Shi Fang Yan

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/3639655/publications.pdf

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		394421	642732
24	3,879	19	23
papers	citations	h-index	g-index

24	24	24	5398
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	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
2	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
3	High Dietary Advanced Glycation End Products Impair Mitochondrial and Cognitive Function. Journal of Alzheimer's Disease, 2020, 76, 165-178.	2.6	33
4	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
5	Overexpression of endophilin A1 exacerbates synaptic alterations in a mouse model of Alzheimerâ€™s disease. Nature Communications, 2018, 9, 2968.	12.8	37
6	Mitochondrial permeability transition pore: a potential drug target for neurodegeneration. Drug Discovery Today, 2018, 23, 1983-1989.	6.4	77
7	Identification and Characterization of Amyloid-Î² Accumulation in Synaptic Mitochondria. Methods in Molecular Biology, 2018, 1779, 415-433.	0.9	9
8	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
9	PINK1 signalling rescues amyloid pathology and mitochondrial dysfunction in Alzheimerâ€™s disease. Brain, 2017, 140, 3233-3251.	7.6	211
10	Polyol pathway and RAGE: a central metabolic and signaling axis in diabetic complications. Expert Review of Endocrinology and Metabolism, 2010, 5, 65-75.	2.4	4
11	Soluble RAGE: Therapy and biomarker in unraveling the RAGE axis in chronic disease and aging. Biochemical Pharmacology, 2010, 79, 1379-1386.	4.4	150
12	The RAGE Axis. Circulation Research, 2010, 106, 842-853.	4.5	337
13	Role of mDia1 in RAGE signaling and smooth muscle cell migration. FASEB Journal, 2010, 24, 895.6.	0.5	0
14	Tempering the wrath of RAGE: An emerging therapeutic strategy against diabetic complications, neurodegeneration, and inflammation. Annals of Medicine, 2009, 41, 408-422.	3.8	115
15	Receptor for AGE (RAGE) and its ligandsâ€”cast into leading roles in diabetes and the inflammatory response. Journal of Molecular Medicine, 2009, 87, 235-247.	3.9	199
16	Mechanisms of Disease: advanced glycation end-products and their receptor in inflammation and diabetes complications. Nature Clinical Practice Endocrinology and Metabolism, 2008, 4, 285-293.	2.8	346
17	The biology of RAGE and its ligands: Uncovering mechanisms at the heart of diabetes and its complications. Current Diabetes Reports, 2007, 7, 146-153.	4.2	51
18	Receptor for Advanced Glycation Endproducts (RAGE): a formidable force in the pathogenesis of the cardiovascular complications of diabetes & aging. Current Molecular Medicine, 2007, 7, 699-710.	1.3	27

#	Article	IF	Citations
19	Receptor for Advanced Glycation End Products and the Cardiovascular Complications of Diabetes and Beyond: Lessons from AGEing. Endocrinology and Metabolism Clinics of North America, 2006, 35, 511-524.	3.2	20
20	The ligand/RAGE axis: Lighting the fuse and igniting vascular stress. Current Atherosclerosis Reports, 2006, 8, 232-239.	4.8	17
21	RAGE potentiates AÎ²-induced perturbation of neuronal function in transgenic mice. EMBO Journal, 2004, 23, 4096-4105.	7.8	311
22	Glycation, Inflammation, and RAGE. Circulation Research, 2003, 93, 1159-1169.	4.5	464
23	The multiligand receptor RAGE as a progression factor amplifying immune and inflammatory responses. Journal of Clinical Investigation, 2001, 108, 949-955.	8.2	916
24	Non-enzymatically glycated tau in Alzheimer's disease induces neuronal oxidant stress resulting in cytokine gene expression and release of amyloid Î²-peptide. Nature Medicine, 1995, 1, 693-699.	30.7	416