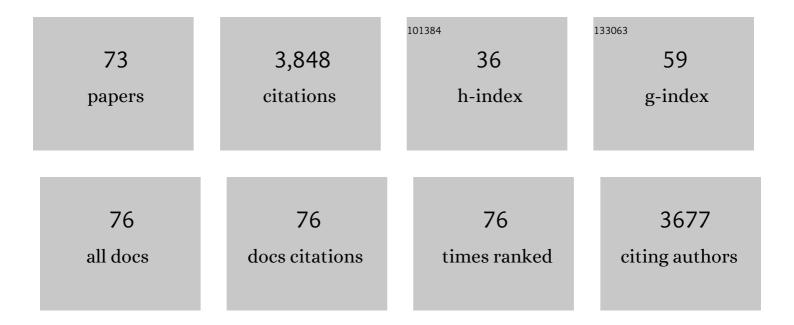
List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Treatment with a GSK-3β/HDAC Dual Inhibitor Restores Neuronal Survival and Maturation in an In Vitro and In Vivo Model of CDKL5 Deficiency Disorder. International Journal of Molecular Sciences, 2021, 22, 5950.	1.8	10
2	A GABAB receptor antagonist rescues functional and structural impairments in the perirhinal cortex of a mouse model of CDKL5 deficiency disorder. Neurobiology of Disease, 2021, 153, 105304.	2.1	9
3	Inhibition of microglia overactivation restores neuronal survival in a mouse model of CDKL5 deficiency disorder. Journal of Neuroinflammation, 2021, 18, 155.	3.1	21
4	Pharmacotherapy with sertraline rescues brain development and behavior in a mouse model of CDKL5 deficiency disorder. Neuropharmacology, 2020, 167, 107746.	2.0	12
5	Increased DNA Damage and Apoptosis in CDKL5-Deficient Neurons. Molecular Neurobiology, 2020, 57, 2244-2262.	1.9	15
6	Functional and Structural Impairments in the Perirhinal Cortex of a Mouse Model of CDKL5 Deficiency Disorder Are Rescued by a TrkB Agonist. Frontiers in Cellular Neuroscience, 2019, 13, 169.	1.8	35
7	Site-specific abnormalities in the visual system of a mouse model of CDKL5 deficiency disorder. Human Molecular Genetics, 2019, 28, 2851-2861.	1.4	30
8	CDKL5 deficiency predisposes neurons to cell death through the deregulation of SMAD3 signaling. Brain Pathology, 2019, 29, 658-674.	2.1	17
9	CDKL5 protein substitution therapy rescues neurological phenotypes of a mouse model of CDKL5 disorder. Human Molecular Genetics, 2018, 27, 1572-1592.	1.4	49
10	Treatment with the <scp>GSK</scp> 3â€beta inhibitor Tideglusib improves hippocampal development and memory performance in juvenile, but not adult, <i>Cdkl5</i> knockout mice. European Journal of Neuroscience, 2018, 47, 1054-1066.	1.2	33
11	Heterozygous CDKL5 Knockout Female Mice Are a Valuable Animal Model for CDKL5 Disorder. Neural Plasticity, 2018, 2018, 1-18.	1.0	39
12	Epigallocatechin gallate: A useful therapy for cognitive disability in Down syndrome?. Neurogenesis (Austin, Tex), 2017, 4, e1270383.	1.5	13
13	Long-term effect of neonatal inhibition of APP gamma-secretase on hippocampal development in the Ts65Dn mouse model of Down syndrome. Neurobiology of Disease, 2017, 103, 11-23.	2.1	14
14	<scp>CDKL</scp> 5 deficiency entails sleep apneas in mice. Journal of Sleep Research, 2017, 26, 495-497.	1.7	32
15	Lithium Restores Age-related Olfactory Impairment in the Ts65Dn Mouse Model of Down Syndrome. CNS and Neurological Disorders - Drug Targets, 2017, 16, 812-819.	0.8	10
16	HDAC4: a key factor underlying brain developmental alterations in CDKL5 disorder. Human Molecular Genetics, 2016, 25, 3887-3907.	1.4	77
17	Short- and long-term effects of neonatal pharmacotherapy with epigallocatechin-3-gallate on hippocampal development in the Ts65Dn mouse model of Down syndrome. Neuroscience, 2016, 333, 277-301.	1.1	60
18	Timing of therapies for Down syndrome: the sooner, the better. Frontiers in Behavioral Neuroscience, 2015, 9, 265.	1.0	94

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19	Inhibition of GSK3Î ² rescues hippocampal development and learning in a mouse model of CDKL5 disorder. Neurobiology of Disease, 2015, 82, 298-310.	2.1	55
20	Inhibition of APP gamma-secretase restores Sonic Hedgehog signaling and neurogenesis in the Ts65Dn mouse model of Down syndrome. Neurobiology of Disease, 2015, 82, 385-396.	2.1	37
21	Long-term effects of neonatal treatment with fluoxetine on cognitive performance in Ts65Dn mice. Neurobiology of Disease, 2015, 74, 204-218.	2.1	44
22	Mapping Pathological Phenotypes in a Mouse Model of CDKL5 Disorder. PLoS ONE, 2014, 9, e91613.	1.1	145
23	Prenatal pharmacotherapy rescues brain development in a Down's syndrome mouse model. Brain, 2014, 137, 380-401.	3.7	71
24	Loss of CDKL5 impairs survival and dendritic growth of newborn neurons by altering AKT/GSK-3β signaling. Neurobiology of Disease, 2014, 70, 53-68.	2.1	105
25	APP-dependent alteration of GSK3β activity impairs neurogenesis in the Ts65Dn mouse model of Down syndrome. Neurobiology of Disease, 2014, 67, 24-36.	2.1	33
26	Age-related impairment of olfactory bulb neurogenesis in the Ts65Dn mouse model of Down syndrome. Experimental Neurology, 2014, 251, 1-11.	2.0	18
27	The Amyloid Precursor Protein (APP) Triplicated Gene Impairs Neuronal Precursor Differentiation and Neurite Development through Two Different Domains in the Ts65Dn Mouse Model for Down Syndrome. Journal of Biological Chemistry, 2013, 288, 20817-20829.	1.6	46
28	Early Pharmacotherapy with Fluoxetine Rescues Dendritic Pathology in the <scp>Ts65Dn</scp> Mouse Model of <scp>D</scp> own Syndrome. Brain Pathology, 2013, 23, 129-143.	2.1	61
29	Pharmacotherapy with Fluoxetine Restores Functional Connectivity from the Dentate Gyrus to Field CA3 in the Ts65Dn Mouse Model of Down Syndrome. PLoS ONE, 2013, 8, e61689.	1.1	42
30	Early-occurring proliferation defects in peripheral tissues of the Ts65Dn mouse model of Down syndrome are associated with patched1 over expression. Laboratory Investigation, 2012, 92, 1648-1660.	1.7	21
31	CDKL5, a novel MYCN-repressed gene, blocks cell cycle and promotes differentiation of neuronal cells. Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms, 2012, 1819, 1173-1185.	0.9	31
32	Widespread Proliferation Impairment and Hypocellularity in the Cerebellum of Fetuses with Down Syndrome. Brain Pathology, 2011, 21, 361-373.	2.1	137
33	Impact of environmental enrichment on neurogenesis in the dentate gyrus during the early postnatal period. Brain Research, 2011, 1415, 23-33.	1.1	30
34	ls it possible to improve neurodevelopmental abnormalities in Down syndrome?. Reviews in the Neurosciences, 2011, 22, 419-455.	1.4	66
35	APP-dependent up-regulation of Ptch1 underlies proliferation impairment of neural precursors in Down syndrome. Human Molecular Genetics, 2011, 20, 1560-1573.	1.4	106
36	Lithium Restores Neurogenesis in the Subventricular Zone of the Ts65Dn Mouse, a Model for Down Syndrome. Brain Pathology, 2010, 20, 106-118.	2.1	75

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37	Early Pharmacotherapy Restores Neurogenesis and Cognitive Performance in the Ts65Dn Mouse Model for Down Syndrome. Journal of Neuroscience, 2010, 30, 8769-8779.	1.7	164
38	CB1 Cannabinoid Receptors Increase Neuronal Precursor Proliferation through AKT/Glycogen Synthase Kinase-3β/β-Catenin Signaling. Journal of Biological Chemistry, 2010, 285, 10098-10109.	1.6	73
39	Lot1 Is a Key Element of the Pituitary Adenylate Cyclase-activating Polypeptide (PACAP)/Cyclic AMP Pathway That Negatively Regulates Neuronal Precursor Proliferation. Journal of Biological Chemistry, 2009, 284, 15325-15338.	1.6	18
40	Cell Cycle Elongation Impairs Proliferation of Cerebellar Granule Cell Precursors in the Ts65Dn Mouse, an Animal Model for Down Syndrome. Brain Pathology, 2009, 19, 224-237.	2.1	60
41	RESEARCH ARTICLE: Neurogenesis Impairment and Increased Cell Death Reduce Total Neuron Number in the Hippocampal Region of Fetuses with Down Syndrome. Brain Pathology, 2008, 18, 180-197.	2.1	230
42	The Place of Choline Acetyltransferase Activity Measurement in the "Cholinergic Hypothesis―of Neurodegenerative Diseases. Neurochemical Research, 2008, 33, 318-327.	1.6	56
43	Neonatal isolation impairs neurogenesis in thedentate gyrus of the guinea pig. Hippocampus, 2007, 17, 78-91.	0.9	23
44	Cell cycle alteration and decreased cell proliferation in the hippocampal dentate gyrus and in the neocortical germinal matrix of fetuses with down syndrome and in Ts65Dn mice. Hippocampus, 2007, 17, 665-678.	0.9	234
45	Choline acetyltransferase activity at different ages in brain of Ts65Dn mice, an animal model for Down's syndrome and related neurodegenerative diseases. Journal of Neurochemistry, 2006, 97, 515-526.	2.1	63
46	Proliferation of cerebellar precursor cells is negatively regulated by nitric oxide in newborn rat. Journal of Cell Science, 2006, 119, 3161-3170.	1.2	35
47	Postnatal neurogenesis in the dentate gyrus of the guinea pig. Hippocampus, 2005, 15, 285-301.	0.9	52
48	Cyclic AMP-mediated Regulation of Transcription Factor Lot1 Expression in Cerebellar Granule Cells. Journal of Biological Chemistry, 2005, 280, 33541-33551.	1.6	17
49	Neurochemical Correlates of Nicotine Neurotoxicity on Rat Habenulo-Interpeduncular Cholinergic Neurons. NeuroToxicology, 2005, 26, 467-474.	1.4	22
50	Nitric oxide negatively regulates proliferation and promotes neuronal differentiation through N-Myc downregulation. Journal of Cell Science, 2004, 117, 4727-4737.	1.2	69
51	Dietary restriction differentially protects from neurodegeneration in animal models of excitotoxicity. Brain Research, 2004, 1002, 162-166.	1.1	47
52	Nitric oxide regulates cGMP-dependent cAMP-responsive element binding protein phosphorylation and Bcl-2 expression in cerebellar neurons: implication for a survival role of nitric oxide. Journal of Neurochemistry, 2004, 82, 1282-1289.	2.1	128
53	Role of nitric oxide in the regulation of neuronal proliferation, survival and differentiation. Neurochemistry International, 2004, 45, 903-914.	1.9	149
54	Developmental expression of the cell cycle and apoptosis controlling gene, Lot1, in the rat cerebellum and in cultures of cerebellar granule cells. Developmental Brain Research, 2003, 142, 193-202.	2.1	21

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55	Transcriptional Activities of the Zinc Finger Protein Zac Are Differentially Controlled by DNA Binding. Molecular and Cellular Biology, 2003, 23, 988-1003.	1.1	65
56	Brain Nitric Oxide and Its Dual Role in Neurodegeneration / Neuroprotection: Understanding Molecular Mechanisms to Devise Drug Approaches. Current Medicinal Chemistry, 2003, 10, 2147-2174.	1.2	79
57	Nitric Oxide Protects Neuroblastoma Cells from Apoptosis Induced by Serum Deprivation through cAMP-response Element-binding Protein (CREB) Activation. Journal of Biological Chemistry, 2002, 277, 49896-49902.	1.6	76
58	Akt pathway mediates a cGMP-dependent survival role of nitric oxide in cerebellar granule neurones. Journal of Neurochemistry, 2002, 81, 218-228.	2.1	81
59	Sustained, long-lasting inhibition of nitric oxide synthase aggravates the neural damage in some models of excitotoxic brain injury. Brain Research Bulletin, 2001, 56, 29-35.	1.4	14
60	Inhibition of Zac1, a New Gene Differentially Expressed in the Anterior Pituitary, Increases Cell Proliferation*. Endocrinology, 1999, 140, 987-996.	1.4	47
61	Induction of the PAC1-R (PACAP-type I receptor) gene by p53 and Zac. Molecular Brain Research, 1999, 69, 290-294.	2.5	42
62	Induction of Type I PACAP Receptor Expression by the New Zinc Finger Protein Zac1 and p53. Annals of the New York Academy of Sciences, 1998, 865, 49-58.	1.8	24
63	Activation of the ornithine decarboxylase-polyamine system and induction of c- fos and p53 expression in relation to excitotoxic neuronal apoptosis in normal and microencephalic rats. Experimental Brain Research, 1998, 120, 519-526.	0.7	8
64	Neurotoxicity of Polyamines and Pharmacological Neuroprotection in Cultures of Rat Cerebellar Granule Cells. Experimental Neurology, 1997, 148, 157-166.	2.0	49
65	Chronic pre-explant blockade of the NMDA receptor affects survival of cerebellar granule cells explanted in vitro. Developmental Brain Research, 1997, 99, 112-117.	2.1	20
66	Toxicity of ricin and volkensin, two ribosome-inactivating proteins, to microglia, astrocyte, and neuron cultures. , 1997, 20, 203-209.		18
67	Absence of excitotoxic neuropathology in microencephalic rats after systemic kainic acid administration. Neuroscience Letters, 1996, 218, 57-61.	1.0	3
68	Inhibition of free radical production or free radical scavenging protects from the excitotoxic cell death mediated by glutamate in cultures of cerebellar granule neurons. Brain Research, 1996, 728, 1-6.	1.1	115
69	Activation of a reporter gene responsive to NGFI-B in cultured neurons and astrocytes. Journal of Molecular Neuroscience, 1995, 6, 131-139.	1.1	15
70	Fos protein induction, neuropathology, and pharmacological protection after excitotoxic brain insult. Experimental Brain Research, 1994, 98, 421-30.	0.7	7
71	Immunohistochemical localization of calbindin-D28K in telencephalic regions of microencephalic rats. Neuroscience Letters, 1994, 171, 41-44.	1.0	7
72	An endogenous ligand for the kainate-type binding sites from rat brain. Comparative Biochemistry and Physiology C, Comparative Pharmacology and Toxicology, 1994, 108, 205-214.	0.5	0

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73	Decreased excitotoxic sensitivity in the olfactory cortex of adult rats after neonatal NMDA blockade. NeuroReport, 1994, 5, 2141-2144.	0.6	4