

Jing Wu

List of Publications by Year in descending order

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Version: 2024-02-01

26
papers

946
citations

516710

16
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552781

26
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26
all docs

26
docs citations

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times ranked

1339
citing authors

#	ARTICLE	IF	CITATIONS
1	Mitochondrial ATP synthase c-subunit leak channel triggers cell death upon loss of its F1 subcomplex. <i>Cell Death and Differentiation</i> , 2022, 29, 1874-1887.	11.2	23
2	ATP Synthase c-Subunit Leak Causes Aberrant Cellular Metabolism in Fragile X Syndrome. <i>Cell</i> , 2020, 182, 1170-1185.e9.	28.9	64
3	Elamipretide Attenuates Pyroptosis and Perioperative Neurocognitive Disorders in Aged Mice. <i>Frontiers in Cellular Neuroscience</i> , 2020, 14, 251.	3.7	18
4	Iron overload contributes to general anaesthesia-induced neurotoxicity and cognitive deficits. <i>Journal of Neuroinflammation</i> , 2020, 17, 110.	7.2	68
5	Parkinson's disease protein DJ-1 regulates ATP synthase protein components to increase neuronal process outgrowth. <i>Cell Death and Disease</i> , 2019, 10, 469.	6.3	70
6	NLRP3/Caspase-1 Pathway-Induced Pyroptosis Mediated Cognitive Deficits in a Mouse Model of Sepsis-Associated Encephalopathy. <i>Inflammation</i> , 2019, 42, 306-318.	3.8	145
7	Renal Prognosis and Related Risk Factors for Henoch-Schönlein Purpura Nephritis: A Chinese Adult Patient Cohort. <i>Scientific Reports</i> , 2018, 8, 5585.	3.3	21
8	Significance of histological crescent formation in patients with IgA vasculitis (Henoch-Schönlein) Tj ETQq0 0 0 rgBT /Overlock 10 Tf 50	1.8	10
9	Inhibiting the NLRP3 Inflammasome With MCC950 Ameliorates Isoflurane-Induced Pyroptosis and Cognitive Impairment in Aged Mice. <i>Frontiers in Cellular Neuroscience</i> , 2018, 12, 426.	3.7	51
10	NLRP3 inflammasome-dependent pyroptosis is proposed to be involved in the mechanism of age-dependent isoflurane-induced cognitive impairment. <i>Journal of Neuroinflammation</i> , 2018, 15, 266.	7.2	12
11	Elamipretide (SS-31) Ameliorates Isoflurane-Induced Long-Term Impairments of Mitochondrial Morphogenesis and Cognition in Developing Rats. <i>Frontiers in Cellular Neuroscience</i> , 2017, 11, 119.	3.7	36
12	Amelioration of oxidative stress-induced phenotype loss of parvalbumin interneurons might contribute to the beneficial effects of environmental enrichment in a rat model of post-traumatic stress disorder. <i>Behavioural Brain Research</i> , 2016, 312, 84-92.	2.2	34
13	BDNF pathway is involved in the protective effects of SS-31 on isoflurane-induced cognitive deficits in aging mice. <i>Behavioural Brain Research</i> , 2016, 305, 115-121.	2.2	48
14	Environmental Enrichment Prevent the Juvenile Hypoxia-Induced Developmental Loss of Parvalbumin-Immunoreactive Cells in the Prefrontal Cortex and Neurobehavioral Alterations Through Inhibition of NADPH Oxidase-2-Derived Oxidative Stress. <i>Molecular Neurobiology</i> , 2016, 53, 7341-7350.	4.0	10
15	Protective Effects of Antioxidant Peptide SS-31 Against Multiple Organ Dysfunctions During Endotoxemia. <i>Inflammation</i> , 2016, 39, 54-64.	3.8	19
16	Developmental loss of parvalbumin-positive cells in the prefrontal cortex and psychiatric anxiety after intermittent hypoxia exposures in neonatal rats might be mediated by NADPH oxidase-2. <i>Behavioural Brain Research</i> , 2016, 296, 134-140.	2.2	13
17	Mitochondrion-Targeted Peptide SS-31 Inhibited Oxidized Low-Density Lipoproteins-Induced Foam Cell Formation through both ROS Scavenging and Inhibition of Cholesterol Influx in RAW264.7 Cells. <i>Molecules</i> , 2015, 20, 21287-21297.	3.8	31
18	How to treat type 2 diabetes-induced encephalopathy: Regulating the autophagic pathway?. <i>Nutrition</i> , 2015, 31, 1055.	2.4	2

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19	Intermittent Hypoxia-Induced Parvalbumin-Immunoreactive Interneurons Loss and Neurobehavioral Impairment is Mediated by NADPH-Oxidase-2. <i>Neurochemical Research</i> , 2015, 40, 1232-1242.	3.3	16
20	Autophagic activation may be involved in the mechanism of hesperidin's therapeutic effects on cognitive impairment. <i>Journal of the Neurological Sciences</i> , 2015, 351, 202-203.	0.6	2
21	Mitochondria-Targeted Peptide Reverses Mitochondrial Dysfunction and Cognitive Deficits in Sepsis-Associated Encephalopathy. <i>Molecular Neurobiology</i> , 2015, 52, 783-791.	4.0	84
22	A Mitochondrion-Targeted Antioxidant Ameliorates Isoflurane-Induced Cognitive Deficits in Aging Mice. <i>PLoS ONE</i> , 2015, 10, e0138256.	2.5	36
23	Valproic Acid Attenuates Lipopolysaccharide-Induced Acute Lung Injury in Mice. <i>Inflammation</i> , 2013, 36, 1453-1459.	3.8	43
24	Class I Histone Deacetylase Inhibitor Valproic Acid Reverses Cognitive Deficits in a Mouse Model of Septic Encephalopathy. <i>Neurochemical Research</i> , 2013, 38, 2440-2449.	3.3	60
25	Alpha 2A-adrenoreceptor blockade improves sepsis-induced acute lung injury accompanied with depressed high mobility group box-1 levels in rats. <i>Cytokine</i> , 2012, 60, 639-645.	3.2	15
26	Effects of Combined Levosimendan and Vasopressin on Pulmonary Function in Porcine Septic Shock. <i>Inflammation</i> , 2012, 35, 871-880.	3.8	15