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List of Publications by Year
in descending order

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112	10,428	41323	32815
papers	citations	49 h-index	100 g-index

117	117	117	11208
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Reversal of Neuropathology and Motor Dysfunction in a Conditional Model of Huntington's Disease. Cell, 2000, 101, 57-66.	13.5	1,011
2	Role of Tau Protein in Both Physiological and Pathological Conditions. Physiological Reviews, 2004, 84, 361-384.	13.1	787
3	Decreased nuclear beta-catenin, tau hyperphosphorylation and neurodegeneration in GSK-3beta conditional transgenic mice. EMBO Journal, 2001, 20, 27-39.	3.5	783
4	Structural Insights and Biological Effects of Glycogen Synthase Kinase 3-specific Inhibitor AR-A014418. Journal of Biological Chemistry, 2003, 278, 45937-45945.	1.6	451
5	Spatial learning deficit in transgenic mice that conditionally over-express GSK-3Î² in the brain but do not form tau filaments. Journal of Neurochemistry, 2002, 83, 1529-1533.	2.1	323
6	Increased vulnerability to cocaine in mice lacking the serotonin-1B receptor. Nature, 1998, 393, 175-178.	13.7	309
7	Glycogen synthase kinase-3 inhibition is integral to long-term potentiation. European Journal of Neuroscience, 2007, 25, 81-86.	1.2	300
8	GSK3: A possible link between beta amyloid peptide and tau protein. Experimental Neurology, 2010, 223, 322-325.	2.0	240
9	GSK3 and Tau: Two Convergence Points in Alzheimer's Disease. Journal of Alzheimer's Disease, 2012, 33, S141-S144.	1.2	238
10	Full Reversal of Alzheimer's Disease-Like Phenotype in a Mouse Model with Conditional Overexpression of Glycogen Synthase Kinase-3. Journal of Neuroscience, 2006, 26, 5083-5090.	1.7	234
11	Neuronal Induction of the Immunoproteasome in Huntington's Disease. Journal of Neuroscience, 2003, 23, 11653-11661.	1.7	228
12	FTDP-17 Mutations in tau Transgenic Mice Provoke Lysosomal Abnormalities and Tau Filaments in Forebrain. Molecular and Cellular Neurosciences, 2001, 18, 702-714.	1.0	207
13	Altered P2X7â€receptor level and function in mouse models of Huntington's disease and therapeutic efficacy of antagonist administration. FASEB Journal, 2009, 23, 1893-1906.	0.2	206
14	Chronic lithium administration to FTDP-17 tau and GSK-3? overexpressing mice prevents tau hyperphosphorylation and neurofibrillary tangle formation, but pre-formed neurofibrillary tangles do not revert. Journal of Neurochemistry, 2006, 99, 1445-1455.	2.1	197
15	Huntington's disease is a four-repeat tauopathy with tau nuclear rods. Nature Medicine, 2014, 20, 881-885.	15.2	183
16	Altered Machinery of Protein Synthesis in Alzheimer's: From the Nucleolus to the Ribosome. Brain Pathology, 2016, 26, 593-605.	2.1	180
17	Loss of striatal type 1 cannabinoid receptors is a key pathogenic factor in Huntingtonâ€™s disease. Brain, 2011, 134, 119-136.	3.7	178
18	Tauopathies with parkinsonism: clinical spectrum, neuropathologic basis, biological markers, and treatment options. European Journal of Neurology, 2009, 16, 297-309.	1.7	170

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19	Proteasomal-Dependent Aggregate Reversal and Absence of Cell Death in a Conditional Mouse Model of Huntington's Disease. Journal of Neuroscience, 2001, 21, 8772-8781.	1.7	153
20	Absence of Fenfluramine-Induced Anorexia and Reduced c-fos Induction in the Hypothalamus and Central Amygdaloid Complex of Serotonin 1B Receptor Knock-Out Mice. Journal of Neuroscience, 1998, 18, 5537-5544.	1.7	149
21	Glycogen Synthase Kinase-3 Plays a Crucial Role in Tau Exon 10 Splicing and Intranuclear Distribution of SC35. Journal of Biological Chemistry, 2004, 279, 3801-3806.	1.6	122
22	Reduced expression of the TrkB receptor in Huntington's disease mouse models and in human brain. European Journal of Neuroscience, 2006, 23, 649-658.	1.2	121
23	N-terminal Cleavage of GSK-3 by Calpain. Journal of Biological Chemistry, 2007, 282, 22406-22413.	1.6	120
24	Autism-like phenotype and risk gene mRNA deadenylation by CPEB4 mis-splicing. Nature, 2018, 560, 441-446.	13.7	113
25	New players in the 5-HT receptor field: genes and knockouts. Trends in Pharmacological Sciences, 1995, 16, 246-252.	4.0	108
26	Loss of mRNA levels, binding and activation of GTP-binding proteins for cannabinoid CB1 receptors in the basal ganglia of a transgenic model of Huntingtonâ€™s disease. Brain Research, 2002, 929, 236-242.	1.1	107
27	Cooexpression of FTDP-17 tau and GSK-3Î² in transgenic mice induce tau polymerization and neurodegeneration. Neurobiology of Aging, 2006, 27, 1258-1268.	1.5	105
28	Tau-knockout mice show reduced GSK3-induced hippocampal degeneration and learning deficits. Neurobiology of Disease, 2010, 37, 622-629.	2.1	100
29	CHOP regulates the p53â€“MDM2 axis and is required for neuronal survival after seizures. Brain, 2013, 136, 577-592.	3.7	95
30	In vivo inhibition of the mitochondrial H+-ATP synthase in neurons promotes metabolic preconditioning. EMBO Journal, 2014, 33, 762-778.	3.5	93
31	Molecular mechanisms of pain: Serotonin1A receptor agonists trigger transactivation by c-fos of the prodynorphin gene in spinal cord neurons. Neuron, 1993, 10, 599-611.	3.8	92
32	Modulation of the effects of cocaine by 5-HT1B receptors: a comparison of knockouts and antagonists. Pharmacology Biochemistry and Behavior, 2000, 67, 559-566.	1.3	92
33	5-Hydroxytryptamine_{1B} Receptors Modulate the Effect of Cocaine on c-fos Expression: Converging Evidence Using 5-Hydroxytryptamine_{1B} Knockout Mice and the 5-Hydroxytryptamine_{1B/1D} Antagonist GR127935. Molecular Pharmacology, 1997, 51, 755-763.	1.0	90
34	Inhibition of 26S proteasome activity by huntingtin filaments but not inclusion bodies isolated from mouse and human brain. Journal of Neurochemistry, 2006, 98, 1585-1596.	2.1	89
35	Proteasomal Expression, Induction of Immunoproteasome Subunits, and Local MHC Class I Presentation in Myofibrillar Myopathy and Inclusion Body Myositis. Journal of Neuropathology and Experimental Neurology, 2004, 63, 484-498.	0.9	84
36	UbiquitinÃ¢â‚¬â€œproteasome system involvement in HuntingtonÃ¢â‚¬â„¢s disease. Frontiers in Molecular Neuroscience, 2014, 7, 77.	1.4	84

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37	Accumulation of ubiquitin conjugates in a polyglutamine disease model occurs without global ubiquitin/proteasome system impairment. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 13986-13991.	3.3	82
38	Acute Polyglutamine Expression in Inducible Mouse Model Unravels Ubiquitin/Proteasome System Impairment and Permanent Recovery Attributable to Aggregate Formation. Journal of Neuroscience, 2010, 30, 3675-3688.	1.7	82
39	Protein oxidation in Huntington disease affects energy production and vitamin B6 metabolism. Free Radical Biology and Medicine, 2010, 49, 612-621.	1.3	77
40	NFAT/Fas signaling mediates the neuronal apoptosis and motor side effects of GSK-3 inhibition in a mouse model of lithium therapy. Journal of Clinical Investigation, 2010, 120, 2432-2445.	3.9	75
41	Full Motor Recovery Despite Striatal Neuron Loss and Formation of Irreversible Amyloid-Like Inclusions in a Conditional Mouse Model of Huntington's Disease. Journal of Neuroscience, 2005, 25, 9773-9781.	1.7	73
42	GSK3Î² overexpression induces neuronal death and a depletion of the neurogenic niches in the dentate gyrus. Hippocampus, 2011, 21, 910-922.	0.9	71
43	Is the ubiquitin-proteasome system impaired in Huntingtonâ€™s disease?. Cellular and Molecular Life Sciences, 2007, 64, 2245-2257.	2.4	67
44	The role of GSK3 in Alzheimer disease. Brain Research Bulletin, 2009, 80, 248-250.	1.4	64
45	GSK-3 mouse models to study neuronal apoptosis and neurodegeneration. Frontiers in Molecular Neuroscience, 2011, 4, 45.	1.4	64
46	Striatal-Enriched Protein Tyrosine Phosphatase Expression and Activity in Huntington's Disease: A STEP in the Resistance to Excitotoxicity. Journal of Neuroscience, 2011, 31, 8150-8162.	1.7	63
47	Neuronal apoptosis and reversible motor deficit in dominant-negative GSK-3 conditional transgenic mice. EMBO Journal, 2007, 26, 2743-2754.	3.5	59
48	Co-induction of jun B and c-fos in a subset of neurons in the spinal cord. Oncogene, 1991, 6, 223-7.	2.6	57
49	Biochemical, Ultrastructural, and Reversibility Studies on Huntingtin Filaments Isolated from Mouse and Human Brain. Journal of Neuroscience, 2004, 24, 9361-9371.	1.7	52
50	Prion-mediated neurodegeneration is associated with early impairment of the ubiquitinâ€“proteasome system. Acta Neuropathologica, 2016, 131, 411-425.	3.9	51
51	The Ubiquitin-Proteasome System in Huntingtonâ€™s Disease. Neuroscientist, 2005, 11, 583-594.	2.6	50
52	PH domain leucine-rich repeat protein phosphatase 1 contributes to maintain the activation of the PI3K/Akt pro-survival pathway in Huntington's disease striatum. Cell Death and Differentiation, 2010, 17, 324-335.	5.0	49
53	Protective neuronal induction of ATF5 in endoplasmic reticulum stress induced by status epilepticus. Brain, 2013, 136, 1161-1176.	3.7	49
54	Tau in neurodegenerative diseases: Tau phosphorylation and assembly. Neurotoxicity Research, 2004, 6, 477-482.	1.3	47

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55	Lithium, a Potential Protective Drug in Alzheimerâ€™s Disease. Neurodegenerative Diseases, 2008, 5, 247-249.	0.8	44
56	Impaired ATF6Îª processing, decreased Rheb and neuronal cell cycle re-entry in Huntington's disease. Neurobiology of Disease, 2011, 41, 23-32.	2.1	43
57	Enhaced induction of the immunoproteasome by interferon gamma in neurons expressing mutant huntingtin. Neurotoxicity Research, 2004, 6, 463-468.	1.3	41
58	GSK-3 dependent phosphoepitopes recognized by PHF-1 and AT-8 antibodies are present in different tau isoforms. Neurobiology of Aging, 2003, 24, 1087-1094.	1.5	40
59	Increased Neurotransmitter Release at the Neuromuscular Junction in a Mouse Model of Polyglutamine Disease. Journal of Neuroscience, 2011, 31, 1106-1113.	1.7	39
60	Testing the ubiquitinâ€“proteasome hypothesis of neurodegeneration in vivo. Trends in Neurosciences, 2004, 27, 66-69.	4.2	36
61	Reduced calcineurin protein levels and activity in exon-1 mouse models of Huntington's disease: Role in excitotoxicity. Neurobiology of Disease, 2009, 36, 461-469.	2.1	36
62	Impaired development of neocortical circuits contributes to the neurological alterations in DYRK1A haploinsufficiency syndrome. Neurobiology of Disease, 2019, 127, 210-222.	2.1	35
63	Îª-Synuclein accumulates in huntingtin inclusions but forms independent filaments and its deficiency attenuates early phenotype in a mouse model of Huntington's disease. Human Molecular Genetics, 2012, 21, 495-510.	1.4	34
64	Decreased glycogen synthase kinase-3 levels and activity contribute to Huntington's disease. Human Molecular Genetics, 2015, 24, 5040-5052.	1.4	33
65	Bi-directional genetic modulation of GSK-3Î² exacerbates hippocampal neuropathology in experimental status epilepticus. Cell Death and Disease, 2018, 9, 969.	2.7	32
66	Huntingtonâ€™s disease-specific mis-splicing unveils key effector genes and altered splicing factors. Brain, 2021, 144, 2009-2023.	3.7	32
67	BH3-only proteins Bid and BimEL are differentially involved in neuronal dysfunction in mouse models of Huntington's disease. Journal of Neuroscience Research, 2007, 85, 2756-2769.	1.3	30
68	Faulty splicing and cytoskeleton abnormalities in <scp>H</scp>untington's disease. Brain Pathology, 2016, 26, 772-778.	2.1	30
69	Effects of partial suppression of parkin on huntingtin mutant R6/1 mice. Brain Research, 2009, 1281, 91-100.	1.1	28
70	Assembly In Vitro of Tau Protein and its Implications in Alzheimers Disease. Current Alzheimer Research, 2004, 1, 97-101.	0.7	27
71	Mice with a naturally occurring DISC1 mutation display a broad spectrum of behaviors associated to psychiatric disorders. Frontiers in Behavioral Neuroscience, 2014, 8, 253.	1.0	27
72	Altered Levels and Isoforms of Tau and Nuclear Membrane Invaginations in Huntingtonâ€™s Disease. Frontiers in Cellular Neuroscience, 2019, 13, 574.	1.8	27

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73	Nuclear localization of N-terminal mutant huntingtin is cell cycle dependent. European Journal of Neuroscience, 2002, 16, 355-359.	1.2	26
74	Presynaptic dysfunction in Huntington's disease. Biochemical Society Transactions, 2010, 38, 488-492.	1.6	26
75	MAP2 Splicing is Altered in Huntington's Disease. Brain Pathology, 2017, 27, 181-189.	2.1	26
76	P2X7 Receptor Upregulation in Huntingtonâ€™s Disease Brains. Frontiers in Molecular Neuroscience, 2020, 13, 567430.	1.4	25
77	Tau phosphorylation in hippocampus results in toxic gain-of-function. Biochemical Society Transactions, 2010, 38, 977-980.	1.6	24
78	Spatiotemporal progression of ubiquitin-proteasome system inhibition after status epilepticus suggests protective adaptation against hippocampal injury. Molecular Neurodegeneration, 2017, 12, 21.	4.4	23
79	Age-dependent decline of motor neocortex but not hippocampal performance in heterozygous BDNF mice correlates with a decrease of cortical PSD-95 but an increase of hippocampal TrkB levels. Experimental Neurology, 2012, 237, 335-345.	2.0	22
80	Î±-synuclein levels affect autophagosome numbers in vivo and modulate Huntington disease pathology. Autophagy, 2012, 8, 431-432.	4.3	22
81	Sulfo-glycosaminoglycan content affects PHF-tau solubility and allows the identification of different types of PHFs. Brain Research, 2002, 935, 65-72.	1.1	21
82	Overexpression of synphilin-1 promotes clearance of soluble and misfolded alpha-synuclein without restoring the motor phenotype in aged A30P transgenic mice. Human Molecular Genetics, 2014, 23, 767-781.	1.4	20
83	Huntingtin-mediated axonal transport requires arginine methylation by PRMT6. Cell Reports, 2021, 35, 108980.	2.9	20
84	A new non-aggregative splicing isoform of human Tau is decreased in Alzheimerâ€™s disease. Acta Neuropathologica, 2021, 142, 159-177.	3.9	20
85	Tauâ€positive nuclear indentations in P301S tauopathy mice. Brain Pathology, 2017, 27, 314-322.	2.1	17
86	High concordance between hippocampal transcriptome of the mouse intraâ€amygdala kainic acid model and human temporal lobe epilepsy. Epilepsia, 2020, 61, 2795-2810.	2.6	17
87	Pathogenic SREK1 decrease in Huntingtonâ€™s disease lowers TAF1 mimicking X-linked dystonia parkinsonism. Brain, 2020, 143, 2207-2219.	3.7	17
88	Looking for novel functions of tau. Biochemical Society Transactions, 2012, 40, 653-655.	1.6	16
89	Mutant huntingtin affects endocytosis in striatal cells by altering the binding of AP-2 to membranes. Experimental Neurology, 2013, 241, 75-83.	2.0	16
90	The neuroprotective transcription factor ATF5 is decreased and sequestered into polyglutamine inclusions in Huntingtonâ€™s disease. Acta Neuropathologica, 2017, 134, 839-850.	3.9	16

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91	CPEB alteration and aberrant transcriptome-polyadenylation lead to a treatable SLC19A3 deficiency in Huntingtonâ€™s disease. Science Translational Medicine, 2021, 13, eabe7104.	5.8	14
92	Reduced striatal dopamine DA D2 receptor function in dominant-negative GSK-3 transgenic mice. European Neuropsychopharmacology, 2014, 24, 1524-1533.	0.3	13
93	Peripheral Noxious Stimulation Induces CREM Expression in Dorsal Horn: Involvement of Glutamate. European Journal of Neuroscience, 1997, 9, 2778-2783.	1.2	12
94	Nuclear localization of Î²-catenin in adult mouse thalamus correlates with low levels of GSK-3Î². NeuroReport, 1999, 10, 2699-2703.	0.6	12
95	Polyadenylation of mRNA as a novel regulatory mechanism of gene expression in temporal lobe epilepsy. Brain, 2020, 143, 2139-2153.	3.7	11
96	Prion-Associated Neurodegeneration Causes Both Endoplasmic Reticulum Stress and Proteasome Impairment in a Murine Model of Spontaneous Disease. International Journal of Molecular Sciences, 2021, 22, 465.	1.8	11
97	Mice Lacking Functional Fas Death Receptors Are Protected from Kainic Acid-Induced Apoptosis in the Hippocampus. Molecular Neurobiology, 2015, 52, 120-129.	1.9	9
98	The regulation of proteostasis in glial cells by nucleotide receptors is key in acute neuroinflammation. FASEB Journal, 2018, 32, 3020-3032.	0.2	9
99	Differential regulation of Kidins220 isoforms in Huntington's disease. Brain Pathology, 2020, 30, 120-136.	2.1	9
100	CK2 alpha prime and alpha-synuclein pathogenic functional interaction mediates synaptic dysregulation in Huntingtonâ€™s disease. Acta Neuropathologica Communications, 2022, 10, .	2.4	9
101	Hippocampal neuronal subpopulations are differentially affected in double transgenic mice overexpressing frontotemporal dementia and parkinsonism linked to chromosome 17 tau and glycogen synthase kinase-3Î². Neuroscience, 2008, 157, 772-780.	1.1	8
102	A mouse model to study tau pathology related with tau phosphorylation and assembly. Journal of the Neurological Sciences, 2007, 257, 250-254.	0.3	7
103	Impaired PLP-dependent metabolism in brain samples from Huntington disease patients and transgenic R6/1 mice. Metabolic Brain Disease, 2016, 31, 579-586.	1.4	7
104	Testing the possible inhibition of proteasome by direct interaction with ubiquitylated and aggregated huntingtin. Brain Research Bulletin, 2007, 72, 121-123.	1.4	6
105	Tau Kinase I Overexpression Induces Dentate Gyrus Degeneration. Neurodegenerative Diseases, 2010, 7, 13-15.	0.8	5
106	Neuronal Apoptosis and Motor Deficits in Mice with Genetic Inhibition of GSK-3 Are Fas-Dependent. PLoS ONE, 2013, 8, e70952.	1.1	5
107	Regulation of proteasome activity by P2Y 2 receptor underlies the neuroprotective effects of extracellular nucleotides. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2017, 1863, 43-51.	1.8	5
108	Targeting the proteasome in epilepsy. Oncotarget, 2017, 8, 45042-45043.	0.8	3

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109	Profiling of Argonaute-2-loaded microRNAs in a mouse model of frontotemporal dementia with parkinsonism-17. International Journal of Physiology, Pathophysiology and Pharmacology, 2018, 10, 172-183.	0.8	2
110	Animal Models with Modified Expression of GSK-3 for the Study of Its Physiology and of Its Implications in Human Pathologies. , 0, , 203-219.		0
111	Centro de Biologia Molecular â€œSevero Ochoaâ€: A Center for Basic Research into Alzheimer's Disease. Journal of Alzheimer's Disease, 2010, 21, 325-335.	1.2	0
112	Co-expression of FTDP-17 Human Tau and GSK-3ÃŸ (or APPSW) in Transgenic Mice: Induction of Tau Polymerization and Neurodegeneration. , 2008, , 337-342.		0