Clara Penas

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/7446221/publications.pdf Version: 2024-02-01



CLADA DENIAS

#	Article	IF	CITATIONS
1	<scp>BET</scp> protein inhibition in macrophages enhances dorsal root ganglion neurite outgrowth in female mice. Journal of Neuroscience Research, 2022, 100, 1331-1346.	2.9	2
2	Beneficial and detrimental effects of cytokines after spinal cord injury. , 2022, , 105-117.		0
3	Nerve Excitability and Neuropathic Pain is Reduced by BET Protein Inhibition After Spared Nerve Injury. Journal of Pain, 2021, 22, 1617-1630.	1.4	8
4	Time series modeling of cell cycle exit identifies Brd4 dependent regulation of cerebellar neurogenesis. Nature Communications, 2019, 10, 3028.	12.8	33
5	BET protein inhibition regulates cytokine production and promotes neuroprotection after spinal cord injury. Journal of Neuroinflammation, 2019, 16, 124.	7.2	45
6	Epigenetic Modifications Associated to Neuroinflammation and Neuropathic Pain After Neural Trauma. Frontiers in Cellular Neuroscience, 2018, 12, 158.	3.7	90
7	Serum long noncoding RNA HOTAIR as a novel diagnostic and prognostic biomarker in glioblastoma multiforme. Molecular Cancer, 2018, 17, 74.	19.2	213
8	Screening of cell cycle fusion proteins to identify kinase signaling networks. Cell Cycle, 2015, 14, 1274-1281.	2.6	1
9	Casein Kinase 1δ Is an APC/CCdh1 Substrate that Regulates Cerebellar Granule Cell Neurogenesis. Cell Reports, 2015, 11, 249-260.	6.4	30
10	The Epigenetics of Medulloblastoma. , 2015, , 317-337.		0
11	The Bromodomain protein BRD4 controls HOTAIR, a long noncoding RNA essential for glioblastoma proliferation. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, 8326-8331.	7.1	186
12	GSK3 inhibitors stabilize Wee1 and reduce cerebellar granule cell progenitor proliferation. Cell Cycle, 2015, 14, 417-424.	2.6	8
13	The APC/C and CK1 in the developing brain. Oncotarget, 2015, 6, 16792-16793.	1.8	0
14	BET bromodomain proteins are required for glioblastoma cell proliferation. Epigenetics, 2014, 9, 611-620.	2.7	123
15	Casein Kinase 1Î^-dependent Wee1 Protein Degradation. Journal of Biological Chemistry, 2014, 289, 18893-18903.	3.4	22
16	Epigenetic pathways and glioblastoma treatment. Epigenetics, 2013, 8, 785-795.	2.7	54
17	Induction of ER stress in response to oxygen-glucose deprivation of cortical cultures involves the activation of the PERK and IRE-1 pathways and of caspase-12. Cell Death and Disease, 2011, 2, e149-e149.	6.3	137
18	Sigma Receptor Agonist 2-(4-Morpholinethyl)1 Phenylcyclohexanecarboxylate (Pre084) Increases GDNF and BiP Expression and Promotes Neuroprotection after Root Avulsion Injury. Journal of Neurotrauma, 2011, 28, 831-840.	3.4	53

CLARA PENAS

#	Article	IF	CITATIONS
19	Valproate reduces CHOP levels and preserves oligodendrocytes and axons after spinal cord injury. Neuroscience, 2011, 178, 33-44.	2.3	67
20	Autophagy, and BiP level decrease are early key events in retrograde degeneration of motoneurons. Cell Death and Differentiation, 2011, 18, 1617-1627.	11.2	48
21	Lack of a synergistic effect of a non-viral ALS gene therapy based on BDNF and a TTC fusion molecule. Orphanet Journal of Rare Diseases, 2011, 6, 10.	2.7	32
22	The APC/C Ubiquitin Ligase: From Cell Biology to Tumorigenesis. Frontiers in Oncology, 2011, 1, 60.	2.8	44
23	Fragment C of tetanus toxin, more than a carrier. Novel perspectives in non-viral ALS gene therapy. Journal of Molecular Medicine, 2010, 88, 297-308.	3.9	52
24	Cytoskeletal and Activity-Related Changes in Spinal Motoneurons after Root Avulsion. Journal of Neurotrauma, 2009, 26, 763-779.	3.4	40
25	Spinal cord injury induces endoplasmic reticulum stress with different cell-type dependent response. Journal of Neurochemistry, 2007, 102, 1242-1255.	3.9	143