Stefania Trazzi

List of Publications by Year
in descending order

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32	1,525	304743	434195
papers	citations	22 h-index	31 g-index

39	39	39	1742
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Early Pharmacotherapy Restores Neurogenesis and Cognitive Performance in the Ts65Dn Mouse Model for Down Syndrome. Journal of Neuroscience, 2010, 30, 8769-8779.	3.6	164
2	DNMT3B interacts with constitutive centromere protein CENP-C to modulate DNA methylation and the histone code at centromeric regions. Human Molecular Genetics, 2009, 18, 3178-3193.	2.9	132
3	APP-dependent up-regulation of Ptch1 underlies proliferation impairment of neural precursors in Down syndrome. Human Molecular Genetics, 2011, 20, 1560-1573.	2.9	106
4	Loss of CDKL5 impairs survival and dendritic growth of newborn neurons by altering AKT/GSK-3Î² signaling. Neurobiology of Disease, 2014, 70, 53-68.	4.4	105
5	HDAC4: a key factor underlying brain developmental alterations in CDKL5 disorder. Human Molecular Genetics, 2016, 25, 3887-3907.	2.9	77
6	CB1 Cannabinoid Receptors Increase Neuronal Precursor Proliferation through AKT/Glycogen Synthase Kinase-3Î²/Î²-Catenin Signaling. Journal of Biological Chemistry, 2010, 285, 10098-10109.	3.4	73
7	CENP-C binds the alpha-satellite DNA in vivo at specific centromere domains. Journal of Cell Science, 2002, 115, 2317-2327.	2.0	67
8	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.	4.1	61
9	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.	2.3	60
10	Inhibition of GSK3Î² rescues hippocampal development and learning in a mouse model of CDKL5 disorder. Neurobiology of Disease, 2015, 82, 298-310.	4.4	55
11	CENP-C binds the alpha-satellite DNA in vivo at specific centromere domains. Journal of Cell Science, 2002, 115, 2317-27.	2.0	54
12	The C-Terminal Domain of CENP-C Displays Multiple and Critical Functions for Mammalian Centromere Formation. PLoS ONE, 2009, 4, e5832.	2.5	50
13	CDKL5 protein substitution therapy rescues neurological phenotypes of a mouse model of CDKL5 disorder. Human Molecular Genetics, 2018, 27, 1572-1592.	2.9	49
14	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.	3.4	46
15	Heterozygous CDKL5 Knockout Female Mice Are a Valuable Animal Model for CDKL5 Disorder. Neural Plasticity, 2018, 2018, 1-18.	2.2	39
16	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.	4.4	37
17	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.	3.7	35
18	APP-dependent alteration of GSK3Î² activity impairs neurogenesis in the Ts65Dn mouse model of Down syndrome. Neurobiology of Disease, 2014, 67, 24-36.	4.4	33

#	Article	IF	Citations
19	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.	2.6	33
20	In vivo functional dissection of human inner kinetochore protein CENP-C. Journal of Structural Biology, 2002, 140, 39-48.	2.8	32
21	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.	1.9	31
22	Functional cooperation between TrkA and p75NTR accelerates neuronal differentiation by increased transcription of GAP-43 and p21(CIP/WAF) genes via ERK1/2 and AP-1 activities. Experimental Cell Research, 2007, 313, 2980-2992.	2.6	28
23	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.	3.7	21
24	Inhibition of microglia overactivation restores neuronal survival in a mouse model of CDKL5 deficiency disorder. Journal of Neuroinflammation, 2021, 18, 155.	7.2	21
25	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.	3.4	18
26	Age-related impairment of olfactory bulb neurogenesis in the Ts65Dn mouse model of Down syndrome. Experimental Neurology, 2014, 251, 1-11.	4.1	18
27	CDKL5 deficiency predisposes neurons to cell death through the deregulation of SMAD3 signaling. Brain Pathology, 2019, 29, 658-674.	4.1	17
28	Age-Related Cognitive and Motor Decline in a Mouse Model of CDKL5 Deficiency Disorder is Associated with Increased Neuronal Senescence and Death. , 2021, 12, 764.		16
29	Increased DNA Damage and Apoptosis in CDKL5-Deficient Neurons. Molecular Neurobiology, 2020, 57, 2244-2262.	4.0	15
30	Pharmacotherapy with sertraline rescues brain development and behavior in a mouse model of CDKL5 deficiency disorder. Neuropharmacology, 2020, 167, 107746.	4.1	12
31	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.	4.1	10
32	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.	4.4	9