Alan I Faden

List of Publications by Year
in descending order

154 papers	13,714 citations	12330 69 h-index	23533 111 g-index
162 all docs	162 docs citations	162 times ranked	14093 citing authors

#	Article	IF	Citations
1	Sexual dimorphism in neurological function after SCI is associated with disrupted neuroinflammation in both injured spinal cord and brain. Brain, Behavior, and Immunity, 2022, 101, 1-22.	4.1	17
2	Functional and transcriptional profiling of microglial activation during the chronic phase of TBI identifies an age-related driver of poor outcome in old mice. GeroScience, 2022, 44, 1407-1440.	4.6	16
3	Enhanced Akt/GSKâ€3Î²/CREB signaling mediates the antiâ€inflammatory actions of mGluR5 positive allosteric modulators in microglia and following traumatic brain injury in male mice. Journal of Neurochemistry, 2021, 156, 225-248.	3.9	24
4	Spinal cord injury alters microRNA and CD81+ exosome levels in plasma extracellular nanoparticles with neuroinflammatory potential. Brain, Behavior, and Immunity, 2021, 92, 165-183.	4.1	62
5	Proton extrusion during oxidative burst in microglia exacerbates pathological acidosis following traumatic brain injury. Glia, 2021, 69, 746-764.	4.9	42
6	Acute colitis during chronic experimental traumatic brain injury in mice induces dysautonomia and persistent extraintestinal, systemic, and CNS inflammation with exacerbated neurological deficits. Journal of Neuroinflammation, 2021, 18, 24.	7.2	31
7	Bidirectional Brain-Systemic Interactions and Outcomes After TBI. Trends in Neurosciences, 2021, 44, 406-418.	8.6	17
8	PLA2G4A/cPLA2-mediated lysosomal membrane damage leads to inhibition of autophagy and neurodegeneration after brain trauma. Autophagy, 2020, 16, 466-485.	9.1	95
9	Sustained neuronal and microglial alterations are associated with diverse neurobehavioral dysfunction long after experimental brain injury. Neurobiology of Disease, 2020, 136, 104713.	4.4	41
10	Delayed microglial depletion after spinal cord injury reduces chronic inflammation and neurodegeneration in the brain and improves neurological recovery in male mice. Theranostics, 2020, 10, 11376-11403.	10.0	88
11	Mithramycin selectively attenuates DNA-damage-induced neuronal cell death. Cell Death and Disease, 2020, 11, 587.	6.3	8
12	Irradiation-Induced Upregulation of miR-711 Inhibits DNA Repair and Promotes Neurodegeneration Pathways. International Journal of Molecular Sciences, 2020, 21, 5239.	4.1	7
13	Early or Late Bacterial Lung Infection Increases Mortality After Traumatic Brain Injury in Male Mice and Chronically Impairs Monocyte Innate Immune Function. Critical Care Medicine, 2020, 48, e418-e428.	0.9	22
14	Longitudinal Assessment of Sensorimotor Function after Controlled Cortical Impact in Mice: Comparison of Beamwalk, Rotarod, and Automated Gait Analysis Tests. Journal of Neurotrauma, 2020, 37, 2709-2717.	3.4	6
15	Function and Mechanisms of Truncated BDNF Receptor TrkB.T1 in Neuropathic Pain. Cells, 2020, 9, 1194.	4.1	47
16	Putative mGluR4 positive allosteric modulators activate Gi-independent anti-inflammatory mechanisms in microglia. Neurochemistry International, 2020, 138, 104770.	3.8	2
17	Down-Regulation of miR-23a-3p Mediates Irradiation-Induced Neuronal Apoptosis. International Journal of Molecular Sciences, 2020, 21, 3695.	4.1	17
18	Dementia, Depression, and Associated Brain Inflammatory Mechanisms after Spinal Cord Injury. Cells, 2020, 9, 1420.	4.1	38

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19	Microglial Depletion with CSF1R Inhibitor During Chronic Phase of Experimental Traumatic Brain Injury Reduces Neurodegeneration and Neurological Deficits. Journal of Neuroscience, 2020, 40, 2960-2974.	3.6	193
20	Interferon-Î² Plays a Detrimental Role in Experimental Traumatic Brain Injury by Enhancing Neuroinflammation That Drives Chronic Neurodegeneration. Journal of Neuroscience, 2020, 40, 2357-2370.	3.6	78
21	Inhibition of microRNA-711 limits angiopoietin-1 and Akt changes, tissue damage, and motor dysfunction after contusive spinal cord injury in mice. Cell Death and Disease, 2019, 10, 839.	6.3	24
22	Old age increases microglial senescence, exacerbates secondary neuroinflammation, and worsens neurological outcomes after acute traumatic brain injury in mice. Neurobiology of Aging, 2019, 77, 194-206.	3.1	99
23	Inhibition of NOX2 signaling limits pain-related behavior and improves motor function in male mice after spinal cord injury: Participation of IL-10/miR-155 pathways. Brain, Behavior, and Immunity, 2019, 80, 73-87.	4.1	48
24	Inhibition of miR-155 Limits Neuroinflammation and Improves Functional Recovery After Experimental Traumatic Brain Injury in Mice. Neurotherapeutics, 2019, 16, 216-230.	4.4	57
25	Neutral Sphingomyelinase Inhibition Alleviates LPS-Induced Microglia Activation and Neuroinflammation after Experimental Traumatic Brain Injury. Journal of Pharmacology and Experimental Therapeutics, 2019, 368, 338-352.	2.5	42
26	Sex Differences in Acute Neuroinflammation after Experimental Traumatic Brain Injury Are Mediated by Infiltrating Myeloid Cells. Journal of Neurotrauma, 2019, 36, 1040-1053.	3.4	105
27	Traumatic meningeal injury and repair mechanisms. Nature Immunology, 2018, 19, 431-432.	14.5	1
28	Chronic Alterations in Systemic Immune Function after Traumatic Brain Injury. Journal of Neurotrauma, 2018, 35, 1419-1436.	3.4	79
29	Comparing effects of CDK inhibition and E2F1/2 ablation on neuronal cell death pathways in vitro and after traumatic brain injury. Cell Death and Disease, 2018, 9, 1121.	6.3	17
30	MicroRNA-711â€"Induced Downregulation of Angiopoietin-1 Mediates Neuronal Cell Death. Journal of Neurotrauma, 2018, 35, 2462-2481.	3.4	23
31	Colitisâ€Induced Neurobehavioral Deficits Following Chronic Brain Injury. FASEB Journal, 2018, 32, 921.8.	0.5	0
32	Truncated TrkB.T1-Mediated Astrocyte Dysfunction Contributes to Impaired Motor Function and Neuropathic Pain after Spinal Cord Injury. Journal of Neuroscience, 2017, 37, 3956-3971.	3.6	72
33	Microglial-derived microparticles mediate neuroinflammation after traumatic brain injury. Journal of Neuroinflammation, 2017, 14, 47.	7.2	228
34	Bidirectional brain-gut interactions and chronic pathological changes after traumatic brain injury in mice. Brain, Behavior, and Immunity, 2017, 66, 56-69.	4.1	109
35	NOX2 deficiency alters macrophage phenotype through an IL-10/STAT3 dependent mechanism: implications for traumatic brain injury. Journal of Neuroinflammation, 2017, 14, 65.	7.2	65
36	Cell cycle inhibition limits development and maintenance of neuropathic pain following spinal cord injury. Pain, 2016, 157, 488-503.	4.2	51

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37	Endoplasmic Reticulum Stress and Disrupted Neurogenesis in the Brain Are Associated with Cognitive Impairment and Depressive-Like Behavior after Spinal Cord Injury. Journal of Neurotrauma, 2016, 33, 1919-1935.	3.4	94
38	NOX2 drives M1-like microglial/macrophage activation and neurodegeneration following experimental traumatic brain injury. Brain, Behavior, and Immunity, 2016, 58, 291-309.	4.1	152
39	Cell cycle inhibition reduces inflammatory responses, neuronal loss, and cognitive deficits induced by hypobaria exposure following traumatic brain injury. Journal of Neuroinflammation, 2016, 13, 299.	7.2	34
40	Simulated Aeromedical Evacuation Exacerbates Experimental Brain Injury. Journal of Neurotrauma, 2016, 33, 1292-1302.	3.4	29
41	Microglial/Macrophage Polarization Dynamics following Traumatic Brain Injury. Journal of Neurotrauma, 2016, 33, 1732-1750.	3.4	248
42	Progressive inflammationâ€mediated neurodegeneration after traumatic brain or spinal cord injury. British Journal of Pharmacology, 2016, 173, 681-691.	5.4	217
43	Chronic Decrease in Wakefulness and Disruption of Sleep-Wake Behavior after Experimental Traumatic Brain Injury. Journal of Neurotrauma, 2015, 32, 289-296.	3.4	62
44	Voluntary Exercise Preconditioning Activates Multiple Antiapoptotic Mechanisms and Improves Neurological Recovery after Experimental Traumatic Brain Injury. Journal of Neurotrauma, 2015, 32, 1347-1360.	3.4	43
45	Neuroprotection for traumatic brain injury. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2015, 127, 343-366.	1.8	68
46	S100B Inhibition Reduces Behavioral and Pathologic Changes in Experimental Traumatic Brain Injury. Journal of Cerebral Blood Flow and Metabolism, 2015, 35, 2010-2020.	4.3	37
47	Acyl-2-aminobenzimidazoles: A novel class of neuroprotective agents targeting mGluR5. Bioorganic and Medicinal Chemistry, 2015, 23, 2211-2220.	3.0	21
48	Neuroprotection in acute brain injury: an up-to-date review. Critical Care, 2015, 19, 186.	5.8	120
49	Cyclopropyl-containing positive allosteric modulators of metabotropic glutamate receptor subtype 5. Bioorganic and Medicinal Chemistry Letters, 2015, 25, 2275-2279.	2.2	9
50	Function and Mechanisms of Autophagy in Brain and Spinal Cord Trauma. Antioxidants and Redox Signaling, 2015, 23, 565-577.	5.4	164
51	Ablation of the transcription factors E2F1-2 limits neuroinflammation and associated neurological deficits after contusive spinal cord injury. Cell Cycle, 2015, 14, 3698-3712.	2.6	32
52	Chronic Neurodegeneration After Traumatic Brain Injury: Alzheimer Disease, Chronic Traumatic Encephalopathy, or Persistent Neuroinflammation?. Neurotherapeutics, 2015, 12, 143-150.	4.4	199
53	Downregulation of miR-23a and miR-27a following Experimental Traumatic Brain Injury Induces Neuronal Cell Death through Activation of Proapoptotic Bcl-2 Proteins. Journal of Neuroscience, 2014, 34, 10055-10071.	3.6	129
54	Novel mGluR5 Positive Allosteric Modulator Improves Functional Recovery, Attenuates Neurodegeneration, and Alters Microglial Polarization after Experimental Traumatic Brain Injury. Neurotherapeutics, 2014, 11, 857-869.	4.4	70

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55	Neuroprotective Strategies for Traumatic Brain Injury: Improving Clinical Translation. International Journal of Molecular Sciences, 2014, 15, 1216-1236.	4.1	143
56	Isolated spinal cord contusion in rats induces chronic brain neuroinflammation, neurodegeneration, and cognitive impairment. Cell Cycle, 2014, 13, 2446-2458.	2.6	90
57	Progressive Neurodegeneration After Experimental Brain Trauma. Journal of Neuropathology and Experimental Neurology, 2014, 73, 14-29.	1.7	406
58	Boc-protected 1-(3-oxocycloalkyl)ureas via a one-step Curtius rearrangement: mechanism and scope. Tetrahedron Letters, 2014, 55, 842-844.	1.4	16
59	Neurotherapeutics: Concept, Translation, Transition. Neurotherapeutics, 2014, 11, 1.	4.4	1
60	Impaired autophagy flux is associated with neuronal cell death after traumatic brain injury. Autophagy, 2014, 10, 2208-2222.	9.1	256
61	Spinal Cord Injury Causes Brain Inflammation Associated with Cognitive and Affective Changes: Role of Cell Cycle Pathways. Journal of Neuroscience, 2014, 34, 10989-11006.	3.6	201
62	PARP-1 Inhibition Attenuates Neuronal Loss, Microglia Activation and Neurological Deficits after Traumatic Brain Injury. Journal of Neurotrauma, 2014, 31, 758-772.	3.4	103
63	CR8, a Novel Inhibitor of CDK, Limits Microglial Activation, Astrocytosis, Neuronal Loss, and Neurologic Dysfunction after Experimental Traumatic Brain Injury. Journal of Cerebral Blood Flow and Metabolism, 2014, 34, 502-513.	4.3	56
64	Repeated Mild Traumatic Brain Injury Causes Chronic Neuroinflammation, Changes in Hippocampal Synaptic Plasticity, and Associated Cognitive Deficits. Journal of Cerebral Blood Flow and Metabolism, 2014, 34, 1223-1232.	4.3	207
65	Inhibition of amyloid precursor protein secretases reduces recovery after spinal cord injury. Brain Research, 2014, 1560, 73-82.	2.2	22
66	Positive Allosteric Modulators (PAMs) of Metabotropic Glutamate Receptor 5 (mGluR5) Attenuate Microglial Activation. CNS and Neurological Disorders - Drug Targets, 2014, 13, 558-566.	1.4	19
67	Selective CDK inhibitors: promising candidates for future clinical traumatic brain injury trials. Neural Regeneration Research, 2014, 9, 1578.	3.0	19
68	Cell Cycle Activation Contributes to Increased Neuronal Activity in the Posterior Thalamic Nucleus and Associated Chronic Hyperesthesia after Rat Spinal Cord Contusion. Neurotherapeutics, 2013, 10, 520-538.	4.4	37
69	TrkB.T1 Contributes to Neuropathic Pain after Spinal Cord Injury through Regulation of Cell Cycle Pathways. Journal of Neuroscience, 2013, 33, 12447-12463.	3.6	70
70	Late exercise reduces neuroinflammation and cognitive dysfunction after traumatic brain injury. Neurobiology of Disease, 2013, 54, 252-263.	4.4	127
71	Neuroprotective Effects of Geranylgeranylacetone in Experimental Traumatic Brain Injury. Journal of Cerebral Blood Flow and Metabolism, 2013, 33, 1897-1908.	4.3	39
72	Traumatic brain injury in aged animals increases lesion size and chronically alters microglial/macrophage classical and alternative activation states. Neurobiology of Aging, 2013, 34, 1397-1411.	3.1	213

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73	Estimation of Ligand Efficacies of Metabotropic Glutamate Receptors from Conformational Forces Obtained from Molecular Dynamics Simulations. Journal of Chemical Information and Modeling, 2013, 53, 1337-1349.	5.4	3
74	Activation of mGluR5 and Inhibition of NADPH Oxidase Improves Functional Recovery after Traumatic Brain Injury. Journal of Neurotrauma, 2013, 30, 403-412.	3.4	78
75	Propofol Limits Microglial Activation after Experimental Brain Trauma through Inhibition of Nicotinamide Adenine Dinucleotide Phosphate Oxidase. Anesthesiology, 2013, 119, 1370-1388.	2.5	66
76	Selective CDK Inhibitor Limits Neuroinflammation and Progressive Neurodegeneration after Brain Trauma. Journal of Cerebral Blood Flow and Metabolism, 2012, 32, 137-149.	4.3	82
77	Delayed cell cycle pathway modulation facilitates recovery after spinal cord injury. Cell Cycle, 2012, 11, 1782-1795.	2.6	41
78	Comparing the Predictive Value of Multiple Cognitive, Affective, and Motor Tasks after Rodent Traumatic Brain Injury. Journal of Neurotrauma, 2012, 29, 2475-2489.	3.4	91
79	Cyclin D1 Gene Ablation Confers Neuroprotection in Traumatic Brain Injury. Journal of Neurotrauma, 2012, 29, 813-827.	3.4	53
80	Delayed expression of cell cycle proteins contributes to astroglial scar formation and chronic inflammation after rat spinal cord contusion. Journal of Neuroinflammation, 2012, 9, 169.	7.2	53
81	Overâ€expression of HSP70 attenuates caspaseâ€dependent and caspaseâ€independent pathways and inhibits neuronal apoptosis. Journal of Neurochemistry, 2012, 123, 542-554.	3.9	104
82	Characterization of inflammatory gene expression and galectin-3 function after spinal cord injury in mice. Brain Research, 2012, 1475, 96-105.	2.2	32
83	Metabotropic glutamate receptorâ€mediated signaling in neuroglia. Environmental Sciences Europe, 2012, 1, 136-150.	5.5	36
84	CR8, a Selective and Potent CDK Inhibitor, Provides Neuroprotection in Experimental Traumatic Brain Injury. Neurotherapeutics, 2012, 9, 405-421.	4.4	49
85	Combined inhibition of cell death induced by apoptosis inducing factor and caspases provides additive neuroprotection in experimental traumatic brain injury. Neurobiology of Disease, 2012, 46, 745-758.	4.4	52
86	Delayed mGluR5 activation limits neuroinflammation and neurodegeneration after traumatic brain injury. Journal of Neuroinflammation, 2012, 9, 43.	7.2	144
87	Inhibition of E2F1/CDK1 Pathway Attenuates Neuronal Apoptosis In Vitro and Confers Neuroprotection after Spinal Cord Injury In Vivo. PLoS ONE, 2012, 7, e42129.	2.5	46
88	Cell Cycle Activation and Spinal Cord Injury. Neurotherapeutics, 2011, 8, 221-228.	4.4	63
89	Delayed inflammatory mRNA and protein expression after spinal cord injury. Journal of Neuroinflammation, 2011, 8, 130.	7.2	66
90	Microglial activation and traumatic brain injury. Annals of Neurology, 2011, 70, 345-346.	5.3	21

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91	Cell Death Mechanisms and Modulation in Traumatic Brain Injury. Neurotherapeutics, 2010, 7, 3-12.	4.4	236
92	Fluid-percussionâ€“induced traumatic brain injury model in rats. Nature Protocols, 2010, 5, 1552-1563.	12.0	138
93	A combined scoring method to assess behavioral recovery after mouse spinal cord injury. Neuroscience Research, 2010, 67, 117-125.	1.9	55
94	Neuroprotection for traumatic brain injury: translational challenges and emerging therapeutic strategies. Trends in Pharmacological Sciences, 2010, 31, 596-604.	8.7	485
95	Programmed Neuronal Cell Death Mechanisms in CNS Injury. , 2010, , 169-200.		4
96	Activation of Metabotropic Glutamate Receptor 5 Modulates Microglial Reactivity and Neurotoxicity by Inhibiting NADPH Oxidase. Journal of Biological Chemistry, 2009, 284, 15629-15639.	3.4	96
97	Activation of metabotropic glutamate receptor 5 improves recovery after spinal cord injury in rodents. Annals of Neurology, 2009, 66, 63-74.	5.3	71
98	Metabotropic glutamate receptor 5 activation inhibits microglial associated inflammation and neurotoxicity. Glia, 2009, 57, 550-560.	4.9	157
99	Metabotropic Glutamate Receptors as Targets for Multipotential Treatment of Neurological Disorders. Neurotherapeutics, 2009, 6, 94-107.	4.4	112
100	Cell Cycle Activation and CNS Injury. Neurotoxicity Research, 2009, 16, 221-237.	2.7	55
101	Amyloid precursor protein secretases as therapeutic targets for traumatic brain injury. Nature Medicine, 2009, 15, 377-379.	30.7	219
102	Roscovitine Reduces Neuronal Loss, Glial Activation, and Neurologic Deficits after Brain Trauma. Journal of Cerebral Blood Flow and Metabolism, 2008, 28, 1845-1859.	4.3	108
103	Cell cycle activation contributes to post-mitotic cell death and secondary damage after spinal cord injury. Brain, 2007, 130, 2977-2992.	7.6	149
104	Neuroprotection. Archives of Neurology, 2007, 64, 794.	4.5	110
105	Role of Cell Cycle Proteins in CNS Injury. Neurochemical Research, 2007, 32, 1799-1807.	3.3	92
106	Expression of two temporally distinct microglia-related gene clusters after spinal cord injury. Glia, 2006, 53, 420-433.	4.9	72
107	Gene expression profiling of experimental traumatic spinal cord injury as a function of distance from impact site and injury severity. Physiological Genomics, 2005, 22, 368-381.	2.3	95
108	Novel small peptides with neuroprotective and nootropic properties. Journal of Alzheimer's Disease, 2005, 6, S93-S97.	2.6	35

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109	Identification of Novel Neuroprotective Agents Using Pharmacophore Modeling. Chemistry and Biodiversity, 2005, 2, 1564-1570.	2.1	6
110	Neurotrauma. , 2005, , 95-127.		0
111	Role of the Cell Cycle in the Pathobiology of Central Nervous System Trauma. Cell Cycle, 2005, 4, 1286-1293.	2.6	107
112	Cell cycle inhibition provides neuroprotection and reduces glial proliferation and scar formation after traumatic brain injury. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 8333-8338.	7.1	355
113	Ceramide induces neuronal apoptosis through mitogen-activated protein kinases and causes release of multiple mitochondrial proteins. Molecular and Cellular Neurosciences, 2005, 29, 355-371.	2.2	92
114	Neuroprotective effects of novel small peptides in vitro and after brain injury. Neuropharmacology, 2005, 49, 410-424.	4.1	90
115	Novel Neuroprotective Tripeptides and Dipeptides. Annals of the New York Academy of Sciences, 2005, 1053, 472-481.	3.8	12
116	BOK and NOXA Are Essential Mediators of p53-dependent Apoptosis. Journal of Biological Chemistry, 2004, 279, 28367-28374.	3.4	127
117	MGLuR5 activation reduces Î²â€amyloidâ€induced cell death in primary neuronal cultures and attenuates translocation of cytochrome c and apoptosisâ€inducing factor. Journal of Neurochemistry, 2004, 89, 1528-1536.	3.9	66
118	Mechanisms of neural cell death: Implications for development of neuroprotective treatment strategies. Neurotherapeutics, 2004, 1, 5-16.	4.4	0
119	Gene profiling in spinal cord injury shows role of cell cycle in neuronal death. Annals of Neurology, 2003, 53, 454-468.	5.3	261
120	Novel Diketopiperazine Enhances Motor and Cognitive Recovery after Traumatic Brain Injury in Rats and Shows Neuroprotection<i>In Vitro</i>and<i>In Vivo</i>. Journal of Cerebral Blood Flow and Metabolism, 2003, 23, 342-354.	4.3	72
121	Neuroprotective and Nootropic Actions of a Novel Cyclized Dipeptide after Controlled Cortical Impact Injury in Mice. Journal of Cerebral Blood Flow and Metabolism, 2003, 23, 355-363.	4.3	43
122	Ceramide-induced neuronal apoptosis is associated with dephosphorylation of Akt, BAD, FKHR, GSK-3Î², and induction of the mitochondrial-dependent intrinsic caspase pathway. Molecular and Cellular Neurosciences, 2003, 22, 365-382.	2.2	150
123	Gene Expression Profile Changes Are Commonly Modulated across Models and Species after Traumatic Brain Injury. Journal of Neurotrauma, 2003, 20, 907-927.	3.4	109
124	Multiple Caspases Are Activated after Traumatic Brain Injury: Evidence for Involvement in Functional Outcome. Journal of Neurotrauma, 2002, 19, 1155-1170.	3.4	111
125	Neuroprotection and traumatic brain injury: theoretical option or realistic proposition. Current Opinion in Neurology, 2002, 15, 707-712.	3.6	92
126	Neuroprotection and traumatic brain injury: theoretical option or realistic proposition. Current Opinion in Neurology, 2002, 15, 707-712.	3.6	79

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127	Neuronal and glial mGluR5 modulation prevents stretch-induced enhancement of NMDA receptor current. Pharmacology Biochemistry and Behavior, 2002, 73, 287-298.	2.9	54
128	Exacerbation of Neuronal Cell Death by Activation of Group I Metabotropic Glutamate Receptors: Role of NMDA Receptors and Arachidonic Acid Release. Experimental Neurology, 2001, 169, 449-460.	4.1	29
129	Differential Expression of Apoptotic Protease-Activating Factor-1 and Caspase-3 Genes and Susceptibility to Apoptosis during Brain Development and after Traumatic Brain Injury. Journal of Neuroscience, 2001, 21, 7439-7446.	3.6	249
130	Traumatic brain injury: Developmental differences in glutamate receptor response and the impact on treatment. Mental Retardation and Developmental Disabilities Research Reviews, 2001, 7, 235-248.	3.6	33
131	Caspase-Dependent Apoptotic Pathways in CNS Injury. Molecular Neurobiology, 2001, 24, 131-144.	4.0	144
132	Selective mGluR5 antagonists MPEP and SIB-1893 decrease NMDA or glutamate-mediated neuronal toxicity through actions that reflect NMDA receptor antagonism. British Journal of Pharmacology, 2000, 131, 1429-1437.	5.4	179
133	Early neuronal expression of tumor necrosis factor-Î± after experimental brain injury contributes to neurological impairment. Journal of Neuroimmunology, 1999, 95, 115-125.	2.3	248
134	Behavioral Responses of C57BL/6, FVB/N, and 129/SvEMS Mouse Strains to Traumatic Brain Injury: Implications for Gene Targeting Approaches to Neurotrauma. Journal of Neurotrauma, 1999, 16, 377-389.	3.4	95
135	Traumatic brain injury causes delayed motor and cognitive impairment in a mutant mouse strain known to exhibit delayed wallerian degeneration. Journal of Neuroscience Research, 1998, 53, 718-727.	2.9	50
136	Interleukin-10 Improves Outcome and Alters Proinflammatory Cytokine Expression after Experimental Traumatic Brain Injury. Experimental Neurology, 1998, 153, 143-151.	4.1	234
137	Sustained Sensory/Motor and Cognitive Deficits With Neuronal Apoptosis Following Controlled Cortical Impact Brain Injury in the Mouse. Journal of Neurotrauma, 1998, 15, 599-614.	3.4	290
138	Effect of Traumatic Brain Injury on Mouse Spatial and Nonspatial Learning in the Barnes Circular Maze. Journal of Neurotrauma, 1998, 15, 1037-1046.	3.4	114
139	Traumatic brain injury causes delayed motor and cognitive impairment in a mutant mouse strain known to exhibit delayed wallerian degeneration. Journal of Neuroscience Research, 1998, 53, 718-727.	2.9	2
140	Activation of Metabotropic Glutamate Receptor Subtype mGluR1 Contributes to Post-Traumatic Neuronal Injury. Journal of Neuroscience, 1996, 16, 6012-6020.	3.6	113
141	Pharmacological Treatment of Central Nervous System Trauma. Basic and Clinical Pharmacology and Toxicology, 1996, 78, 12-17.	0.0	73
142	Hypoglycemia prevents increase in lactic acidosis during reperfusion after temporary cerebral ischemia in rats. NMR in Biomedicine, 1995, 8, 171-178.	2.8	8
143	Dissociation of Adenosine Levels from Bioenergetic State in Experimental Brain Trauma: Potential Role in Secondary Injury. Journal of Cerebral Blood Flow and Metabolism, 1994, 14, 853-861.	4.3	70
144	Pretreatment with NMDA antagonists limits release of excitatory amino acids following traumatic brain injury. Neuroscience Letters, 1992, 136, 165-168.	2.1	116

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145	Effect of Dichloroacetate on Recovery of Brain Lactate, Phosphorus Energy Metabolites, and Glutamate during Reperfusion after Complete Cerebral Ischemia in Rats. Journal of Cerebral Blood Flow and Metabolism, 1992, 12, 1030-1038.	4.3	43
146	Effects of Hyperglycemia on the Time Course of Changes in Energy Metabolism and pH during Global Cerebral Ischemia and Reperfusion in Rats: Correlation of¹H and³¹P NMR Spectroscopy with Fatty Acid and Excitatory Amino Acid Levels. Journal of Cerebral Blood Flow and Metabolism, 1992, 12, 456-468.	4.3	55
147	Metabolic changes in rabbit spinal cord after trauma: Magnetic resonance spectroscopy studies. Annals of Neurology, 1989, 25, 26-31.	5.3	37
148	Effect of Impact Trauma on Neurotransmitter and Nonneurotransmitter Amino Acids in Rat Spinal Cord. Journal of Neurochemistry, 1989, 52, 1529-1536.	3.9	80
149	A potential role for excitotoxins in the pathophysiology of spinal cord injury. Annals of Neurology, 1988, 23, 623-626.	5.3	358
150	31P NMR characterization of graded traumatic brain injury in rats. Magnetic Resonance in Medicine, 1988, 6, 37-48.	3.0	48
151	Nonedited1H NMR lactate/n-acetyl aspartate ratios and thein vivo determination of lactate concentration in brain. Magnetic Resonance in Medicine, 1988, 7, 95-99.	3.0	21
152	Changes in Cellular Bioenergetic State Following Graded Traumatic Brain Injury in Rats: Determination by Phosphorus 31 Magnetic Resonance Spectroscopy. Journal of Neurotrauma, 1988, 5, 315-330.	3.4	92
153	31P magnetic resonance spectroscopy of traumatic spinal cord injury. Magnetic Resonance in Medicine, 1987, 5, 390-394.	3.0	23
154	Alterations in Lipid Metabolism, Na+,K+-ATPase Activity, and Tissue Water Content of Spinal Cord Following Experimental Traumatic Injury. Journal of Neurochemistry, 1987, 48, 1809-1816.	3.9	84