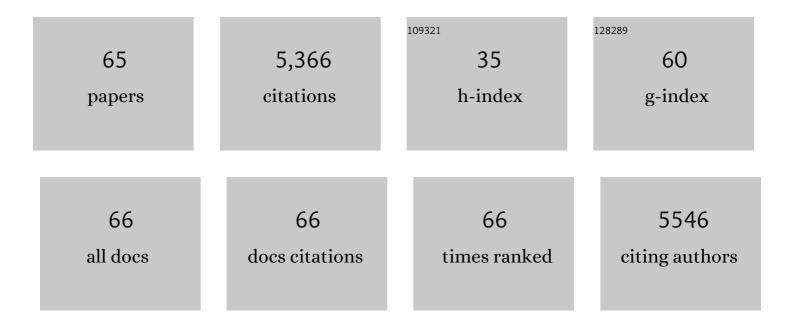
## Jaroslaw Aronowski

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/6824878/publications.pdf

Version: 2024-02-01



#	Article	IF	CITATIONS
1	Mechanisms of Damage After Cerebral Hemorrhage. , 2022, , 92-102.e9.		Ο
2	The Stroke Preclinical Assessment Network: Rationale, Design, Feasibility, and Stage 1 Results. Stroke, 2022, 53, 1802-1812.	2.0	22
3	Lactoferrin and hematoma detoxification after intracerebral hemorrhage. Biochemistry and Cell Biology, 2021, 99, 97-101.	2.0	9
4	Agonism of the Î $\pm$ 7-acetylcholine receptor/PI3K/Akt pathway promotes neuronal survival after subarachnoid hemorrhage in mice. Experimental Neurology, 2021, 344, 113792.	4.1	6
5	Optimized lactoferrin as a highly promising treatment for intracerebral hemorrhage: Pre-clinical experience. Journal of Cerebral Blood Flow and Metabolism, 2021, 41, 53-66.	4.3	21
6	Excitatory pathway engaging glutamate, calcineurin, and NFAT upregulates IL-4 in ischemic neurons to polarize microglia. Journal of Cerebral Blood Flow and Metabolism, 2020, 40, 513-527.	4.3	29
7	Brain Cleanup as a Potential Target for Poststroke Recovery. Stroke, 2020, 51, 958-966.	2.0	34
8	The Mitochondria-Derived Peptide Humanin Improves Recovery from Intracerebral Hemorrhage: Implication of Mitochondria Transfer and Microglia Phenotype Change. Journal of Neuroscience, 2020, 40, 2154-2165.	3.6	43
9	Contribution of TRPC Channels in Neuronal Excitotoxicity Associated With Neurodegenerative Disease and Ischemic Stroke. Frontiers in Cell and Developmental Biology, 2020, 8, 618663.	3.7	18
10	Aging exacerbates neutrophil pathogenicity in ischemic stroke. Aging, 2020, 12, 436-461.	3.1	33
11	International Collaborations Are Essential for Stroke. Stroke, 2019, 50, 2993-2994.	2.0	1
12	Serial Metabolic Evaluation of Perihematomal Tissues in the Intracerebral Hemorrhage Pig Model. Frontiers in Neuroscience, 2019, 13, 888.	2.8	12
13	Neutrophils, the Felons of the Brain. Stroke, 2019, 50, e42-e43.	2.0	19
14	Aspirin in stroke patients modifies the immunomodulatory interactions of marrow stromal cells and monocytes. Brain Research, 2019, 1720, 146298.	2.2	10
15	Beneficial Role of Neutrophils Through Function of Lactoferrin After Intracerebral Hemorrhage. Stroke, 2018, 49, 1241-1247.	2.0	34
16	Association Between Splenic Contraction and the Systemic Inflammatory Response After Acute Ischemic Stroke Varies with Age and Race. Translational Stroke Research, 2018, 9, 484-492.	4.2	16
17	Delivery of xenon-containing echogenic liposomes inhibits early brain injury following subarachnoid hemorrhage. Scientific Reports, 2018, 8, 450.	3.3	36
18	Serial quantitative neuroimaging of iron in the intracerebral hemorrhage pig model. Journal of Cerebral Blood Flow and Metabolism, 2018, 38, 375-381.	4.3	18

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19	Protective Effects of Autologous Bone Marrow Mononuclear Cells After Administering t-PA in an Embolic Stroke Model. Translational Stroke Research, 2018, 9, 135-145.	4.2	26
20	Call for Basic Science Papers. Stroke, 2018, 49, 1803-1804.	2.0	0
21	High Appraisal of Methodological Quality of Basic Science Articles Published in Stroke. Stroke, 2017, 48, 2337-2338.	2.0	0
22	Neutrophil polarization by IL-27 as a therapeutic target for intracerebral hemorrhage. Nature Communications, 2017, 8, 602.	12.8	114
23	Soluble CD163 in intracerebral hemorrhage: biomarker for perihematomal edema. Annals of Clinical and Translational Neurology, 2017, 4, 793-800.	3.7	19
24	Cryopreservation of Bone Marrow Mononuclear Cells Alters Their Viability and Subpopulation Composition but Not Their Treatment Effects in a Rodent Stroke Model. Stem Cells International, 2016, 2016, 1-7.	2.5	11
25	Various Cell Populations Within the Mononuclear Fraction of Bone Marrow Contribute to the Beneficial Effects of Autologous Bone Marrow Cell Therapy in a Rodent Stroke Model. Translational Stroke Research, 2016, 7, 322-330.	4.2	28
26	Reporting Standards for Preclinical Studies of Stroke Therapy. Stroke, 2016, 47, 2435-2438.	2.0	33
27	Acute splenic responses in patients with ischemic stroke and intracerebral hemorrhage. Journal of Cerebral Blood Flow and Metabolism, 2016, 36, 1012-1021.	4.3	51
28	Mechanisms of Cerebral Hemorrhage. , 2016, , 102-112.e6.		0
29	Phagocytosis Assay of Microglia for Dead Neurons in Primary Rat Brain Cell Cultures. Bio-protocol, 2016, 6, .	0.4	6
30	Cleaning up after <scp>ICH</scp> : the role of Nrf2 in modulating microglia function and hematoma clearance. Journal of Neurochemistry, 2015, 133, 144-152.	3.9	138
31	Autologous Bone Marrow Mononuclear Cells Exert Broad Effects on Short- and Long-Term Biological and Functional Outcomes in Rodents with Intracerebral Hemorrhage. Stem Cells and Development, 2015, 24, 2756-2766.	2.1	24
32	Dimethyl Fumarate Protects Brain From Damage Produced by Intracerebral Hemorrhage by Mechanism Involving Nrf2. Stroke, 2015, 46, 1923-1928.	2.0	108
33	Neuronal Interleukin-4 as a Modulator of Microglial Pathways and Ischemic Brain Damage. Journal of Neuroscience, 2015, 35, 11281-11291.	3.6	230
34	Pleiotropic Role of <scp>PPAR</scp> <i>γ</i> in Intracerebral Hemorrhage: An Intricate System Involving Nrf2, <scp>RXR</scp> , and <scp>NF</scp> â€ <i>κ</i> B. CNS Neuroscience and Therapeutics, 2015, 21, 357-366.	3.9	99
35	Polymorphonuclear Neutrophil in Brain Parenchyma After Experimental Intracerebral Hemorrhage. Translational Stroke Research, 2014, 5, 554-561.	4.2	53

36 The Role of PPARÎ<sup>3</sup> in Stroke. , 2014, , 301-320.

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37	Design of a Prospective, Dose-Escalation Study Evaluating the Safety of Pioglitazone for Hematoma Resolution in Intracerebral Hemorrhage (SHRINC). International Journal of Stroke, 2013, 8, 388-396.	5.9	65
38	Nrf2 to Pre-condition the Brain Against Injury Caused by Products of Hemolysis After ICH. Translational Stroke Research, 2013, 4, 71-75.	4.2	47
39	Intra-Arterial Delivery Is Not Superior to Intravenous Delivery of Autologous Bone Marrow Mononuclear Cells in Acute Ischemic Stroke. Stroke, 2013, 44, 3463-3472.	2.0	95
40	Proteasome Inhibitor Reduces Astrocytic iNOS Expression and Functional Deficit after Experimental Intracerebral Hemorrhage in Rats. Translational Stroke Research, 2012, 3, 146-153.	4.2	12
41	Molecular Pathophysiology of Cerebral Hemorrhage. Stroke, 2011, 42, 1781-1786.	2.0	662
42	Cytoprotective Role of Haptoglobin in Brain After Experimental Intracerebral Hemorrhage. Acta Neurochirurgica Supplementum, 2011, 111, 107-112.	1.0	20
43	Bone marrow mononuclear cells protect neurons and modulate microglia in cell culture models of ischemic stroke. Journal of Neuroscience Research, 2010, 88, 2869-2876.	2.9	59
44	In Vivo Therapeutic Gas Delivery for Neuroprotection With Echogenic Liposomes. Circulation, 2010, 122, 1578-1587.	1.6	65
45	Caffeinol at the Receptor Level. Stroke, 2010, 41, 363-367.	2.0	15
46	Hematoma Resolution as a Therapeutic Target. Stroke, 2009, 40, S92-4.	2.0	150
47	Neuronal PPARÎ <sup>3</sup> Deficiency Increases Susceptibility to Brain Damage after Cerebral Ischemia. Journal of Neuroscience, 2009, 29, 6186-6195.	3.6	148
48	Neuroprotective Role of Haptoglobin after Intracerebral Hemorrhage. Journal of Neuroscience, 2009, 29, 15819-15827.	3.6	136
49	Transcription Factor Nrf2 Protects the Brain From Damage Produced by Intracerebral Hemorrhage. Stroke, 2007, 38, 3280-3286.	2.0	202
50	Hematoma resolution as a target for intracerebral hemorrhage treatment: Role for peroxisome proliferatorâ€activated receptor γ in microglia/macrophages. Annals of Neurology, 2007, 61, 352-362.	5.3	319
51	Distinct patterns of intracerebral hemorrhage-induced alterations in NF-κB subunit, iNOS, and COX-2 expression. Journal of Neurochemistry, 2006, 101, 652-663.	3.9	113
52	15d-Prostaglandin J <sub>2</sub> Activates Peroxisome Proliferator-Activated Receptor-γ, Promotes Expression of Catalase, and Reduces Inflammation, Behavioral Dysfunction, and Neuronal Loss after Intracerebral Hemorrhage in Rats. Journal of Cerebral Blood Flow and Metabolism, 2006, 26, 811-820.	4.3	222
53	Peroxisome-proliferator-activated receptor-gamma (PPARγ) activation protects neurons from NMDA excitotoxicity. Brain Research, 2006, 1073-1074, 460-469.	2.2	80
54	Neuronal expression of peroxisome proliferator-activated receptor-gamma (PPARγ) and 15d-prostaglandin J2—Mediated protection of brain after experimental cerebral ischemia in rat. Brain Research, 2006, 1096, 196-203.	2.2	74

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55	New Horizons for Primary Intracerebral Hemorrhage Treatment: Experience From Preclinical Studies. Neurological Research, 2005, 27, 268-279.	1.3	260
56	Ethanol Plus Caffeine (Caffeinol) for Treatment of Ischemic Stroke. Stroke, 2003, 34, 1246-1251.	2.0	106
57	Cell death in experimental intracerebral hemorrhage: The "black hole―model of hemorrhagic damage. Annals of Neurology, 2002, 51, 517-524.	5.3	183
58	Early Exclusive Use of the Affected Forelimb After Moderate Transient Focal Ischemia in Rats. Stroke, 2000, 31, 1144-1152.	2.0	172
59	Interplay Between the Gamma Isoform of PKC and Calcineurin in Regulation of Vulnerability to Focal Cerebral Ischemia. Journal of Cerebral Blood Flow and Metabolism, 2000, 20, 343-349.	4.3	50
60	Nuclear Factor-κB and Cell Death After Experimental Intracerebral Hemorrhage in Rats. Stroke, 1999, 30, 2472-2478.	2.0	166
61	Neurofilament Proteolysis after Focal Ischemia; When Do Cells Die after Experimental Stroke?. Journal of Cerebral Blood Flow and Metabolism, 1999, 19, 652-660.	4.3	47
62	Reperfusion Injury: Demonstration of Brain Damage Produced by Reperfusion after Transient Focal Ischemia in Rats. Journal of Cerebral Blood Flow and Metabolism, 1997, 17, 1048-1056.	4.3	342
63	Citicoline for treatment of experimental focal ischemia: Histologic and behavioral outcome. Neurological Research, 1996, 18, 570-574.	1.3	63
64	Ischemia-Induced Neuronal Damage: A Role for Calcium/Calmodulin-Dependent Protein Kinase II. Journal of Cerebral Blood Flow and Metabolism, 1996, 16, 1-6.	4.3	97
65	An Alternative Method for the Quantitation of Neuronal Damage after Experimental Middle Cerebral Artery Occlusion in Rats: Analysis of Behavioral Deficit. Journal of Cerebral Blood Flow and Metabolism, 1996, 16, 705-713.	4.3	93