Elisabetta Ciani

List of Publications by Year
in descending order

73 papers	3,848 citations	101384 36 h-index	133063 59 g-index
76 all docs	76 docs citations	76 times ranked	3677 citing authors

#	Article	IF	Citations
1	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
2	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
3	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
4	Role of nitric oxide in the regulation of neuronal proliferation, survival and differentiation. Neurochemistry International, 2004, 45, 903-914.	1.9	149
5	Mapping Pathological Phenotypes in a Mouse Model of CDKL5 Disorder. PLoS ONE, 2014, 9, e91613.	1.1	145
6	Widespread Proliferation Impairment and Hypocellularity in the Cerebellum of Fetuses with Down Syndrome. Brain Pathology, 2011, 21, 361-373.	2.1	137
7	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
8	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
9	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
10	Loss of CDKL5 impairs survival and dendritic growth of newborn neurons by altering AKT/GSK-3Î² signaling. Neurobiology of Disease, 2014, 70, 53-68.	2.1	105
11	Timing of therapies for Down syndrome: the sooner, the better. Frontiers in Behavioral Neuroscience, 2015, 9, 265.	1.0	94
12	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
13	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
14	HDAC4: a key factor underlying brain developmental alterations in CDKL5 disorder. Human Molecular Genetics, 2016, 25, 3887-3907.	1.4	77
15	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
16	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
17	CB1 Cannabinoid Receptors Increase Neuronal Precursor Proliferation through AKT/Glycogen Synthase Kinase-3Î²/Î²-Catenin Signaling. Journal of Biological Chemistry, 2010, 285, 10098-10109.	1.6	73
18	Prenatal pharmacotherapy rescues brain development in a Downâ€™s syndrome mouse model. Brain, 2014, 137, 380-401.	3.7	71

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19	Nitric oxide negatively regulates proliferation and promotes neuronal differentiation through N-Myc downregulation. Journal of Cell Science, 2004, 117, 4727-4737.	1.2	69
20	Is it possible to improve neurodevelopmental abnormalities in Down syndrome?. Reviews in the Neurosciences, 2011, 22, 419-455.	1.4	66
21	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
22	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
23	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
24	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
25	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
26	The Place of Choline Acetyltransferase Activity Measurement in the â€œCholinergic Hypothesisâ€•of Neurodegenerative Diseases. Neurochemical Research, 2008, 33, 318-327.	1.6	56
27	Inhibition of GSK3Î² rescues hippocampal development and learning in a mouse model of CDKL5 disorder. Neurobiology of Disease, 2015, 82, 298-310.	2.1	55
28	Postnatal neurogenesis in the dentate gyrus of the guinea pig. Hippocampus, 2005, 15, 285-301.	0.9	52
29	Neurotoxicity of Polyamines and Pharmacological Neuroprotection in Cultures of Rat Cerebellar Granule Cells. Experimental Neurology, 1997, 148, 157-166.	2.0	49
30	CDKL5 protein substitution therapy rescues neurological phenotypes of a mouse model of CDKL5 disorder. Human Molecular Genetics, 2018, 27, 1572-1592.	1.4	49
31	Inhibition of Zac1, a New Gene Differentially Expressed in the Anterior Pituitary, Increases Cell Proliferation*. Endocrinology, 1999, 140, 987-996.	1.4	47
32	Dietary restriction differentially protects from neurodegeneration in animal models of excitotoxicity. Brain Research, 2004, 1002, 162-166.	1.1	47
33	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
34	Long-term effects of neonatal treatment with fluoxetine on cognitive performance in Ts65Dn mice. Neurobiology of Disease, 2015, 74, 204-218.	2.1	44
35	Induction of the PAC1-R (PACAP-type I receptor) gene by p53 and Zac. Molecular Brain Research, 1999, 69, 290-294.	2.5	42
36	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

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37	Heterozygous CDKL5 Knockout Female Mice Are a Valuable Animal Model for CDKL5 Disorder. Neural Plasticity, 2018, 2018, 1-18.	1.0	39
38	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
39	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
40	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
41	APP-dependent alteration of GSK3Î² activity impairs neurogenesis in the Ts65Dn mouse model of Down syndrome. Neurobiology of Disease, 2014, 67, 24-36.	2.1	33
42	Treatment with the <scp>GSK</scp>3â€beta inhibitor Tideglusib improves hippocampal development and memory performance in juvenile, but not adult, Cdkl5 knockout mice. European Journal of Neuroscience, 2018, 47, 1054-1066.	1.2	33
43	<scp>CDKL</scp>5 deficiency entails sleep apneas in mice. Journal of Sleep Research, 2017, 26, 495-497.	1.7	32
44	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
45	Impact of environmental enrichment on neurogenesis in the dentate gyrus during the early postnatal period. Brain Research, 2011, 1415, 23-33.	1.1	30
46	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
47	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
48	Neonatal isolation impairs neurogenesis in thedentate gyrus of the guinea pig. Hippocampus, 2007, 17, 78-91.	0.9	23
49	Neurochemical Correlates of Nicotine Neurotoxicity on Rat Habenulo-Interpeduncular Cholinergic Neurons. NeuroToxicology, 2005, 26, 467-474.	1.4	22
50	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
51	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
52	Inhibition of microglia overactivation restores neuronal survival in a mouse model of CDKL5 deficiency disorder. Journal of Neuroinflammation, 2021, 18, 155.	3.1	21
53	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
54	Toxicity of ricin and volkensin, two ribosome-inactivating proteins, to microglia, astrocyte, and neuron cultures. , 1997, 20, 203-209.		18

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55	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
56	Age-related impairment of olfactory bulb neurogenesis in the Ts65Dn mouse model of Down syndrome. Experimental Neurology, 2014, 251, 1-11.	2.0	18
57	Cyclic AMP-mediated Regulation of Transcription Factor Lot1 Expression in Cerebellar Granule Cells. Journal of Biological Chemistry, 2005, 280, 33541-33551.	1.6	17
58	CDKL5 deficiency predisposes neurons to cell death through the deregulation of SMAD3 signaling. Brain Pathology, 2019, 29, 658-674.	2.1	17
59	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
60	Increased DNA Damage and Apoptosis in CDKL5-Deficient Neurons. Molecular Neurobiology, 2020, 57, 2244-2262.	1.9	15
61	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
62	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
63	Epigallocatechin gallate: A useful therapy for cognitive disability in Down syndrome?. Neurogenesis (Austin, Tex), 2017, 4, e1270383.	1.5	13
64	Pharmacotherapy with sertraline rescues brain development and behavior in a mouse model of CDKL5 deficiency disorder. Neuropharmacology, 2020, 167, 107746.	2.0	12
65	Treatment with a GSK-3Î²/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
66	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
67	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
68	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
69	Fos protein induction, neuropathology, and pharmacological protection after excitotoxic brain insult. Experimental Brain Research, 1994, 98, 421-30.	0.7	7
70	Immunohistochemical localization of calbindin-D28K in telencephalic regions of microencephalic rats. Neuroscience Letters, 1994, 171, 41-44.	1.0	7
71	Decreased excitotoxic sensitivity in the olfactory cortex of adult rats after neonatal NMDA blockade. NeuroReport, 1994, 5, 2141-2144.	0.6	4
72	Absence of excitotoxic neuropathology in microencephalic rats after systemic kainic acid administration. Neuroscience Letters, 1996, 218, 57-61.	1.0	3

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73	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