Shirley Yan

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/4997720/publications.pdf

Version: 2024-02-01

39 papers	1,960 citations	218677 26 h-index	302126 39 g-index
39 all docs	39 docs citations	39 times ranked	3212 citing authors

#	Article	IF	Citations
1	Mitochondrial permeability transition pore is a potential drug target for neurodegeneration. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 1267-1272.	3.8	232
2	PINK1 signalling rescues amyloid pathology and mitochondrial dysfunction in Alzheimerâ€™s disease. Brain, 2017, 140, 3233-3251.	7.6	211
3	Inhibition of ERK-DLP1 signaling and mitochondrial division alleviates mitochondrial dysfunction in Alzheimer's disease cybrid cell. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 220-231.	3.8	151
4	Drp1-Mediated Mitochondrial Abnormalities Link to Synaptic Injury in Diabetes Model. Diabetes, 2015, 64, 1728-1742.	0.6	121
5	Multi-faced neuroprotective effects of geniposide depending on the RAGE-mediated signaling in an Alzheimer mouse model. Neuropharmacology, 2015, 89, 175-184.	4.1	80
6	Mitochondrial permeability transition pore: a potential drug target for neurodegeneration. Drug Discovery Today, 2018, 23, 1983-1989.	6.4	77
7	Mfn2 is Required for Mitochondrial Development and Synapse Formation in Human Induced Pluripotent Stem Cells/hiPSC Derived Cortical Neurons. Scientific Reports, 2016, 6, 31462.	3.3	74
8	Cyclophilin D deficiency rescues AÎ²-impaired PKA/CREB signaling and alleviates synaptic degeneration. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 2517-2527.	3.8	73
9	Increased neuronal PreP activity reduces AÎ² accumulation, attenuates neuroinflammation and improves mitochondrial and synaptic function in Alzheimer disease's mouse model. Human Molecular Genetics, 2015, 24, 5198-5210.	2.9	70
10	Entorhinal Cortex dysfunction can be rescued by inhibition of microglial RAGE in an Alzheimerâ€™s disease mouse model. Scientific Reports, 2017, 7, 42370.	3.3	64
11	RAGE mediates AÎ² accumulation in a mouse model of Alzheimerâ€™s disease via modulation of Î²- and Î³-secretase activity. Human Molecular Genetics, 2018, 27, 1002-1014.	2.9	62
12	Oxidative stress-mediated activation of extracellular signal-regulated kinase contributes to mild cognitive impairment-related mitochondrial dysfunction. Free Radical Biology and Medicine, 2014, 75, 230-240.	2.9	55
13	RAGE Inhibition in Microglia Prevents Ischemia-Dependent Synaptic Dysfunction in an Amyloid-Enriched Environment. Journal of Neuroscience, 2014, 34, 8749-8760.	3.6	47
14	Blockade of Drp1 rescues oxidative stress-induced osteoblast dysfunction. Biochemical and Biophysical Research Communications, 2015, 468, 719-725.	2.1	47
15	Astrocytes Attenuate Mitochondrial Dysfunctions in Human Dopaminergic Neurons Derived from iPSC. Stem Cell Reports, 2018, 10, 366-374.	4.8	43
16	RAGE is a key cellular target for Abeta-induced perturbation in Alzheimer's disease. Frontiers in Bioscience - Scholar, 2012, S4, 240.	2.1	41
17	F1F0 ATP Synthaseâ€“Cyclophilin D Interaction Contributes to Diabetes-Induced Synaptic Dysfunction and Cognitive Decline. Diabetes, 2016, 65, 3482-3494.	0.6	41
18	Identification of a Small Molecule Cyclophilin D Inhibitor for Rescuing AÎ²-Mediated Mitochondrial Dysfunction. ACS Medicinal Chemistry Letters, 2016, 7, 294-299.	2.8	38

#	Article	IF	Citations
19	Mitochondrial Dysfunction Triggers Synaptic Deficits via Activation of p38 MAP Kinase Signaling in Differentiated Alzheimerâ€™s Disease Trans-Mitochondrial Cybrid Cells. Journal of Alzheimer's Disease, 2017, 59, 223-239.	2.6	38
20	Overexpression of endophilin A1 exacerbates synaptic alterations in a mouse model of Alzheimerâ€™s disease. Nature Communications, 2018, 9, 2968.	12.8	37
21	Increased Electron Paramagnetic Resonance Signal Correlates with Mitochondrial Dysfunction and Oxidative Stress in an Alzheimerâ€™s disease Mouse Brain. Journal of Alzheimer's Disease, 2016, 51, 571-580.	2.6	36
22	Age-dependent accumulation of dicarbonyls and advanced glycation endproducts (AGEs) associates with mitochondrial stress. Free Radical Biology and Medicine, 2021, 164, 429-438.	2.9	33
23	High Dietary Advanced Glycation End Products Impair Mitochondrial and Cognitive Function. Journal of Alzheimer's Disease, 2020, 76, 165-178.	2.6	33
24	Antioxidants Rescue Mitochondrial Transport in Differentiated Alzheimerâ€™s Disease Trans-Mitochondrial Cybrid Cells. Journal of Alzheimer's Disease, 2016, 54, 679-690.	2.6	32
25	Synergistic Exacerbation of Mitochondrial and Synaptic Dysfunction and Resultant Learning and Memory Deficit in a Mouse Model of Diabetic Alzheimer's Disease. Journal of Alzheimer's Disease, 2014, 43, 451-463.	2.6	30
26	Development and Dynamic Regulation of Mitochondrial Network in Human Midbrain Dopaminergic Neurons Differentiated from iPSCs. Stem Cell Reports, 2016, 7, 678-692.	4.8	30
27	Identification of human presequence protease (hPreP) agonists for the treatment of Alzheimer's disease. European Journal of Medicinal Chemistry, 2014, 76, 506-516.	5.5	25
28	Mitochondrial oxidative stress contributes to the pathological aggregation and accumulation of tau oligomers in Alzheimerâ€™s disease. Human Molecular Genetics, 2022, 31, 2498-2507.	2.9	25
29	Overexpression of 17Î²-hydroxysteroid dehydrogenase type 10 increases pheochromocytoma cell growth and resistance to cell death. BMC Cancer, 2015, 15, 166.	2.6	19
30	Anxiety and task performance changes in an aging mouse model. Biochemical and Biophysical Research Communications, 2019, 514, 246-251.	2.1	17
31	Unlocking the Door to Neuronal Woes in Alzheimerâ€™s Disease: AÎ² and Mitochondrial Permeability Transition Pore. Pharmaceuticals, 2010, 3, 1936-1948.	3.8	12
32	Determination of Small Molecule ABAD Inhibitors Crossing Blood-Brain Barrier and Pharmacokinetics. Journal of Alzheimer's Disease, 2014, 42, 333-344.	2.6	11
33	The potential role of damage-associated molecular patterns derived from mitochondria in osteocyte apoptosis and bone remodeling. Bone, 2014, 62, 67-68.	2.9	11
34	PINK1 Activation Attenuates Impaired Neuronal-Like Differentiation and Synaptogenesis and Mitochondrial Dysfunction in Alzheimerâ€™s Disease Trans-Mitochondrial Cybrid Cells. Journal of Alzheimer's Disease, 2021, 81, 1749-1761.	2.6	11
35	NR2B-dependent cyclophilin D translocation suppresses the recovery of synaptic transmission after oxygenâ€“glucose deprivation. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 2225-2234.	3.8	9
36	Identification and Characterization of Amyloid-Î² Accumulation in Synaptic Mitochondria. Methods in Molecular Biology, 2018, 1779, 415-433.	0.9	9

#	Article	IF	Citations
37	Gain of PITRM1 peptidase in cortical neurons affords protection of mitochondrial and synaptic function in an advanced age mouse model of Alzheimerâ€™s disease. Aging Cell, 2021, 20, e13368.	6.7	6
38	High-resolution crystal structures of two crystal forms of human cyclophilin D in complex with PEG 400 molecules. Acta Crystallographica Section F, Structural Biology Communications, 2014, 70, 717-722.	0.8	5
39	From a cell's viewpoint: targeting mitochondria in Alzheimer's disease. Drug Discovery Today: Therapeutic Strategies, 2013, 10, e91-e98.	0.5	4