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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	Zn2+ entry through the mitochondrial calcium uniporter is a critical contributor to mitochondrial dysfunction and neurodegeneration. Experimental Neurology, 2020, 325, 113161.	2.0	41
2	Mitochondrial Zn²⁺Accumulation: A Potential Trigger of Hippocampal Ischemic Injury. Neuroscientist, 2019, 25, 126-138.	2.6	35
3	Zn2+-induced disruption of neuronal mitochondrial function: Synergism with Ca2+, critical dependence upon cytosolic Zn2+ buffering, and contributions to neuronal injury. Experimental Neurology, 2018, 302, 181-195.	2.0	23
4	Differential Vulnerability of CA1 versus CA3 Pyramidal Neurons After Ischemia: Possible Relationship to Sources of Zn²⁺Accumulation and Its Entry into and Prolonged Effects on Mitochondria. Journal of Neuroscience, 2017, 37, 726-737.	1.7	64
5	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. Journal of Neuroscience, 2017, 37, 726-737.	1.7	8
6	Intramitochondrial Zn2+ accumulation via the Ca2+ uniporter contributes to acute ischemic neurodegeneration. Neurobiology of Disease, 2014, 68, 137-144.	2.1	49
7	Intrathecal infusion of BMAA induces selective motor neuron damage and astrogliosis in the ventral horn of the spinal cord. Experimental Neurology, 2014, 261, 1-9.	2.0	44
8	A progressive translational mouse model of human valosinâ€containing protein disease: The VCP^{R155H/+} mouse. Muscle and Nerve, 2013, 47, 260-270.	1.0	58
9	Mechanisms of Rapid Reactive Oxygen Species Generation in Response to Cytosolic Ca2+ or Zn2+ Loads in Cortical Neurons. PLoS ONE, 2013, 8, e83347.	1.1	53
10	Slow development of ALS-like spinal cord pathology in mutant valosin-containing protein gene knock-in mice. Cell Death and Disease, 2012, 3, e374-e374.	2.7	65
11	17Î²-Estradiol prevents cell death and mitochondrial dysfunction by an estrogen receptor-dependent mechanism in astrocytes after oxygenâ€glucose deprivation/reperfusion. Free Radical Biology and Medicine, 2012, 52, 2151-2160.	1.3	72
12	Marked synergism between mutant SOD1 and glutamate transport inhibition in the induction of motor neuronal degeneration in spinal cord slice cultures. Brain Research, 2012, 1448, 153-162.	1.1	11
13	Mitochondrial dysfunction in CA1 hippocampal neurons of the UBE3A deficient mouse model for Angelman syndrome. Neuroscience Letters, 2011, 487, 129-133.	1.0	65
14	Zinc: new clues to diverse roles in brain ischemia. Trends in Pharmacological Sciences, 2011, 32, 480-486.	4.0	162
15	Zn2+ chelation improves recovery by delaying spreading depression-like events. NeuroReport, 2010, 21, 1060-1064.	0.6	5
16	Estrogen-Receptor-Mediated Protection of Cerebral Endothelial Cell Viability and Mitochondrial Function after Ischemic Insult in vitro. Journal of Cerebral Blood Flow and Metabolism, 2010, 30, 545-554.	2.4	74
17	Histological and Functional Benefit Following Transplantation of Motor Neuron Progenitors to the Injured Rat Spinal Cord. PLoS ONE, 2010, 5, e11852.	1.1	90
18	BMAA â€“ an unusual cyanobacterial neurotoxin. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders, 2009, 10, 50-55.	2.3	21

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19	Intracellular Zn²⁺Accumulation Contributes to Synaptic Failure, Mitochondrial Depolarization, and Cell Death in an Acute Slice Oxygenâ€“Glucose Deprivation Model of Ischemia. Journal of Neuroscience, 2009, 29, 1105-1114.	1.7	148
20	Intracellular Zn2+ increases contribute to the progression of excitotoxic Ca2+ increases in apical dendrites of CA1 pyramidal neurons. Neuroscience, 2009, 159, 104-114.	1.1	31
21	Mechanisms of seizure-induced â€˜transcriptional channelopathyâ€™ of hyperpolarization-activated cyclic nucleotide gated (HCN) channels. Neurobiology of Disease, 2008, 29, 297-305.	2.1	82
22	Zn²⁺Influx Is Critical for Some Forms of Spreading Depression in Brain Slices. Journal of Neuroscience, 2008, 28, 8014-8024.	1.7	84
23	Calcium-permeable AMPA channels in neurodegenerative disease and ischemia. Current Opinion in Neurobiology, 2006, 16, 281-287.	2.0	227
24	Tumor necrosis-factor-alpha (TNF-Î±) induces rapid insertion of Ca2+-permeable Î±-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
25	A Sodium Zinc Exchange Mechanism Is Mediating Extrusion of Zinc in Mammalian Cells. Journal of Biological Chemistry, 2004, 279, 4278-4284.	1.6	64
26	Method for identifying neuronal cells suffering zinc toxicity by use of a novel fluorescent sensor. Journal of Neuroscience Methods, 2004, 139, 79-89.	1.3	52
27	Excitotoxic and oxidative cross-talk between motor neurons and glia in ALS pathogenesis. Trends in Neurosciences, 2004, 27, 17-23.	4.2	152
28	A new mitochondrial fluorescent zinc sensor. Cell Calcium, 2003, 34, 281-284.	1.1	132
29	Modulation of mitochondrial function by endogenous Zn2+ pools. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 6157-6162.	3.3	387
30	Heterogeneity of Ca2+-Permeable AMPA/Kainate Channel Expression in Hippocampal Pyramidal Neurons: Fluorescence Imaging and Immunocytochemical Assessment. Journal of Neuroscience, 2003, 23, 10521-10530.	1.7	50
31	Disruption of Glial Glutamate Transport by Reactive Oxygen Species Produced in Motor Neurons. Journal of Neuroscience, 2003, 23, 2627-2633.	1.7	129
32	Mitochondrial Sequestration and Ca2+-Dependent Release of Cytosolic Zn2+ Loads in Cortical Neurons. Neurobiology of Disease, 2002, 10, 100-108.	2.1	80
33	Blockade of Ca²⁺-Permeable AMPA/Kainate Channels Decreases Oxygenâ€“Glucose Deprivation-Induced Zn²⁺Accumulation and Neuronal Loss in Hippocampal Pyramidal Neurons. Journal of Neuroscience, 2002, 22, 1273-1279.	1.7	139
34	Measuring zinc in living cells.. Cell Calcium, 2002, 31, 245-251.	1.1	232
35	Zn 2+ currents are mediated by calciumâ€permeable AMPA/Kainate channels in cultured murine hippocampal neurones. Journal of Physiology, 2002, 543, 35-48.	1.3	106
36	Zn2+ 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

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37	AMPA/kainate receptor-triggered Zn2+entry into cortical neurons induces mitochondrial Zn2+uptake and persistent mitochondrial dysfunction. European Journal of Neuroscience, 2000, 12, 3813-3818.	1.2	140
38	AMPA Exposures Induce Mitochondrial Ca²⁺Overload and ROS Generation in Spinal Motor NeuronsIn Vitro. Journal of Neuroscience, 2000, 20, 240-250.	1.7	284
39	Zn2+: a novel ionic mediator of neural injury in brain disease. Trends in Pharmacological Sciences, 2000, 21, 395-401.	4.0	536
40	Ca2+â€“Zn2+ permeable AMPA or kainate receptors: possible key factors in selective neurodegeneration. Trends in Neurosciences, 2000, 23, 365-371.	4.2	232
41	Preferential Zn2+ influx through Ca2+-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
42	Enhanced survival and morphological features of basal forebrain cholinergic neurons in vitro: Role of neurotrophins and other potential cortically derived cholinergic trophic factors. Journal of Comparative Neurology, 1999, 406, 156-170.	0.9	28
43	Dendritic localization of Ca2+-permeable AMPA/kainate channels in hippocampal pyramidal neurons. Journal of Comparative Neurology, 1999, 409, 250-260.	0.9	74
44	Glutamate triggers preferential Zn2+ flux through Ca2+ permeable AMPA channels and consequent ROS production. NeuroReport, 1999, 10, 1723-1727.	0.6	60
45	Kainate-stimulated Zn2+ uptake labels cortical neurons with Ca2+-permeable AMPA/kainate channels. Brain Research, 1998, 781, 45-56.	1.1	36
46	Cholinergic neurons from different subdivisions of the basal forebrain lack connectional specificity for cerebral cortical target sites in vitro. Developmental Brain Research, 1996, 97, 143-147.	2.1	8
47	Cultured basal forebrain cholinergic neurons in contact with cortical cells display synapses, enhanced morphological features, and decreased dependence on nerve growth factor. Journal of Comparative Neurology, 1996, 373, 451-465.	0.9	33
48	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
49	Zn2+ permeates Ca2+permeable AMPA/kainate channels and triggers selective neural injury. NeuroReport, 1995, 6, 2553-2556.	0.6	107
50	Ca2+ and in vitro kainate damage to cortical and hippocampal SMI-32(+) neurons. NeuroReport, 1995, 6, 629-632.	0.6	13
51	In vitro kainate injury to large, SMI-32(+) spinal neurons is Ca2+ dependent. NeuroReport, 1995, 6, 945-947.	0.6	77
52	Ca2+ permeable AMPA/kainate channels permit rapid injurious Ca2+ entry. NeuroReport, 1995, 6, 1089-1092.	0.6	21
53	Spinal cord neurons are vulnerable to rapidly triggered kainate neurotoxicity in vitro. Brain Research, 1995, 689, 265-270.	1.1	18
54	Basal forebrain cholinergic neurons are selectively vulnerable to AMPA/kainate receptor-mediated neurotoxicity. Neuroscience, 1994, 60, 659-664.	1.1	57

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55	Cortical neurones with Ca2+permeable AMPA/kainate channels display distinct receptor immunoreactivity and are GABAergic. Neurobiology of Disease, 1994, 1, 43-49.	2.1	61
56	Cortical neurones exhibiting kainate-activated Co2+uptake are selectively vulnerable to AMPA/kainate receptor-mediated toxicity. Neurobiology of Disease, 1994, 1, 101-110.	2.1	61
57	AMPA/kainate receptor-mediated damage to NADPH-diaphorase-containing neurons is Ca2+ dependent. Neuroscience Letters, 1994, 167, 93-96.	1.0	48
58	Kainate injury to cultured basal forebrain cholinergic neurons is Ca2+ dependent. NeuroReport, 1994, 5, 1477-1480.	0.6	17
59	Secobarbital Attenuates Excitotoxicity but Potentiates Oxygenâ€"Glucose Deprivation Neuronal Injury in Cortical Cell Culture. Journal of Cerebral Blood Flow and Metabolism, 1993, 13, 803-810.	2.4	22
60	AMPA receptor activation potentiates zinc neurotoxicity. Neuron, 1993, 10, 43-49.	3.8	271
61	Extracellular alkalinity exacerbates injury of cultured cortical neurons.. Stroke, 1992, 23, 1817-1821.	1.0	55
62	Differential Vulnerability to Excitatory Amino Acid-Induced Toxicity and Selective Neuronal Loss in Neurodegenerative Diseases. Canadian Journal of Neurological Sciences, 1991, 18, 394-397.	0.3	20
63	Zinc and LTP. Nature, 1989, 338, 212-212.	13.7	50
64	Bicarbonate dependence of glutamate receptor activation by Î²-N-methylamino-l-alanine: Channel recording and study with related compounds. Neuron, 1989, 3, 321-326.	3.8	81
65	Neurotoxicity ofÎ²-N-methylamino-l-alanine (BMAA) andÎ²-N-oxalylamino-l-alamine (BOAA) on cultured cortical neurons. Brain Research, 1989, 497, 64-71.	1.1	205
66	Adenosine reduces cortical neuronal injury induced by oxygen or glucose deprivation in vitro. Neuroscience Letters, 1988, 89, 323-327.	1.0	123
67	Beta-N-methylamino-L-alanine neurotoxicity: requirement for bicarbonate as a cofactor. Science, 1988, 241, 973-975.	6.0	196
68	Ketamine protects cultured neocortical neurons from hypoxic injury. Brain Research, 1986, 380, 186-190.	1.1	144