military blast exposures. Brain Pathology, 2021, 31, e12936.	4.1	6
4	Excitotoxic stimulation activates distinct pathogenic and protective expression signatures in the hippocampus. Journal of Cellular and Molecular Medicine, 2021, 25, 9011-9027.	3.6	7
5	Endosomal-lysosomal dysfunction in metabolic diseases and Alzheimer's disease. International Review of Neurobiology, 2020, 154, 303-324.	2.0	14
6	Discovery of small molecules that normalize the transcriptome and enhance cysteine cathepsin activity in progranulin-deficient microglia. Scientific Reports, 2020, 10, 13688.	3.3	13
7	The Role of Lysosomes in a Broad Disease-Modifying Approach Evaluated across Transgenic Mouse Models of Alzheimerâ€™s Disease and Parkinsonâ€™s Disease and Models of Mild Cognitive Impairment. International Journal of Molecular Sciences, 2019, 20, 4432.	4.1	31
8	Piperidine and piperazine inhibitors of fatty acid amide hydrolase targeting excitotoxic pathology. Bioorganic and Medicinal Chemistry, 2019, 27, 115096.	3.0	9
9	Early Synaptic Alterations and Selective Adhesion Signaling in Hippocampal Dendritic Zones Following Organophosphate Exposure. Scientific Reports, 2019, 9, 6532.	3.3	13
10	Engulfment and cell motility protein 1 potentiates diabetic cardiomyopathy via Rac-dependent and Rac-independent ROS production. JCI Insight, 2019, 4, .	5.0	11
11	Glutamate-induced and NMDA receptor-mediated neurodegeneration entails P2Y1 receptor activation. Cell Death and Disease, 2018, 9, 297.	6.3	58
12	Effects on Neurons and Hippocampal Slices by Single and Multiple Primary Blast Pressure Waves From Detonating Spherical Cyclotrimethylenetrinitramine (RDX) Explosive Charges. Military Medicine, 2018, 183, 269-275.	0.8	5
13	Endo-lysosomal dysfunction: a converging mechanism in neurodegenerative diseases. Current Opinion in Neurobiology, 2018, 48, 52-58.	4.2	97
14	Poor cognitive ageing: Vulnerabilities, mechanisms and the impact of nutritional interventions. Ageing Research Reviews, 2018, 42, 40-55.	10.9	136
15	Inhibitor of Endocannabinoid Deactivation Protects Against In Vitro and In Vivo Neurotoxic Effects of Paraoxon. Journal of Molecular Neuroscience, 2017, 63, 115-122.	2.3	9
16	AÎ²42-mediated proteasome inhibition and associated tau pathology in hippocampus are governed by a lysosomal response involving cathepsin B: Evidence for protective crosstalk between protein clearance pathways. PLoS ONE, 2017, 12, e0182895.	2.5	18
17	Potential Alzheimerâ€™s Disease Therapeutics Among Weak Cysteine Protease Inhibitors Exhibit Mechanistic Differences Regarding Extent of Cathepsin B Up-Regulation and Ability to Block Calpain. European Scientific Journal, 2017, 13, 38-59.	0.1	5
18	Paraoxon: An Anticholinesterase That Triggers an Excitotoxic Cascade of Oxidative Stress, Adhesion Responses, and Synaptic Compromise. European Scientific Journal, 2017, 13, 29-37.	0.1	3

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19	Military blast-induced synaptic changes with distinct vulnerability may explain behavioral alterations in the absence of obvious brain damage. Journal of Nature and Science, 2017, 3, .	1.1	1
20	Blast waves from detonated military explosive reduce GluR1 and synaptophysin levels in hippocampal slice cultures. Experimental Neurology, 2016, 286, 107-115.	4.1	18
21	Abnormal response of distal Schwann cells to denervation in a mouse model of motor neuron disease. Experimental Neurology, 2016, 278, 116-126.	4.1	17
22	A Single Pathway Targets Several Health Challenges of the Elderly. Rejuvenation Research, 2014, 17, 382-384.	1.8	1
23	Z-Phe-Ala-diazomethylketone (PADK) Disrupts and Remodels Early Oligomer States of the Alzheimer Disease AÎ²42 Protein. Journal of Biological Chemistry, 2012, 287, 6084-6088.	3.4	34
24	Positive Lysosomal Modulation As a Unique Strategy to Treat Age-Related Protein Accumulation Diseases. Rejuvenation Research, 2012, 15, 189-197.	1.8	43
25	Equipotent Inhibition of Fatty Acid Amide Hydrolase and Monoacylglycerol Lipase â€“ Dual Targets of the Endocannabinoid System to Protect against Seizure Pathology. Neurotherapeutics, 2012, 9, 801-813.	4.4	49
26	Nonpeptidic Lysosomal Modulators Derived from Z-Phe-Ala-Diazomethylketone for Treating Protein Accumulation Diseases. ACS Medicinal Chemistry Letters, 2012, 3, 920-924.	2.8	12
27	Submicromolar AÎ²42 reduces hippocampal glutamate receptors and presynaptic markers in an aggregation-dependent manner. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2011, 1812, 1664-1674.	3.8	18
28	Protective Effects of Positive Lysosomal Modulation in Alzheimer's Disease Transgenic Mouse Models. PLoS ONE, 2011, 6, e20501.	2.5	77
29	A New Generation Fatty Acid Amide Hydrolase Inhibitor Protects Against Kainate-Induced Excitotoxicity. Journal of Molecular Neuroscience, 2011, 43, 493-502.	2.3	45
30	Selective modulation of the endocannabinoid system for targeted protection in kainic acid models of excitotoxicity. FASEB Journal, 2011, 25, lb420.	0.5	0
31	Enhancement of endocannabinoid signaling by fatty acid amide hydrolase inhibition: A neuroprotective therapeutic modality. Life Sciences, 2010, 86, 615-623.	4.3	80
32	Lysosomal Modulatory Drugs for a Broad Strategy Against Protein Accumulation Disorders. Current Alzheimer Research, 2009, 6, 438-445.	1.4	14
33	Gephyrin Alterations Due to Protein Accumulation Stress are Reduced by the Lysosomal Modulator Z-Phe-Ala-Diazomethylketone. Journal of Molecular Neuroscience, 2008, 34, 131-139.	2.3	10
34	Calpain activation is involved in early caspaseâ€independent neurodegeneration in the hippocampus following status epilepticus. Journal of Neurochemistry, 2008, 105, 666-676.	3.9	46
35	Endocannabinoid Enhancement Protects against Kainic Acid-Induced Seizures and Associated Brain Damage. Journal of Pharmacology and Experimental Therapeutics, 2007, 322, 1059-1066.	2.5	83
36	Quantitative Method for the Profiling of the Endocannabinoid Metabolome by LC-Atmospheric Pressure Chemical Ionization-MS. Analytical Chemistry, 2007, 79, 5582-5593.	6.5	79

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37	Microtubule-stabilizing agent prevents protein accumulation-induced loss of synaptic markers. European Journal of Pharmacology, 2007, 562, 20-27.	3.5	56
38	Targeting the endocannabinoid system in treating brain disorders. Expert Opinion on Investigational Drugs, 2006, 15, 351-365.	4.1	46
39	Oxidative Stress and Lysosomes: CNS-Related Consequences and Implications for Lysosomal Enhancement Strategies and Induction of Autophagy. Antioxidants and Redox Signaling, 2006, 8, 185-196.	5.4	86
40	3-Nitropropionic acid toxicity in hippocampus: Protection throughN-methyl-D-aspartate receptor antagonism. Hippocampus, 2006, 16, 834-842.	1.9	33
41	Potential Compensatory Responses Through Autophagic/Lysosomal Pathways in Neurodegenerative Diseases. Autophagy, 2006, 2, 234-237.	9.1	53
42	Blocking cannabinoid activation of FAK and ERK1/2 compromises synaptic integrity in hippocampus. European Journal of Pharmacology, 2005, 508, 47-56.	3.5	49
43	Dual Modulation of Endocannabinoid Transport and Fatty Acid Amide Hydrolase Protects against Excitotoxicity. Journal of Neuroscience, 2005, 25, 7813-7820.	3.6	109
44	Cellular Responses to Protein Accumulation Involve Autophagy and Lysosomal Enzyme Activation. Rejuvenation Research, 2005, 8, 227-237.	1.8	44
45	Neural Cell Adhesion Molecule-associated Polysialic Acid Potentiates Î±-Amino-3-hydroxy-5-methylisoxazole-4-propionic Acid Receptor Currents. Journal of Biological Chemistry, 2004, 279, 47975-47984.	3.4	86
46	The pathogenic activation of calpain: a marker and mediator of cellular toxicity and disease states. International Journal of Experimental Pathology, 2004, 81, 323-339.	1.3	98
47	Biphasic NF-?B activation in the excitotoxic hippocampus. Acta Neuropathologica, 2004, 108, 173-82.	7.7	15
48	Repeated contact with subtoxic soman leads to synaptic vulnerability in hippocampus. Journal of Neuroscience Research, 2004, 77, 739-746.	2.9	18
49	Lysosomal Activation Is a Compensatory Response Against Protein Accumulation and Associated Synaptopathogenesisâ€"An Approach for Slowing Alzheimer Disease?. Journal of Neuropathology and Experimental Neurology, 2003, 62, 451-463.	1.7	140
50	Intracellular Deposition, Microtubule Destabilization, and Transport Failure: An â€œEarlyâ€•Pathogenic Cascade Leading to Synaptic Decline. Journal of Neuropathology and Experimental Neurology, 2002, 61, 640-650.	1.7	59
51	Survival Signaling and Selective Neuroprotection Through Glutamatergic Transmission. Experimental Neurology, 2002, 174, 37-47.	4.1	65
52	Peptidyl ?-keto amide inhibitor of calpain blocks excitotoxic damage without affecting signal transduction events. Journal of Neuroscience Research, 2002, 67, 787-794.	2.9	29
53	The neuropathogenic contributions of lysosomal dysfunction. Journal of Neurochemistry, 2002, 83, 481-489.	3.9	181
54	Positive Modulation of Î±-Amino-3-hydroxy-5-methyl-4-isoxazolepropionic Acid-Type Glutamate Receptors Elicits Neuroprotection after Trimethyltin Exposure in Hippocampus. Toxicology and Applied Pharmacology, 2002, 185, 111-118.	2.8	16

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55	Delayed and isoform-specific effect of NMDA exposure on neural cell adhesion molecules in hippocampus. Neuroscience Research, 2001, 39, 167-173.	1.9	23
56	Integrin-type signaling has a distinct influence on NMDA-induced cytoskeletal disassembly. , 2000, 59, 827-832.		27
57	The pathogenic activation of calpain: a marker and mediator of cellular toxicity and disease states. International Journal of Experimental Pathology, 2000, 81, 323-339.	1.3	148
58	Activation of NMDA receptors stimulates extracellular proteolysis of cell adhesion molecules in hippocampus. Brain Research, 1998, 811, 152-155.	2.2	43
59	Amyloid ? protein is internalized selectively by hippocampal field CA1 and causes neurons to accumulate amyloidogenic carboxyterminal fragments of the amyloid precursor protein. , 1998, 397, 139-147.		81
60	Age-Related Phosphorylation and Fragmentation Events Influence the Distribution Profiles of Distinct Tau Isoforms in Mouse Brain. Journal of Neuropathology and Experimental Neurology, 1998, 57, 111-121.	1.7	21
61	Arg-Gly-Asp-Ser-Selective Adhesion and the Stabilization of Long-Term Potentiation: Pharmacological Studies and the Characterization of a Candidate Matrix Receptor. Journal of Neuroscience, 1997, 17, 1320-1329.	3.6	165
62	Stable maintenance of glutamate receptors and other synaptic components in long-term hippocampal slices. Hippocampus, 1995, 5, 425-439.	1.9	86
63	Induction of Î²-Amyloid-Containing Polypeptides in Hippocampus: Evidence for a Concomitant Loss of Synaptic Proteins and Interactions with an Excitotoxin. Experimental Neurology, 1994, 129, 81-94.	4.1	68
64	Age-related changes in neural cell adhesion molecule (NCAM) isoforms in the mouse telencephalon. Brain Research, 1993, 628, 286-292.	2.2	15
65	Translational suppression of a glutamate receptor subunit impairs long-term potentiation. Synapse, 1992, 12, 333-337.	1.2	44