## **Carole A Bartlett**

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/7204506/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Early Events of Secondary Degeneration after Partial Optic Nerve Transection: An Immunohistochemical Study. Journal of Neurotrauma, 2010, 27, 439-452.	3.4	98
2	Secondary Retinal Ganglion Cell Death and the Neuroprotective Effects of the Calcium Channel Blocker Lomerizine. , 2009, 50, 5456.		78
3	Myelin Sheath Decompaction, Axon Swelling, and Functional Loss during Chronic Secondary Degeneration in Rat Optic Nerve. , 2012, 53, 6093.		72
4	Secondary degeneration of the optic nerve following partial transection: The benefits of lomerizine. Experimental Neurology, 2009, 216, 219-230.	4.1	63
5	Oligodendroglia Are Particularly Vulnerable to Oxidative Damage after Neurotrauma <i>In Vivo</i> . Journal of Neuroscience, 2018, 38, 6491-6504.	3.6	63
6	Differential Effects of 670 and 830 nm Red near Infrared Irradiation Therapy: A Comparative Study of Optic Nerve Injury, Retinal Degeneration, Traumatic Brain and Spinal Cord Injury. PLoS ONE, 2014, 9, e104565.	2.5	39
7	Three Ca2+ channel inhibitors in combination limit chronic secondary degeneration following neurotrauma. Neuropharmacology, 2013, 75, 380-390.	4.1	36
8	An Unexpected Transient Breakdown of the Blood Brain Barrier Triggers Passage of Large Intravenously Administered Nanoparticles. Scientific Reports, 2016, 6, 22595.	3.3	34
9	Reactive species and oxidative stress in optic nerve vulnerable to secondary degeneration. Experimental Neurology, 2014, 261, 136-146.	4.1	32
10	Early Proliferation Does Not Prevent the Loss of Oligodendrocyte Progenitor Cells during the Chronic Phase of Secondary Degeneration in a CNS White Matter Tract. PLoS ONE, 2013, 8, e65710.	2.5	31
11	Paranode Abnormalities and Oxidative Stress in Optic Nerve Vulnerable to Secondary Degeneration: Modulation by 670 nm Light Treatment. PLoS ONE, 2013, 8, e66448.	2.5	30
12	Differential responses to increasing numbers of mild traumatic brain injury in a rodent closedâ€head injury model. Journal of Neurochemistry, 2019, 149, 660-678.	3.9	20
13	Low Intensity Repetitive Transcranial Magnetic Stimulation Does Not Induce Cell Survival or Regeneration in a Mouse Optic Nerve Crush Model. PLoS ONE, 2015, 10, e0126949.	2.5	19
14	Specific combinations of ion channel inhibitors reduce excessive Ca2+ influx as a consequence of oxidative stress and increase neuronal and glial cell viability in vitro. Neuroscience, 2016, 339, 450-462.	2.3	12
15	Prolonged glutamate excitotoxicity increases GluR1 immunoreactivity but decreases mRNA of GluR1 and associated regulatory proteins inÂdissociated rat retinae inÂvitro. Biochimie, 2015, 112, 160-171.	2.6	10
16	Acute Cellular and Functional Changes With a Combinatorial Treatment of Ion Channel Inhibitors Following Spinal Cord Injury. Frontiers in Molecular Neuroscience, 2020, 13, 85.	2.9	7
17	Comparative assessment of phototherapy protocols for reduction of oxidative stress in partially transected spinal cord slices undergoing secondary degeneration. BMC Neuroscience, 2016, 17, 21.	1.9	6
18	The effects of a combination of ion channel inhibitors on pathology in a model of demyelinating disease. Multiple Sclerosis and Related Disorders, 2019, 34, 1-8.	2.0	6

#	Article	IF	CITATIONS
19	Comparison of ion channel inhibitor combinations for limiting secondary degeneration following partial optic nerve transection. Experimental Brain Research, 2019, 237, 161-171.	1.5	4
20	Comparing modes of delivery of a combination of ion channel inhibitors for limiting secondary degeneration following partial optic nerve transection. Scientific Reports, 2019, 9, 15297.	3.3	3
21	Delayed treatment of secondary degeneration following acute optic nerve transection using a combination of ion channel inhibitors. Neural Regeneration Research, 2017, 12, 307.	3.0	3
22	Characterization of polymeric nanoparticles for treatment of partial injury to the central nervous system. Data in Brief, 2016, 7, 152-156.	1.0	1