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List of Publications by Year in descending order

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68
papers

6,865
citations

46918

47
h-index

98622

67
g-index

68
all docs

68
docs citations

68
times ranked

5383
citing authors

#	ARTICLE	IF	CITATIONS
1	Zn ²⁺ : a novel ionic mediator of neural injury in brain disease. Trends in Pharmacological Sciences, 2000, 21, 395-401.	4.0	536
2	Modulation of mitochondrial function by endogenous Zn ²⁺ pools. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 6157-6162.	3.3	387
3	Preferential Zn ²⁺ influx through Ca ²⁺ -permeable AMPA/kainate channels triggers prolonged mitochondrial superoxide production. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 2414-2419.	3.3	372
4	AMPA Exposures Induce Mitochondrial Ca ²⁺ Overload and ROS Generation in Spinal Motor Neurons <i>In Vitro</i> . Journal of Neuroscience, 2000, 20, 240-250.	1.7	284
5	AMPA receptor activation potentiates zinc neurotoxicity. Neuron, 1993, 10, 43-49.	3.8	271
6	Zn ²⁺ Induces Permeability Transition Pore Opening and Release of Pro-apoptotic Peptides from Neuronal Mitochondria. Journal of Biological Chemistry, 2001, 276, 47524-47529.	1.6	243
7	Ca ²⁺ and Zn ²⁺ permeable AMPA or kainate receptors: possible key factors in selective neurodegeneration. Trends in Neurosciences, 2000, 23, 365-371.	4.2	232
8	Measuring zinc in living cells.. Cell Calcium, 2002, 31, 245-251.	1.1	232
9	Calcium-permeable AMPA channels in neurodegenerative disease and ischemia. Current Opinion in Neurobiology, 2006, 16, 281-287.	2.0	227
10	Neurotoxicity of β -N-methylamino-L-alanine (BMAA) and β -N-oxalylamino-L-alanine (BOAA) on cultured cortical neurons. Brain Research, 1989, 497, 64-71.	1.1	205
11	Beta-N-methylamino-L-alanine neurotoxicity: requirement for bicarbonate as a cofactor. Science, 1988, 241, 973-975.	6.0	196
12	Zinc: new clues to diverse roles in brain ischemia. Trends in Pharmacological Sciences, 2011, 32, 480-486.	4.0	162
13	Excitotoxic and oxidative cross-talk between motor neurons and glia in ALS pathogenesis. Trends in Neurosciences, 2004, 27, 17-23.	4.2	152
14	Intracellular Zn ²⁺ Accumulation Contributes to Synaptic Failure, Mitochondrial Depolarization, and Cell Death in an Acute Slice Oxygen-Glucose Deprivation Model of Ischemia. Journal of Neuroscience, 2009, 29, 1105-1114.	1.7	148
15	Ketamine protects cultured neocortical neurons from hypoxic injury. Brain Research, 1986, 380, 186-190.	1.1	144
16	AMPA/kainate receptor-triggered Zn ²⁺ entry into cortical neurons induces mitochondrial Zn ²⁺ uptake and persistent mitochondrial dysfunction. European Journal of Neuroscience, 2000, 12, 3813-3818.	1.2	140
17	Blockade of Ca ²⁺ -Permeable AMPA/Kainate Channels Decreases Oxygen-Glucose Deprivation-Induced Zn ²⁺ Accumulation and Neuronal Loss in Hippocampal Pyramidal Neurons. Journal of Neuroscience, 2002, 22, 1273-1279.	1.7	139
18	Tumor necrosis-factor-alpha (TNF- α) induces rapid insertion of Ca ²⁺ -permeable β -amino-3-hydroxyl-5-methyl-4-isoxazole-propionate (AMPA)/kainate (Ca-A/K) channels in a subset of hippocampal pyramidal neurons. Experimental Neurology, 2005, 193, 384-393.	2.0	136

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19	A new mitochondrial fluorescent zinc sensor. <i>Cell Calcium</i> , 2003, 34, 281-284.	1.1	132
20	Disruption of Glial Glutamate Transport by Reactive Oxygen Species Produced in Motor Neurons. <i>Journal of Neuroscience</i> , 2003, 23, 2627-2633.	1.7	129
21	Adenosine reduces cortical neuronal injury induced by oxygen or glucose deprivation in vitro. <i>Neuroscience Letters</i> , 1988, 89, 323-327.	1.0	123
22	Zn ²⁺ permeates Ca ²⁺ -permeable AMPA/kainate channels and triggers selective neural injury. <i>NeuroReport</i> , 1995, 6, 2553-2556.	0.6	107
23	Zn ²⁺ currents are mediated by calcium-permeable AMPA/Kainate channels in cultured murine hippocampal neurones. <i>Journal of Physiology</i> , 2002, 543, 35-48.	1.3	106
24	Histological and Functional Benefit Following Transplantation of Motor Neuron Progenitors to the Injured Rat Spinal Cord. <i>PLoS ONE</i> , 2010, 5, e11852.	1.1	90
25	Zn ²⁺ Influx Is Critical for Some Forms of Spreading Depression in Brain Slices. <i>Journal of Neuroscience</i> , 2008, 28, 8014-8024.	1.7	84
26	Mechanisms of seizure-induced α -transcriptional channelopathy TM of hyperpolarization-activated cyclic nucleotide gated (HCN) channels. <i>Neurobiology of Disease</i> , 2008, 29, 297-305.	2.1	82
27	Bicarbonate dependence of glutamate receptor activation by β -N-methylamino-l-alanine: Channel recording and study with related compounds. <i>Neuron</i> , 1989, 3, 321-326.	3.8	81
28	Mitochondrial Sequestration and Ca ²⁺ -Dependent Release of Cytosolic Zn ²⁺ Loads in Cortical Neurons. <i>Neurobiology of Disease</i> , 2002, 10, 100-108.	2.1	80
29	In vitro kainate injury to large, SMI-32(+) spinal neurons is Ca ²⁺ dependent. <i>NeuroReport</i> , 1995, 6, 945-947.	0.6	77
30	Dendritic localization of Ca ²⁺ -permeable AMPA/kainate channels in hippocampal pyramidal neurons. <i>Journal of Comparative Neurology</i> , 1999, 409, 250-260.	0.9	74
31	Estrogen-Receptor-Mediated Protection of Cerebral Endothelial Cell Viability and Mitochondrial Function after Ischemic Insult <i>in vitro</i> . <i>Journal of Cerebral Blood Flow and Metabolism</i> , 2010, 30, 545-554.	2.4	74
32	17 β -Estradiol prevents cell death and mitochondrial dysfunction by an estrogen receptor-dependent mechanism in astrocytes after oxygen-glucose deprivation/reperfusion. <i>Free Radical Biology and Medicine</i> , 2012, 52, 2151-2160.	1.3	72
33	Mitochondrial dysfunction in CA1 hippocampal neurons of the UBE3A deficient mouse model for Angelman syndrome. <i>Neuroscience Letters</i> , 2011, 487, 129-133.	1.0	65
34	Slow development of ALS-like spinal cord pathology in mutant valosin-containing protein gene knock-in mice. <i>Cell Death and Disease</i> , 2012, 3, e374-e374.	2.7	65
35	A Sodium Zinc Exchange Mechanism Is Mediating Extrusion of Zinc in Mammalian Cells. <i>Journal of Biological Chemistry</i> , 2004, 279, 4278-4284.	1.6	64
36	Differential Vulnerability of CA1 versus CA3 Pyramidal Neurons After Ischemia: Possible Relationship to Sources of Zn ²⁺ Accumulation and Its Entry into and Prolonged Effects on Mitochondria. <i>Journal of Neuroscience</i> , 2017, 37, 726-737.	1.7	64

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37	Cortical neurones with Ca ²⁺ -permeable AMPA/kainate channels display distinct receptor immunoreactivity and are GABAergic. <i>Neurobiology of Disease</i> , 1994, 1, 43-49.	2.1	61
38	Cortical neurones exhibiting kainate-activated Co ²⁺ -uptake are selectively vulnerable to AMPA/kainate receptor-mediated toxicity. <i>Neurobiology of Disease</i> , 1994, 1, 101-110.	2.1	61
39	Glutamate triggers preferential Zn ²⁺ flux through Ca ²⁺ -permeable AMPA channels and consequent ROS production. <i>NeuroReport</i> , 1999, 10, 1723-1727.	0.6	60
40	A progressive translational mouse model of human valosin ϵ -containing protein disease: The <i>VCP</i> ^{R155H/+} mouse. <i>Muscle and Nerve</i> , 2013, 47, 260-270.	1.0	58
41	Basal forebrain cholinergic neurons are selectively vulnerable to AMPA/kainate receptor-mediated neurotoxicity. <i>Neuroscience</i> , 1994, 60, 659-664.	1.1	57
42	Extracellular alkalinity exacerbates injury of cultured cortical neurons.. <i>Stroke</i> , 1992, 23, 1817-1821.	1.0	55
43	Mechanisms of Rapid Reactive Oxygen Species Generation in Response to Cytosolic Ca ²⁺ or Zn ²⁺ Loads in Cortical Neurons. <i>PLoS ONE</i> , 2013, 8, e83347.	1.1	53
44	Method for identifying neuronal cells suffering zinc toxicity by use of a novel fluorescent sensor. <i>Journal of Neuroscience Methods</i> , 2004, 139, 79-89.	1.3	52
45	Zinc and LTP. <i>Nature</i> , 1989, 338, 212-212.	13.7	50
46	Heterogeneity of Ca ²⁺ -Permeable AMPA/Kainate Channel Expression in Hippocampal Pyramidal Neurons: Fluorescence Imaging and Immunocytochemical Assessment. <i>Journal of Neuroscience</i> , 2003, 23, 10521-10530.	1.7	50
47	Intramitochondrial Zn ²⁺ accumulation via the Ca ²⁺ uniporter contributes to acute ischemic neurodegeneration. <i>Neurobiology of Disease</i> , 2014, 68, 137-144.	2.1	49
48	AMPA/kainate receptor-mediated damage to NADPH-diaphorase-containing neurons is Ca ²⁺ dependent. <i>Neuroscience Letters</i> , 1994, 167, 93-96.	1.0	48
49	Intrathecal infusion of BMAA induces selective motor neuron damage and astrogliosis in the ventral horn of the spinal cord. <i>Experimental Neurology</i> , 2014, 261, 1-9.	2.0	44
50	Zn ²⁺ entry through the mitochondrial calcium uniporter is a critical contributor to mitochondrial dysfunction and neurodegeneration. <i>Experimental Neurology</i> , 2020, 325, 113161.	2.0	41
51	Kainate-stimulated Zn ²⁺ uptake labels cortical neurons with Ca ²⁺ -permeable AMPA/kainate channels. <i>Brain Research</i> , 1998, 781, 45-56.	1.1	36
52	Mitochondrial Zn ²⁺ Accumulation: A Potential Trigger of Hippocampal Ischemic Injury. <i>Neuroscientist</i> , 2019, 25, 126-138.	2.6	35
53	Cultured basal forebrain cholinergic neurons in contact with cortical cells display synapses, enhanced morphological features, and decreased dependence on nerve growth factor. <i>Journal of Comparative Neurology</i> , 1996, 373, 451-465.	0.9	33
54	Intracellular Zn ²⁺ increases contribute to the progression of excitotoxic Ca ²⁺ increases in apical dendrites of CA1 pyramidal neurons. <i>Neuroscience</i> , 2009, 159, 104-114.	1.1	31

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55	Enhanced survival and morphological features of basal forebrain cholinergic neurons in vitro: Role of neurotrophins and other potential cortically derived cholinergic trophic factors. <i>Journal of Comparative Neurology</i> , 1999, 406, 156-170.	0.9	28
56	Zn ²⁺ -induced disruption of neuronal mitochondrial function: Synergism with Ca ²⁺ , critical dependence upon cytosolic Zn ²⁺ buffering, and contributions to neuronal injury. <i>Experimental Neurology</i> , 2018, 302, 181-195.	2.0	23
57	Secobarbital Attenuates Excitotoxicity but Potentiates Oxygen-Glucose Deprivation Neuronal Injury in Cortical Cell Culture. <i>Journal of Cerebral Blood Flow and Metabolism</i> , 1993, 13, 803-810.	2.4	22
58	Ca ²⁺ permeable AMPA/kainate channels permit rapid injurious Ca ²⁺ entry. <i>NeuroReport</i> , 1995, 6, 1089-1092.	0.6	21
59	BMAA – an unusual cyanobacterial neurotoxin. <i>Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders</i> , 2009, 10, 50-55.	2.3	21
60	Differential Vulnerability to Excitatory Amino Acid-Induced Toxicity and Selective Neuronal Loss in Neurodegenerative Diseases. <i>Canadian Journal of Neurological Sciences</i> , 1991, 18, 394-397.	0.3	20
61	Spinal cord neurons are vulnerable to rapidly triggered kainate neurotoxicity in vitro. <i>Brain Research</i> , 1995, 689, 265-270.	1.1	18
62	Kainate injury to cultured basal forebrain cholinergic neurons is Ca ²⁺ dependent. <i>NeuroReport</i> , 1994, 5, 1477-1480.	0.6	17
63	Ca ²⁺ and in vitro kainate damage to cortical and hippocampal SMI-32(+) neurons. <i>NeuroReport</i> , 1995, 6, 629-632.	0.6	13
64	Marked synergism between mutant SOD1 and glutamate transport inhibition in the induction of motor neuronal degeneration in spinal cord slice cultures. <i>Brain Research</i> , 2012, 1448, 153-162.	1.1	11
65	Cholinergic neurons from different subdivisions of the basal forebrain lack connectional specificity for cerebral cortical target sites in vitro. <i>Developmental Brain Research</i> , 1996, 97, 143-147.	2.1	8
66	Differential Vulnerability of CA1 versus CA3 Pyramidal Neurons After Ischemia: Possible Relationship to Sources of Zn ²⁺ Accumulation and Its Entry into and Prolonged Effects on Mitochondria. <i>Journal of Neuroscience</i> , 2017, 37, 726-737.	1.7	8
67	Zn ²⁺ chelation improves recovery by delaying spreading depression-like events. <i>NeuroReport</i> , 2010, 21, 1060-1064.	0.6	5
68	Cultured basal forebrain cholinergic neurons in contact with cortical cells display synapses, enhanced morphological features, and decreased dependence on nerve growth factor. , 1996, 373, 451.		1