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List of Publications by Year
in descending order

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64 papers	8,491 citations	70961 41 h-index	123241 61 g-index
70 all docs	70 docs citations	70 times ranked	10087 citing authors

#	Article	IF	Citations
1	The <scp>NLRP3</scp> inflammasome modulates tau pathology and neurodegeneration in a tauopathy model. Glia, 2022, 70, 1117-1132.	2.5	22
2	Blood-based AÎ²42 increases in the earliest pre-pathological stage before decreasing with progressive amyloid pathology in preclinical models and human subjects: opening new avenues for prevention. Acta Neuropathologica, 2022, 144, 489-508.	3.9	6
3	CSF1R inhibition rescues tau pathology and neurodegeneration in an A/T/N model with combined AD pathologies, while preserving plaque associated microglia. Acta Neuropathologica Communications, 2021, 9, 108.	2.4	22
4	Aggregated Tau activates NLRP3â€“ASC inflammasome exacerbating exogenously seeded and non-exogenously seeded Tau pathology in vivo. Acta Neuropathologica, 2019, 137, 599-617.	3.9	259
5	Association of Amyloid and Tau With Cognition in Preclinical Alzheimer Disease. JAMA Neurology, 2019, 76, 915.	4.5	512
6	Dietary Sargassum fusiforme improves memory and reduces amyloid plaque load in an Alzheimerâ€™s disease mouse model. Scientific Reports, 2019, 9, 4908.	1.6	51
7	Tau Interacting Proteins: Gaining Insight into the Roles of Tau in Health and Disease. Advances in Experimental Medicine and Biology, 2019, 1184, 145-166.	0.8	11
8	Sex-regulated gene dosage effect of PPARÎ± on synaptic plasticity. Life Science Alliance, 2019, 2, e201800262.	1.3	16
9	Synaptogyrin-3 Mediates Presynaptic Dysfunction Induced by Tau. Neuron, 2018, 97, 823-835.e8.	3.8	151
10	Tau interactome mappingÂbased identification of Otub1 as Tau deubiquitinase involved in accumulation of pathological Tau forms in vitro and in vivo. Acta Neuropathologica, 2017, 133, 731-749.	3.9	74
11	Preclinical models of Alzheimerâ€™s disease for identification and preclinical validation of therapeutic targets: from fine-tuning strategies for validated targets to new venues for therapy. , 2017, , 115-156.		2
12	Tau association with synaptic vesicles causes presynaptic dysfunction. Nature Communications, 2017, 8, 15295.	5.8	289
13	Cortical cells reveal APP as a new player in the regulation of GABAergic neurotransmission. Scientific Reports, 2017, 7, 370.	1.6	31
14	What is the evidence that tau pathology spreads through prion-like propagation?. Acta Neuropathologica Communications, 2017, 5, 99.	2.4	272
15	Presenilin 2-Dependent Maintenance of Mitochondrial Oxidative Capacity and Morphology. Frontiers in Physiology, 2017, 8, 796.	1.3	40
16	Glycines from the APP GXXXG/GXXXA Transmembrane Motifs Promote Formation of Pathogenic AÎ² Oligomers in Cells. Frontiers in Aging Neuroscience, 2016, 8, 107.	1.7	28
17	Activation of phagocytic activity in astrocytes by reduced expression of the inflammasome component ASC and its implication in a mouse model of Alzheimer disease. Journal of Neuroinflammation, 2016, 13, 20.	3.1	73
18	APPâ€dependent glial cell lineâ€derived neurotrophic factor gene expression drives neuromuscular junction formation. FASEB Journal, 2016, 30, 1696-1711.	0.2	27

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19	Heterotypic seeding of Tau fibrillization by pre-aggregated Abeta provides potent seeds for prion-like seeding and propagation of Tau-pathology in vivo. Acta Neuropathologica, 2016, 131, 549-569.	3.9	129
20	Analysis by a highly sensitive split luciferase assay of the regions involved in APP dimerization and its impact on processing. FEBS Open Bio, 2015, 5, 763-773.	1.0	25
21	Presenilin Transmembrane Domain 8 Conserved AXXXAXXXG Motifs Are Required for the Activity of the Î³-Secretase Complex. Journal of Biological Chemistry, 2015, 290, 7169-7184.	1.6	11
22	Characterization of Pterocarpus erinaceus kino extract and its gamma-secretase inhibitory properties. Journal of Ethnopharmacology, 2015, 163, 192-202.	2.0	17
23	Templated misfolding of Tau by prion-like seeding along neuronal connections impairs neuronal network function and associated behavioral outcomes in Tau transgenic mice. Acta Neuropathologica, 2015, 129, 875-894.	3.9	122
24	Intracerebral injection of preformed synthetic tau fibrils initiates widespread tauopathy and neuronal loss in the brains of tau transgenic mice. Neurobiology of Disease, 2015, 73, 83-95.	2.1	168
25	Models of Î²-amyloid induced Tau-pathology: the long and â€œfoldedâ€•road to understand the mechanism. Molecular Neurodegeneration, 2014, 9, 51.	4.4	220
26	Tauopathy contributes to synaptic and cognitive deficits in a murine model for Alzheimer's disease. FASEB Journal, 2014, 28, 2620-2631.	0.2	37
27	P1-033: AMYLOID-INDUCED TAUOPATHY CONTRIBUTES TO SYNAPTIC AND COGNITIVE DEFICITS IN A TRANSGENIC MODEL FOR ALZHEIMER'S DISEASE. , 2014, 10, P315-P315.		0
28	Neurological characterization of mice deficient in GSK3Î± highlight pleiotropic physiological functions in cognition and pathological activity as Tau kinase. Molecular Brain, 2013, 6, 27.	1.3	32
29	Amyloid precursor protein controls cholesterol turnover needed for neuronal activity. EMBO Molecular Medicine, 2013, 5, 608-625.	3.3	88
30	GSK-3Î±/Î² kinases and amyloid production in vivo. Nature, 2011, 480, E4-E5.	13.7	67
31	The Capsaicin Receptor TRPV1 Is a Crucial Mediator of the Noxious Effects of Mustard Oil. Current Biology, 2011, 21, 316-321.	1.8	189
32	Neuropeptide pituitary adenylate cyclaseâ€activating polypeptide (PACAP) slows down Alzheimer's diseaseâ€like pathology in amyloid precursor proteinâ€transgenic mice. FASEB Journal, 2011, 25, 3208-3218.	0.2	115
33	Alzheimer's disease: Old problem, new views from transgenic and viral models. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2010, 1802, 808-818.	1.8	43
34	AAV-Tau Mediates Pyramidal Neurodegeneration by Cell-Cycle Re-Entry without Neurofibrillary Tangle Formation in Wild-Type Mice. PLoS ONE, 2009, 4, e7280.	1.1	71
35	Beta-site amyloid precursor protein-cleaving enzyme-1 (BACE1)-mediated changes of endogenous amyloid beta in wild-type and transgenic mice in vivo. Neuroscience Letters, 2008, 435, 186-189.	1.0	15
36	Neurodegeneration and Neuroinflammation in cdk5/p25-Inducible Mice. American Journal of Pathology, 2008, 172, 470-485.	1.9	54

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37	Amyloid Activates GSK-3Î² to Aggravate Neuronal Tauopathy in Bigenic Mice. American Journal of Pathology, 2008, 172, 786-798.	1.9	255
38	Loss of Î³-Secretase Function Impairs Endocytosis of Lipoprotein Particles and Membrane Cholesterol Homeostasis. Journal of Neuroscience, 2008, 28, 12097-12106.	1.7	62
39	Mutant Presenilin 1 Alters Synaptic Transmission in Cultured Hippocampal Neurons. Journal of Biological Chemistry, 2007, 282, 1119-1127.	1.6	34
40	Deletion of the transient receptor potential cation channel TRPV4 impairs murine bladder voiding. Journal of Clinical Investigation, 2007, 117, 3453-3462.	3.9	283
41	Neuronal or Glial Expression of Human Apolipoprotein E4 Affects Parenchymal and Vascular Amyloid Pathology Differentially in Different Brain Regions of Double- and Triple-Transgenic Mice. American Journal of Pathology, 2006, 168, 245-260.	1.9	35
42	Nonsteroidal anti-inflammatory drugs repress Â-secretase gene promoter activity by the activation of PPARÂ. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 443-448.	3.3	365
43	Transgenic Mouse Models for APP Processing and Alzheimerâ€™s Disease: Early and Late Defects. Sub-Cellular Biochemistry, 2005, 38, 45-63.	1.0	25
44	Acute treatment with the PPARÎ³ agonist pioglitazone and ibuprofen reduces glial inflammation and AÎ²1â€“42 levels in APPV717I transgenic mice. Brain, 2005, 128, 1442-1453.	3.7	522
45	Focal glial activation coincides with increased BACE1 activation and precedes amyloid plaque deposition in APP[V717I] transgenic mice. Journal of Neuroinflammation, 2005, 2, 22.	3.1	257
46	Therapeutic effects of PKC activators in Alzheimer's disease transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 11141-11146.	3.3	316
47	Î²-Site Amyloid Precursor Protein Cleaving Enzyme 1 Increases Amyloid Deposition in Brain Parenchyma but Reduces Cerebrovascular Amyloid Angiopathy in Aging BACE Ã— APP[V717I] Double-Transgenic Mice. American Journal of Pathology, 2004, 165, 1621-1631.	1.9	50
48	A disintegrin-metalloproteinase prevents amyloid plaque formation and hippocampal defects in an Alzheimer disease mouse model. Journal of Clinical Investigation, 2004, 113, 1456-1464.	3.9	532
49	Neuropathobiology in Transgenic Mice: The Case of Alzheimer's Disease. , 2003, 209, 333-362.		3
50	Reduction of Î²-amyloid plaques in brain of transgenic mouse model of Alzheimerâ€™s disease by EFRH-phage immunization. Vaccine, 2003, 21, 1060-1065.	1.7	82
51	Capacitive Calcium Entry Is Directly Attenuated by Mutant Presenilin-1, Independent of the Expression of the Amyloid Precursor Protein. Journal of Biological Chemistry, 2003, 278, 2484-2489.	1.6	100
52	Capacitative Calcium Entry Induces Hippocampal Long Term Potentiation in the Absence of Presenilin-1. Journal of Biological Chemistry, 2003, 278, 44393-44399.	1.6	29
53	Nonsteroidal Anti-Inflammatory Drugs and Peroxisome Proliferator-Activated Receptor-Î³ Agonists Modulate Immunostimulated Processing of Amyloid Precursor Protein through Regulation of Î²-Secretase. Journal of Neuroscience, 2003, 23, 9796-9804.	1.7	347
54	Reduction of amyloid load and cerebral damage in transgenic mouse model of Alzheimer's disease by treatment with a Î²â€sheet breaker peptide. FASEB Journal, 2002, 16, 860-862.	0.2	224

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55	Neuronal Deficiency of Presenilin 1 Inhibits Amyloid Plaque Formation and Corrects Hippocampal Long-Term Potentiation But Not a Cognitive Defect of Amyloid Precursor Protein [V717I] Transgenic Mice. Journal of Neuroscience, 2002, 22, 3445-3453.	1.7	229
56	Secretases as targets for the treatment of Alzheimer's disease: the prospects. Lancet Neurology, The, 2002, 1, 409-416.	4.9	97
57	Axonal Transport, Tau Protein, and Neurodegeneration in Alzheimer's Disease. NeuroMolecular Medicine, 2002, 2, 151-166.	1.8	117
58	Mutant Presenilins Disturb Neuronal Calcium Homeostasis in the Brain of Transgenic Mice, Decreasing the Threshold for Excitotoxicity and Facilitating Long-term Potentiation. Journal of Biological Chemistry, 2001, 276, 11539-11544.	1.6	118
59	Aging Increased Amyloid Peptide and Caused Amyloid Plaques in Brain of Old APP/V717I Transgenic Mice by a Different Mechanism than Mutant Presenilin1. Journal of Neuroscience, 2000, 20, 6452-6458.	1.7	107
60	Behavioral Disturbances without Amyloid Deposits in Mice Overexpressing Human Amyloid Precursor Protein with Flemish (A692G) or Dutch (E693Q) Mutation. Neurobiology of Disease, 2000, 7, 9-22.	2.1	100
61	Prominent Cerebral Amyloid Angiopathy in Transgenic Mice Overexpressing the London Mutant of Human APP in Neurons. American Journal of Pathology, 2000, 157, 1283-1298.	1.9	213
62	Early Phenotypic Changes in Transgenic Mice That Overexpress Different Mutants of Amyloid Precursor Protein in Brain. Journal of Biological Chemistry, 1999, 274, 6483-6492.	1.6	611
63	Transgenic mice expressing an Î±-secretion mutant of the amyloid precursor protein in the brain develop a progressive CNS disorder. Behavioural Brain Research, 1998, 95, 55-64.	1.2	21
64	Basolateral Secretion of Amyloid Precursor Protein in Madin-Darby Canine Kidney Cells Is Disturbed by Alterations of Intracellular pH and by Introducing a Mutation Associated with Familial Alzheimer's Disease. Journal of Biological Chemistry, 1995, 270, 4058-4065.	1.6	66