Wiep Scheper

List of Publications by Year
in descending order

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66 papers	11,461 citations	117625 34 h-index	118850 62 g-index
70 all docs	70 docs citations	70 times ranked	24440 citing authors

#	Article	IF	Citations
1	Neuronâ€specific translational control shift ensures proteostatic resilience during <scp>ER</scp> stress. EMBO Journal, 2022, 41, .	7.8	11
2	Unfolded protein response activation in C9orf72 frontotemporal dementia is associated with dipeptide pathology and granulovacuolar degeneration in granule cells. Brain Pathology, 2021, 31, 163-173.	4.1	18
3	In vivo tau pathology is associated with synaptic loss and altered synaptic function. Alzheimer's Research and Therapy, 2021, 13, 35.	6.2	47
4	Granulovacuolar degeneration bodies: red alert for neurons with MAPT/tau pathology. Autophagy, 2020, 16, 173-175.	9.1	5
5	No evidence for cellâ€toâ€cell transmission of the unfolded protein response in cell culture. Journal of Neurochemistry, 2020, 152, 208-220.	3.9	10
6	The UPR in Neurodegenerative Disease: Not Just an Inside Job. Biomolecules, 2020, 10, 1090.	4.0	15
7	Untangling the origin and function of granulovacuolar degeneration bodies in neurodegenerative proteinopathies. Acta Neuropathologica Communications, 2020, 8, 153.	5.2	10
8	The seeding of tau pathology alters the endolysosomal system. Alzheimer's and Dementia, 2020, 16, e038117.	0.8	0
9	Neuronâ€selective induction of granulovacuolar degeneration bodies: A lysosomal stress response to tau aggregation?. Alzheimer's and Dementia, 2020, 16, e039378.	0.8	0
10	Regional tau pathology is associated with loss of synapses and reduced synaptic activity: A combined [18 F]flortaucipir, [11 C]UCBâ€J and magnetoencephalography study. Alzheimer's and Dementia, 2020, 16, e045806.	0.8	0
11	Specific targeting of a highly toxic subpopulation of AÎ²42 oligomers for the treatment of Alzheimerâ€™s disease. Alzheimer's and Dementia, 2020, 16, e043003.	0.8	1
12	Granulovacuolar degeneration bodies are neuron-selective lysosomal structures induced by intracellular tau pathology. Acta Neuropathologica, 2019, 138, 943-970.	7.7	48
13	Endolysosome and Autolysosome Dysfunction in Alzheimerâ€™s Disease: Where Intracellular and Extracellular Meet. CNS Drugs, 2019, 33, 639-648.	5.9	23
14	Unconventional secretion factor GRASP55 is increased by pharmacological unfolded protein response inducers in neurons. Scientific Reports, 2019, 9, 1567.	3.3	17
15	The UPR reduces glucose metabolism via IRE1 signaling. Biochimica Et Biophysica Acta - Molecular Cell Research, 2017, 1864, 655-665.	4.1	34
16	Insulin deficiency results in reversible protein kinase A activation and tau phosphorylation. Neurobiology of Disease, 2017, 103, 163-173.	4.4	26
17	Proteasome Activation by Small Molecules. Cell Chemical Biology, 2017, 24, 725-736.e7.	5.2	113
18	IRE1 signaling exacerbates Alzheimerâ€™s disease pathogenesis. Acta Neuropathologica, 2017, 134, 489-506.	7.7	147

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19	Alpha-synuclein induces the unfolded protein response in Parkinsonâ€™s disease SNCA triplication iPSC-derived neurons. Human Molecular Genetics, 2017, 26, 4441-4450.	2.9	119
20	Stall in Canonical Autophagy-Lysosome Pathways Prompts Nucleophagy-Based Nuclear Breakdown in Neurodegeneration. Current Biology, 2017, 27, 3626-3642.e6.	3.9	47
21	[P2â€“203]: SPPL2B: A NOVEL PROTEIN RELATED TO TAU PATHOLOGY IN ALZHEIMER's DISEASE?. Alzheimer's and Dementia, 2017, 13, P684.	0.8	0
22	Effects of Fat and Sugar, Either Consumed or Infused toward the Brain, on Hypothalamic ER Stress Markers. Frontiers in Neuroscience, 2017, 11, 270.	2.8	10
23	Targeting neuronal MAPK14/p38Î± activity to modulate autophagy in the Alzheimer disease brain. Autophagy, 2016, 12, 2516-2520.	9.1	40
24	Activation of the unfolded protein response and granulovacuolar degeneration are not common features of human prion pathology. Acta Neuropathologica Communications, 2016, 4, 113.	5.2	11
25	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	9.1	4,701
26	Neuroinflammation is not a Prerequisite for Diabetes-induced Tau Phosphorylation. Frontiers in Neuroscience, 2015, 9, 432.	2.8	9
27	The unfolded protein response in neurodegenerative diseases: a neuropathological perspective. Acta Neuropathologica, 2015, 130, 315-331.	7.7	305
28	Liposomes bi-functionalized with phosphatidic acid and an ApoE-derived peptide affect AÎ² aggregation features and cross the bloodâ€“brain-barrier: Implications for therapy of Alzheimer disease. Nanomedicine: Nanotechnology, Biology, and Medicine, 2014, 10, 1583-1590.	3.3	121
29	Amyotrophic lateral sclerosis (ALS)-associated VAPB-P56S inclusions represent an ER quality control compartment. Acta Neuropathologica Communications, 2013, 1, 24.	5.2	46
30	Intracellular accumulation of aggregated pyroglutamate amyloid beta: convergence of aging and AÎ² pathology at the lysosome. Age, 2013, 35, 673-687.	3.0	50
31	Unfolded protein response activates glycogen synthase kinase-3 via selective lysosomal degradation. Neurobiology of Aging, 2013, 34, 1759-1771.	3.1	42
32	Inhibition of Endoplasmic Reticulum Associated Degradation Reduces Endoplasmic Reticulum Stress and Alters Lysosomal Morphology and Distribution. Molecules and Cells, 2013, 35, 291-297.	2.6	17
33	A New PERKspective on Neurodegeneration. Science Translational Medicine, 2013, 5, 206fs37.	12.4	19
34	Ubiquilin 2 Is Not Associated with Tau Pathology. PLoS ONE, 2013, 8, e76598.	2.5	8
35	Rab6 is a Modulator of the Unfolded Protein Response: Implications for Alzheimer's Disease. Journal of Alzheimer's Disease, 2012, 28, 917-929.	2.6	25
36	Activation of the Unfolded Protein Response Is an Early Event in Alzheimerâ€™s and Parkinsonâ€™s Disease. Neurodegenerative Diseases, 2012, 10, 212-215.	1.4	173

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37	STOX1A induces phosphorylation of tau proteins at epitopes hyperphosphorylated in Alzheimer's disease. Neuroscience Letters, 2012, 528, 104-109.	2.1	8
38	PEGylated Nanoparticles Bind to and Alter Amyloid-Beta Peptide Conformation: Toward Engineering of Functional Nanomedicines for Alzheimerâ€™s Disease. ACS Nano, 2012, 6, 5897-5908.	14.6	164
39	Endoplasmic reticulum: The unfolded protein response is tangled in neurodegeneration. International Journal of Biochemistry and Cell Biology, 2012, 44, 1295-1298.	2.8	68
40	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	9.1	3,122
41	Disturbed Ca2+ Homeostasis Increases Glutaminyl Cyclase Expression; Connecting Two Early Pathogenic Events in Alzheimerâ€™s Disease In Vitro. PLoS ONE, 2012, 7, e44674.	2.5	23
42	The unfolded protein response is associated with early tau pathology in the hippocampus of tauopathies. Journal of Pathology, 2012, 226, 693-702.	4.5	153
43	Versatile and Efficient Targeting Using a Single Nanoparticulate Platform: Application to Cancer and Alzheimerâ€™s Disease. ACS Nano, 2012, 6, 5866-5879.	14.6	127
44	The unfolded protein response and proteostasis in Alzheimer disease. Autophagy, 2011, 7, 910-911.	9.1	82
45	The Pre-Eclampsia Gene STOX1 Controls a Conserved Pathway in Placenta and Brain Upregulated in Late-Onset Alzheimer's Disease. Journal of Alzheimer's Disease, 2010, 19, 673-679.	2.6	40
46	Stability of AÎ² (1-42) peptide fibrils as consequence of environmental modifications. European Biophysics Journal, 2010, 39, 1613-1623.	2.2	18
47	New Method Based on Capillary Electrophoresis with Laser-Induced Fluorescence Detection (CE-LIF) to Monitor Interaction between Nanoparticles and the Amyloid-Î² Peptide. Analytical Chemistry, 2010, 82, 10083-10089.	6.5	50
48	Endoplasmic Reticulum Protein Quality Control in Neurodegenerative Disease: The Good, the Bad and the Therapy. Current Medicinal Chemistry, 2009, 16, 615-626.	2.4	81
49	Endoplasmic Reticulum Stress in Neurodegeneration. Focus on Structural Biology, 2009, , 111-132.	0.1	4
50	The Unfolded Protein Response Is Activated in Pretangle Neurons in Alzheimer's Disease Hippocampus. American Journal of Pathology, 2009, 174, 1241-1251.	3.8	512
51	Oligomer-specific AÎ² toxicity in cell models is mediated by selective uptake. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2008, 1782, 523-531.	3.8	96
52	Increased Aβ1-42 Production Sensitizes Neuroblastoma Cells for ER Stress Toxicity. Current Alzheimer Research, 2008, 5, 469-474.	1.4	36
53	AÎ² ₁₋₄₂ Induces Mild Endoplasmic Reticulum Stress in an Aggregation Stateâ€“Dependent Manner. Antioxidants and Redox Signaling, 2007, 9, 2245-2254.	5.4	82
54	Branched KLVFF Tetramers Strongly Potentiate Inhibition of Î²â€Amyloid Aggregation. ChemBioChem, 2007, 8, 1857-1864.	2.6	128

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55	Pontocerebellar hypoplasia type 2: a neuropathological update. Acta Neuropathologica, 2007, 114, 373-386.	7.7	65
56	Pin1 levels are downregulated during ER stress in human neuroblastoma cells. Neurogenetics, 2007, 8, 21-27.	1.4	3
57	The Involvement of AÎ² in the Neuroinflammatory Response. , 2007, , 52-82.		1
58	Ubiquitin proteasome system as a pharmacological target in neurodegeneration. Expert Review of Neurotherapeutics, 2006, 6, 1337-1347.	2.8	26
59	The unfolded protein response affects neuronal cell cycle protein expression: Implications for Alzheimer's disease pathogenesis. Experimental Gerontology, 2006, 41, 380-386.	2.8	51
60	Maximal COX-2 and ppRb expression in neurons occurs during early Braak stages prior to the maximal activation of astrocytes and microglia in Alzheimer's disease. Journal of Neuroinflammation, 2005, 2, 27.	7.2	44
61	Alternative splicing in the N-terminus of Alzheimer?s presenilin 1. Neurogenetics, 2004, 5, 223-227.	1.4	19
62	Rab6 membrane association is dependent of Presenilin 1 and cellular phosphorylation events. Molecular Brain Research, 2004, 122, 17-23.	2.3	45
63	Protein translocation across the endoplasmic reticulum membrane in cold-adapted organisms. Journal of Cell Science, 2003, 116, 2875-2883.	2.0	39
64	Coordination of N-Glycosylation and Protein Translocation across the Endoplasmic Reticulum Membrane by Sss1 Protein. Journal of Biological Chemistry, 2003, 278, 37998-38003.	3.4	38
65	Growth-condition-dependent regulation of insulin-like growth factor II mRNA stability. Biochemical Journal, 1996, 318, 195-201.	3.7	15
66	Site-specific cleavage of IGF-II mRNAs requires sequence elements from two distinct regions of the IGF-II gene. Nucleic Acids Research, 1992, 20, 5003-5009.	14.5	50