Claire E Hulsebosch

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Neuronal-Glial Interactions Maintain Chronic Neuropathic Pain after Spinal Cord Injury. Neural Plasticity, 2017, 2017, 1-14.	2.2	90
2	Reactive oxygen species contribute to neuropathic pain and locomotor dysfunction via activation of CamKII in remote segments following spinal cord contusion injury in rats. Pain, 2013, 154, 1699-1708.	4.2	72
3	Spatial and temporal activation of spinal glial cells: Role of gliopathy in central neuropathic pain following spinal cord injury in rats. Experimental Neurology, 2012, 234, 362-372.	4.1	221
4	Calcium/calmodulin dependent kinase II contributes to persistent central neuropathic pain following spinal cord injury. Pain, 2012, 153, 710-721.	4.2	55
5	lonotropic glutamate receptors contribute to maintained neuronal hyperexcitability following spinal cord injury in rats. Experimental Neurology, 2010, 224, 321-324.	4.1	40
6	Peripheral and central sensitization in remote spinal cord regions contribute to central neuropathic pain after spinal cord injury. Pain, 2009, 147, 265-276.	4.2	209
7	Mechanisms of chronic central neuropathic pain after spinal cord injury. Brain Research Reviews, 2009, 60, 202-213.	9.0	262
8	Involvement of metabotropic glutamate receptors in excitatory amino acid and GABA release following spinal cord injury in rat. Journal of Neurochemistry, 2008, 79, 835-848.	3.9	43
9	Propentofylline attenuates allodynia, glial activation and modulates GABAergic tone after spinal cord injury in the rat. Pain, 2008, 138, 410-422.	4.2	121
10	Activation of p38 MAP kinase is involved in central neuropathic pain following spinal cord injury. Experimental Neurology, 2008, 213, 257-267.	4.1	140
11	Gliopathy ensures persistent inflammation and chronic pain after spinal cord injury. Experimental Neurology, 2008, 214, 6-9.	4.1	108
12	Increases in the activated forms of ERK 1/2, p38 MAPK, and CREB are correlated with the expression of at-level mechanical allodynia following spinal cord injury. Experimental Neurology, 2006, 199, 397-407.	4.1	167
13	Activation of Spinal GABA Receptors Attenuates Chronic Central Neuropathic Pain after Spinal Cord Injury. Journal of Neurotrauma, 2006, 23, 1111-1124.	3.4	118
14	RECENT ADVANCES IN PATHOPHYSIOLOGY AND TREATMENT OF SPINAL CORD INJURY. American Journal of Physiology - Advances in Physiology Education, 2002, 26, 238-255.	1.6	291
15	Rapid changes in expression of glutamate transporters after spinal cord injury. Brain Research, 2002, 927, 104-110.	2.2	74
16	IL-1 Receptor Antagonist Prevents Apoptosis and Caspase-3 Activation after Spinal Cord Injury. Journal of Neurotrauma, 2001, 18, 947-956.	3.4	157
17	Changes in Metabotropic Glutamate Receptor Expression Following Spinal Cord Injury. Experimental Neurology, 2001, 170, 244-257.	4.1	68
18	Reduction of Pathological and Behavioral Deficits Following Spinal Cord Contusion Injury with the Selective Cyclooxygenase-2 Inhibitor NS-398. Journal of Neurotrauma, 2001, 18, 409-423.	3.4	98

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#	Article	IF	CITATIONS
19	Bcl-xL Expression after Contusion to the Rat Spinal Cord. Journal of Neurotrauma, 2001, 18, 1267-1278.	3.4	40
20	Chronic Central Pain after Spinal Cord Injury. Journal of Neurotrauma, 1997, 14, 517-537.	3.4	257
21	Mechanical and thermal allodynia in chronic central pain following spinal cord injury. Pain, 1996, 68, 97-107.	4.2	250
22	Status of gross anatomy in the U.S. and Canada: Dilemma for the 21st century. Clinical Anatomy, 1994, 7, 275-296.	2.7	97
23	Schwann cell-neuronal interactions in the rat involve nerve growth factor. Journal of Comparative Neurology, 1990, 296, 114-122.	1.6	26
24	An analysis of the axon populations in the nerves to the pelvic viscera in the rat. Journal of Comparative Neurology, 1982, 211, 1-10.	1.6	179