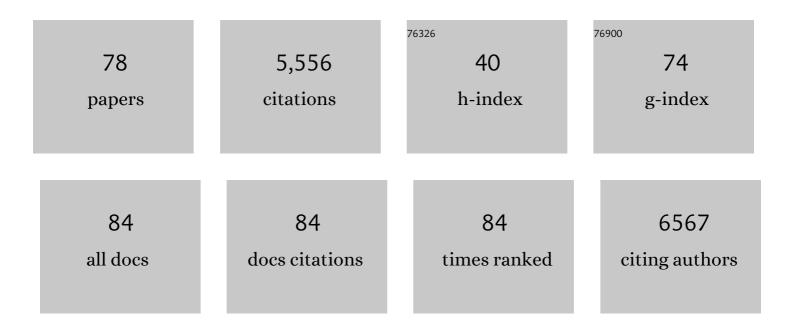
List of Publications by Year in descending order

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ALAIN RUISSON

#	Article	IF	CITATIONS
1	Astrocyte–neuron interplay is critical for Alzheimer's disease pathogenesis and is rescued by TRPA1 channel blockade. Brain, 2022, 145, 388-405.	7.6	41
2	Tubulin tyrosination regulates synaptic function and is disrupted in Alzheimer's disease. Brain, 2022, 145, 2486-2506.	7.6	17
3	Assembly of The Mitochondrial Complex I Assembly Complex Suggests a Regulatory Role for Deflavination. Angewandte Chemie - International Edition, 2021, 60, 4689-4697.	13.8	14
4	Pyr1-Mediated Pharmacological Inhibition of LIM Kinase Restores Synaptic Plasticity and Normal Behavior in a Mouse Model of Schizophrenia. Frontiers in Pharmacology, 2021, 12, 627995.	3.5	8
5	VEGF counteracts amyloid-Î ² -induced synaptic dysfunction. Cell Reports, 2021, 35, 109121.	6.4	19
6	Reduction in the neuronal surface of post and presynaptic GABA _B receptors in the hippocampus in a mouse model of Alzheimer's disease. Brain Pathology, 2020, 30, 554-575.	4.1	22
7	Density of GABAB Receptors Is Reduced in Granule Cells of the Hippocampus in a Mouse Model of Alzheimer's Disease. International Journal of Molecular Sciences, 2020, 21, 2459.	4.1	21
8	Effect of Al ² Oligomers on Neuronal APP Triggers a Vicious Cycle Leading to the Propagation of Synaptic Plasticity Alterations to Healthy Neurons. Journal of Neuroscience, 2020, 40, 5161-5176.	3.6	13
9	Improved optical slicing by stimulated emission depletion light sheet microscopy. Biomedical Optics Express, 2020, 11, 660.	2.9	7
10	Amyloid Fibers Reveal Themselves With Nearâ€Infrared. Movement Disorders, 2019, 34, 1643-1643.	3.9	0
11	Autophagy Is Required for Memory Formation and Reverses Age-Related Memory Decline. Current Biology, 2019, 29, 435-448.e8.	3.9	150
12	Synaptotoxicity in Alzheimer's Disease Involved a Dysregulation of Actin Cytoskeleton Dynamics through Cofilin 1 Phosphorylation. Journal of Neuroscience, 2018, 38, 10349-10361.	3.6	80
13	A key function for microtubule-associated-protein 6 in activity-dependent stabilisation of actin filaments in dendritic spines. Nature Communications, 2018, 9, 3775.	12.8	30
14	The amyloid-β oligomer Aβ*56 induces specific alterations in neuronal signaling that lead to tau phosphorylation and aggregation. Science Signaling, 2017, 10, .	3.6	90
15	GluN2B Subunit Labeling with Fluorescent Probes and High-Resolution Live Imaging. Methods in Molecular Biology, 2017, 1677, 171-183.	0.9	1
16	TRPA1 channels promote astrocytic Ca2+ hyperactivity and synaptic dysfunction mediated by oligomeric forms of amyloid-l ² peptide. Molecular Neurodegeneration, 2017, 12, 53.	10.8	62
17	Involvement of CRF2 signaling in enterocyte differentiation. World Journal of Gastroenterology, 2017, 23, 5127.	3.3	14
18	Specific alterations of tau phosphorylation and neuronal signaling induced by the amyloid-β oligomer Al²*56. Neurobiology of Aging, 2016, 39, S27.	3.1	0

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19	Disruption of dopaminergic transmission remodels tripartite synapse morphology and astrocytic calcium activity within substantia nigra pars reticulata. Glia, 2015, 63, 673-683.	4.9	40
20	Activity-Dependent Tau Protein Translocation to Excitatory Synapse Is Disrupted by Exposure to Amyloid-Beta Oligomers. Journal of Neuroscience, 2014, 34, 6084-6097.	3.6	207
21	Reciprocal disruption of neuronal signaling and $\hat{A^2}$ production mediated by extrasynaptic NMDA receptors: a downward spiral. Cell and Tissue Research, 2014, 356, 279-286.	2.9	40
22	Iron overload accelerates neuronal amyloid-β production and cognitive impairment in transgenic mice model of Alzheimer's disease. Neurobiology of Aging, 2014, 35, 2288-2301.	3.1	106
23	Oxygen glucose deprivation-induced astrocyte dysfunction provokes neuronal death through oxidative stress. Pharmacological Research, 2014, 87, 8-17.	7.1	36
24	NMDA receptor dysfunction contributes to impaired brainâ€derived neurotrophic factorâ€induced facilitation of hippocampal synaptic transmission in a <scp>T</scp> au transgenic model. Aging Cell, 2013, 12, 11-23.	6.7	64
25	Synthesis and in Vitro Characterisation of Ifenprodilâ€Based Fluorescein Conjugates as CluN1/CluN2B <i>N</i> â€Methylâ€ <scp>D</scp> â€aspartate Receptor Antagonists. ChemBioChem, 2013, 14, 759-769.	2.6	6
26	Confocal Microscopy Imaging of NR2B-Containing NMDA Receptors Based on Fluorescent Ifenprodil-Like Conjugates. Bioconjugate Chemistry, 2012, 23, 21-26.	3.6	18
27	Interaction Between ÂCaMKII and GluN2B Controls ERK-Dependent Plasticity. Journal of Neuroscience, 2012, 32, 10767-10779.	3.6	60
28	Ultra-sensitive molecular MRI of cerebrovascular cell activation enables early detection of chronic central nervous system disorders. NeuroImage, 2012, 63, 760-770.	4.2	64
29	Selective Impairment of Some Forms of Synaptic Plasticity by Oligomeric Amyloid-β Peptide in the Mouse Hippocampus: Implication of Extrasynaptic NMDA Receptors. Journal of Alzheimer's Disease, 2012, 32, 183-196.	2.6	37
30	O3-04-01: Amyloid-Beta oligomers accumulate in the postsynaptic density fraction and reveal cognitive impairment in transgenic mice model of Alzheimer's disease. , 2011, 7, S505-S505.		0
31	Synthesis, evaluation and metabolic studies of radiotracers containing a 4-(4-[18F]-fluorobenzyl)piperidin-1-yl moiety for the PET imaging of NR2B NMDA receptors. European Journal of Medicinal Chemistry, 2011, 46, 2295-2309.	5.5	29
32	Synapses, NMDA receptor activity and neuronal Aβ production in Alzheimer's disease. Reviews in the Neurosciences, 2011, 22, 285-294.	2.9	63
33	Activation of Extrasynaptic, But Not Synaptic, NMDA Receptors Modifies Amyloid Precursor Protein Expression Pattern and Increases Amyloid-1² Production. Journal of Neuroscience, 2010, 30, 15927-15942.	3.6	156
34	Copper-catalyzed amination of (bromophenyl)ethanolamine for a concise synthesis of aniline-containing analogues of NMDA NR2B antagonist ifenprodil. Organic and Biomolecular Chemistry, 2010, 8, 1111.	2.8	48
35	Reverse glial glutamate uptake triggers neuronal cell death through extrasynaptic NMDA receptor activation. Molecular and Cellular Neurosciences, 2009, 40, 463-473.	2.2	69
36	p3 peptide, a truncated form of Aβ devoid of synaptotoxic effect, does not assemble into soluble oligomers. FEBS Letters, 2008, 582, 1865-1870.	2.8	40

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37	Neuronal viability is controlled by a functional relation between synaptic and extrasynaptic NMDA receptors. FASEB Journal, 2008, 22, 4258-4271.	0.5	224
38	Comparison of the pharmacological properties of GK11 and MK801, two NMDA receptor antagonists: towards an explanation for the lack of intrinsic neurotoxicity of GK11. Journal of Neurochemistry, 2007, 103, 1682-1696.	3.9	10
39	P22 REDUCTION OF ISCHEMIC BRAIN DAMAGE BY NITROUS OXIDE AND XENON Behavioural Pharmacology, 2006, 17, 547.	1.7	0
40	NMDA Receptor Activation Inhibits α-Secretase and Promotes Neuronal Amyloid-β Production. Journal of Neuroscience, 2005, 25, 9367-9377.	3.6	178
41	Akt-dependent Expression of NAIP-1 Protects Neurons against Amyloid-β Toxicity. Journal of Biological Chemistry, 2005, 280, 24941-24947.	3.4	51
42	Differential role of synaptic an extrasynaptic NMDA receptors in glutamate mediated neuronal injury. Journal of Cerebral Blood Flow and Metabolism, 2005, 25, S445-S445.	4.3	0
43	2,7-Bis-(4-Amidinobenzylidene)-Cycloheptan-1-One Dihydrochloride, tPA Stop, Prevents tPA-Enhanced Excitotoxicity Both In Vitro and In Vivo. Journal of Cerebral Blood Flow and Metabolism, 2004, 24, 1153-1159.	4.3	20
44	Neurotrophin-3-induced PI-3 kinase/Akt signaling rescues cortical neurons from apoptosis. Experimental Neurology, 2004, 187, 38-46.	4.1	50
45	Is tissue-type plasminogen activator a neuromodulator?. Molecular and Cellular Neurosciences, 2004, 25, 594-601.	2.2	65
46	Neuroprotection by Nitrous Oxide and Xenon and Its Relation to Minimum Alveolar Concentration. Anesthesiology, 2004, 101, 260-261.	2.5	14
47	Transforming growth factor-beta and ischemic brain injury. Cellular and Molecular Neurobiology, 2003, 23, 539-550.	3.3	90
48	Reduction of Ischemic Brain Damage by Nitrous Oxide and Xenon. Journal of Cerebral Blood Flow and Metabolism, 2003, 23, 1168-1173.	4.3	127
49	Tissue plasminogen activator and NMDA receptor cleavage. Nature Medicine, 2003, 9, 371-372.	30.7	79
50	Reply to "Tissue plasminogen activator and NMDA receptor cleavage". Nature Medicine, 2003, 9, 372-373.	30.7	18
51	Transforming growth factor αâ€induced expression of typeâ€1 plasminogen activator inhibitor in astrocytes rescues neurons from excitotoxicity. FASEB Journal, 2003, 17, 277-279.	0.5	48
52	Transforming Growth Factor-β1 Potentiates Amyloid-β Generation in Astrocytes and in Transgenic Mice. Journal of Biological Chemistry, 2003, 278, 18408-18418.	3.4	127
53	Le tranforming growth factor-β (TGF-p) a t-il un rÃ1e neuroprotecteur dans l'ischémie cérébrale ?. Société De Biologie Journal, 2003, 197, 145-150.	0.3	3
54	Smad3-Dependent Induction of Plasminogen Activator Inhibitor-1 in Astrocytes Mediates Neuroprotective Activity of Transforming Growth Factor-β1 against NMDA-Induced Necrosis. Molecular and Cellular Neurosciences, 2002, 21, 634-644.	2.2	77

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55	Transforming Growth Factor-β1—Modulated Cerebral Gene Expression. Journal of Cerebral Blood Flow and Metabolism, 2002, 22, 1114-1123.	4.3	24
56	Matching Gene Expression with Hypometabolism after Cerebral Ischemia in the Nonhuman Primate. Journal of Cerebral Blood Flow and Metabolism, 2002, 22, 1165-1169.	4.3	7
57	Matching Gene Expression With Hypometabolism After Cerebral Ischemia in the Nonhuman Primate. Journal of Cerebral Blood Flow and Metabolism, 2002, , 1165-1169.	4.3	2
58	Neuroprotection Mediated by Glial Cell Line-Derived Neurotrophic Factor: Involvement of a Reduction of NMDA-Induced Calcium Influx by the Mitogen-Activated Protein Kinase Pathway. Journal of Neuroscience, 2001, 21, 3024-3033.	3.6	182
59	Complement anaphylatoxin C3a is selectively protective against NMDA-induced neuronal cell death. NeuroReport, 2001, 12, 289-293.	1.2	103
60	The proteolytic activity of tissue-plasminogen activator enhances NMDA receptor-mediated signaling. Nature Medicine, 2001, 7, 59-64.	30.7	678
61	Increased Expression of Transforming Growth Factor-β after Cerebral Ischemia in the Baboon: An Endogenous Marker of Neuronal Stress?. Journal of Cerebral Blood Flow and Metabolism, 2001, 21, 820-827.	4.3	37
62	A Soluble Transforming Growth Factor-β (TGF-β) Type I Receptor Mimics TGF-β Responses. Journal of Biological Chemistry, 2001, 276, 46243-46250.	3.4	13
63	Serine Protease Inhibitors: Novel Therapeutic Targets for Stroke?. Journal of Cerebral Blood Flow and Metabolism, 2000, 20, 755-764.	4.3	91
64	lschemia-Induced Interleukin-6 as a Potential Endogenous Neuroprotective Cytokine against NMDA Receptor-Mediated Excitoxicity in the Brain. Journal of Cerebral Blood Flow and Metabolism, 2000, 20, 956-966.	4.3	176
65	Transforming growth factorâ€Î²l as a regulator of the serpins/tâ€PA axis in cerebral ischemia. FASEB Journal, 1999, 13, 1315-1324.	0.5	96
66	A Transforming Growth Factor-Î ² Antagonist Unmasks the Neuroprotective Role of This Endogenous Cytokine in Excitotoxic and Ischemic Brain Injury. Journal of Cerebral Blood Flow and Metabolism, 1999, 19, 1345-1353.	4.3	121
67	Upâ€regulation of a serine protease inhibitor in astrocytes mediates the neuroprotective activity of transforming growth factor β1. FASEB Journal, 1998, 12, 1683-1691.	0.5	115
68	Evidence of Type I and Type II Transforming Growth Factorâ€Ĵ² Receptors in Central Nervous Tissues: Changes Induced by Focal Cerebral Ischemia. Journal of Neurochemistry, 1998, 70, 2296-2304.	3.9	61
69	Membraneâ€delimited modulation of NMDA currents by metabotropic glutamate receptor subtypes 1/5 in cultured mouse cortical neurons Journal of Physiology, 1997, 499, 721-732.	2.9	76
70	DCC-IV Selectively Attenuates Rapidly Triggered NMDA-induced Neurotoxicity in Cortical Neurons. European Journal of Neuroscience, 1996, 8, 138-143.	2.6	69
71	The inhibitory mGluR agonist, s-4-carboxy-3-hydroxy-phenylglycine selectively attenuates NMDA neurotoxicity and oxygen-glucose deprivation-induced neuronal death. Neuropharmacology, 1995, 34, 1081-1087.	4.1	157
72	Nitric Oxide and Cerebral Ischemia. Annals of the New York Academy of Sciences, 1994, 738, 341-347.	3.8	8

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73	Mechanisms Involved in the Neuroprotective Activity of a Nitric Oxide Synthase Inhibitor During Focal Cerebral Ischemia. Journal of Neurochemistry, 1993, 61, 690-696.	3.9	156
74	The neuroprotective effect of a nitric oxide inhibitor in a rat model of focal cerebral ischaemia. British Journal of Pharmacology, 1992, 106, 766-767.	5.4	221
75	Striatal Protection Induced by Lesioning the Substantia Nigra of Rats Subjected to Focal Ischemia. Journal of Neurochemistry, 1992, 59, 1153-1157.	3.9	90
76	Nigrostriatal pathway modulates striatum vulnerability to quinolinic acid. Neuroscience Letters, 1991, 131, 257-259.	2.1	33
77	Lesioning the substantia nigra reduces striatal infarct volume following focal ischemia in rats. Fundamental and Clinical Pharmacology, 1991, 5, 645-647.	1.9	5
78	Combination of horseradish peroxidase and lucifer yellow staining for selective labeling of neurons at the electron microscopic level Journal of Histochemistry and Cytochemistry, 1991, 39, 1579-1583.	2.5	3