Osvaldo Daniel Uchitel

List of Publications by Year in descending order

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94 papers 3,303 citations

126858 33 h-index 54 g-index

95 all docs 95 docs citations 95 times ranked 2360 citing authors

#	Article	IF	CITATIONS
1	Dynamic Distribution of ASIC1a Channels and Other Proteins within Cells Detected through Fractionation. Membranes, 2022, 12, 389.	1.4	1
2	Ion channels and pain in Fabry disease. Molecular Pain, 2021, 17, 174480692110331.	1.0	7
3	Histamine and Corticosterone Modulate Acid Sensing Ion Channels (ASICs) Dependent Long-term Potentiation at the Mouse Anterior Cingulate Cortex. Neuroscience, 2021, 460, 145-160.	1.1	9
4	Evaluation of early microstructural changes in the R6/1 mouse model of Huntington's disease by ultra-high field diffusion MR imaging. Neurobiology of Aging, 2021, 102, 32-49.	1.5	15
5	Efficacy of a Nasal Spray Containing lota-Carrageenan in the Postexposure Prophylaxis of COVID-19 in Hospital Personnel Dedicated to Patients Care with COVID-19 Disease. International Journal of General Medicine, 2021, Volume 14, 6277-6286.	0.8	43
6	Signaling Pathways in Proton and Non-proton ASIC1a Activation. Frontiers in Cellular Neuroscience, 2021, 15, 735414.	1.8	4
7	Modulation of acid sensing ion channel dependent protonergic neurotransmission at the mouse calyx of Held. Neuroscience, 2020, 439, 195-210.	1.1	14
8	Upregulation of ASIC1a channels in an in vitro model of Fabry disease. Neurochemistry International, 2020, 140, 104824.	1.9	8
9	A new tool to sense pH changes at the neuromuscular junction synaptic cleft. Scientific Reports, 2020, 10, 20480.	1.6	6
10	Assessing neuraxial microstructural changes in a transgenic mouse model of early stage Amyotrophic Lateral Sclerosis by ultraâ∈high field MRI and diffusion tensor metrics. Animal Models and Experimental Medicine, 2020, 3, 117-129.	1.3	4
11	Synaptic signals mediated by protons and acidâ€sensing ion channels. Synapse, 2019, 73, e22120.	0.6	27
12	Unveiling early cortical and subcortical neuronal degeneration in ALS mice by ultra-high field diffusion MRI. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2019, 20, 549-561.	1.1	25
13	Acid-Sensing Ion Channels Activated by Evoked Released Protons Modulate Synaptic Transmission at the Mouse Calyx of Held Synapse. Journal of Neuroscience, 2017, 37, 2589-2599.	1.7	76
14	ASIC channel inhibition enhances excitotoxic neuronal death in an in vitro model of spinal cord injury. Neuroscience, 2017, 343, 398-410.	1.1	24
15	Carbonic anhydrase inhibitor acetazolamide shifts synaptic vesicle recycling to a fast mode at the mouse neuromuscular junction. Synapse, 2017, 71, e22009.	0.6	13
16	Acetazolamide potentiates the afferent drive to prefrontal cortex inÂvivo. Physiological Reports, 2017, 5, e13066.	0.7	9
17	Chronic pregabalin treatment decreases excitability of dentate gyrus and accelerates maturation of adultâ€born granule cells. Journal of Neurochemistry, 2017, 140, 257-267.	2.1	8
18	Acoustic trauma slows AMPA receptorâ€mediated EPSCs in the auditory brainstem, reducing GluA4 subunit expression as a mechanism to rescue binaural function. Journal of Physiology, 2016, 594, 3683-3703.	1.3	28

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19	Analysis of C9orf72 in patients with frontotemporal dementia and amyotrophic lateral sclerosis from Argentina. Neurobiology of Aging, 2016, 40, 192.e13-192.e15.	1.5	18
20	Familial hemiplegic migraine type-1 mutated cav2.1 calcium channels alter inhibitory and excitatory synaptic transmission in the lateral superior olive of mice. Hearing Research, 2015, 319, 56-68.	0.9	6
21	Synaptic Gain-of-Function Effects of Mutant Ca _v 2.1 Channels in a Mouse Model of Familial Hemiplegic Migraine Are Due to Increased Basal [Ca ²⁺] _i . Journal of Neuroscience, 2014, 34, 7047-7058.	1.7	45
22	Calcium channels and synaptic transmission in familial hemiplegic migraine type 1 animal models. Biophysical Reviews, 2014, 6, 15-26.	1.5	4
23	Acid-sensing ion channels 1a (ASIC1a) inhibit neuromuscular transmission in female mice. American Journal of Physiology - Cell Physiology, 2014, 306, C396-C406.	2.1	19
24	Acute effects of pregabalin on the function and cellular distribution of CaV2.1 in HEK293t cells. Brain Research Bulletin, 2013, 90, 107-113.	1.4	10
25	CaV2.1 (P/Q) Voltage Activated Ca2+ Channels and Synaptic Transmission in Genetic and Autoimmune Diseases., 2013,, 263-288.		O
26	Presynaptic Ca $<$ sub $>$ V $<$ /sub $>$ 2.1 calcium channels carrying familial hemiplegic migraine mutation R192Q allow faster recovery from synaptic depression in mouse calyx of Held. Journal of Neurophysiology, 2012, 108, 2967-2976.	0.9	21
27	Unequal gains of function are a headache for migraine mechanisms. Journal of Physiology, 2012, 590, 1-2.	1.3	1
28	CaV2.1 voltage activated calcium channels and synaptic transmission in familial hemiplegic migraine pathogenesis. Journal of Physiology (Paris), 2012, 106, 12-22.	2.1	14
29	P/Q-type calcium channel ablation in a mice glycinergic synapse mediated by multiple types of Ca2+ channels alters transmitter release and short term plasticity. Neuroscience, 2011, 192, 219-230.	1.1	18
30	Autoimmunity in Amyotrophic Lateral Sclerosis: Past and Present. Neurology Research International, 2011, 2011, 1-11.	0.5	55
31	Amyotrophic lateral sclerosisâ€immunoglobulins selectively interact with neuromuscular junctions expressing P/Qâ€type calcium channels. Journal of Neurochemistry, 2011, 119, 826-838.	2.1	19
32	Pregabalin Modulation of Neurotransmitter Release Is Mediated by Change in Intrinsic Activation/Inactivation Properties of Cav2.1 Calcium Channels. Journal of Pharmacology and Experimental Therapeutics, 2011, 336, 973-982.	1.3	28
33	Effects of T-type calcium channel blockers on cocaine-induced hyperlocomotion and thalamocortical GABAergic abnormalities in mice. Psychopharmacology, 2010, 212, 205-214.	1.5	25
34	Adenosine drives recycled vesicles to a slowâ€release pool at the mouse neuromuscular junction. European Journal of Neuroscience, 2010, 32, 985-996.	1.2	9
35	Acute modulation of calcium currents and synaptic transmission by gabapentinoids. Channels, 2010, 4, 490-496.	1.5	42
36	Gain of Function in FHM-1 Cav2.1 Knock-In Mice Is Related to the Shape of the Action Potential. Journal of Neurophysiology, 2010, 104, 291-299.	0.9	33

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37	Lateral olivocochlear (LOC) neurons of the mouse LSO receive excitatory and inhibitory synaptic inputs with slower kinetics than LSO principal neurons. Hearing Research, 2010, 270, 119-126.	0.9	51
38	Corrigendum to "Calcium channels, neuromuscular synaptic transmission and neurological diseases― [J. Neuroimmunol. 201–202, 136–144, 2008]. Journal of Neuroimmunology, 2009, 207, 123.	1.1	O
39	Cocaine Acute "Binge―Administration Results in Altered Thalamocortical Interactions in Mice. Biological Psychiatry, 2009, 66, 769-776.	0.7	28
40	Altered synaptic synchrony in motor nerve terminals lacking P/Q alcium channels. Synapse, 2008, 62, 466-471.	0.6	19
41	Lâ€type calcium channels are involved in fast endocytosis at the mouse neuromuscular junction. European Journal of Neuroscience, 2008, 27, 1333-1344.	1.2	41
42	P/Q Ca2+ channels are functionally coupled to exocytosis of the immediately releasable pool in mouse chromaffin cells. Cell Calcium, 2008, 43, 155-164.	1.1	31
43	Calcium channels, neuromuscular synaptic transmission and neurological diseases. Journal of Neuroimmunology, 2008, 201-202, 136-144.	1.1	17
44	Changes in synaptic transmission properties due to the expression of Nâ€type calcium channels at the calyx of Held synapse of mice lacking P/Qâ€type calcium channels. Journal of Physiology, 2007, 584, 835-851.	1.3	52
45	Calcium Signaling Pathways Mediating Synaptic Potentiation Triggered by Amyotrophic Lateral Sclerosis IgG in Motor Nerve Terminals. Journal of Neuroscience, 2006, 26, 2661-2672.	1.7	53
46	Testosterone modulates Cav2.2 calcium channels' functional expression at rat levator ani neuromuscular junction. Neuroscience, 2005, 134, 817-826.	1.1	16
47	Differential Expression of Ca Channels and Synaptic Transmission in Normal and Ataxic Knock-Out Mice. , 2005, , 73-78.		O
48	Functional Compensation of P/Q by N-Type Channels Blocks Short-Term Plasticity at the Calyx of Held Presynaptic Terminal. Journal of Neuroscience, 2004, 24, 10379-10383.	1.7	134
49	Muscarinic autoreceptors related with calcium channels in the strong and weak inputs at polyinnervated developing rat neuromuscular junctions. Neuroscience, 2004, 123, 61-73.	1.1	42
50	Differential expression of $\hat{l}\pm 1$ and \hat{l}^2 subunits of voltage dependent Ca2+ channel at the neuromuscular junction of normal and p/q Ca2+ channel knockout mouse. Neuroscience, 2004, 123, 75-85.	1.1	58
51	Ca2+Channels and Synaptic Transmission at the Adult, Neonatal, and P/Q-Type Deficient Neuromuscular Junction. Annals of the New York Academy of Sciences, 2003, 998, 11-17.	1.8	37
52	Modulation of ACh release by presynaptic muscarinic autoreceptors in the neuromuscular junction of the newborn and adult rat. European Journal of Neuroscience, 2003, 17, 119-127.	1.2	74
53	Nifedipine-Mediated Mobilization of Intracellular Calcium Stores Increases Spontaneous Neurotransmitter Release at Neonatal Rat Motor Nerve Terminals. Journal of Pharmacology and Experimental Therapeutics, 2003, 306, 658-663.	1.3	14
54	Altered properties of quantal neurotransmitter release at endplates of mice lacking P/Q-type Ca2+ channels. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 3491-3496.	3.3	120

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55	Chapter 5 Calcium channelopathies in neuromuscular transmission. Supplements To Clinical Neurophysiology, 2002, 54, 49-52.	2.1	O
56	Calcium channels involved in neurotransmitter release at adult, neonatal and P/Q-type deficient neuromuscular junctions (Review). Molecular Membrane Biology, 2002, 19, 293-300.	2.0	46
57	Decreased calcium influx into the neonatal rat motor nerve terminals can recruit additional neuromuscular junctions during the synapse elimination period. Neuroscience, 2002, 110, 147-154.	1.1	26
58	Differential Ca2+-dependence of transmitter release mediated by P/Q- and N-type calcium channels at neuromuscular junctions. European Journal of Neuroscience, 2002, 15, 1874-1880.	1.2	36
59	The effect of buffered calcium diffusion on neurotransmitter release. Physica D: Nonlinear Phenomena, 2002, 168-169, 356-364.	1.3	2
60	Calcium channels coupled to neurotransmitter release at dually innervated neuromuscular junctions in the newborn rat. Neuroscience, 2001, 102, 697-708.	1.1	51
61	Coupling of L-type calcium channels to neurotransmitter release at mouse motor nerve terminals. Pflugers Archiv European Journal of Physiology, 2001, 441, 824-831.	1.3	49
62	Amyotrophic lateral sclerosis IgG-treated neuromuscular junctions develop sensitivity to L-type calcium channel blocker., 2000, 23, 543-550.		31
63	Developmental Changes in Calcium Channel Types Mediating Central Synaptic Transmission. Journal of Neuroscience, 2000, 20, 59-65.	1.7	270
64	Reduced facilitation and vesicular uptake in crustacean and mammalian neuromuscular junction by T-588, a neuroprotective compound. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 14588-14593.	3.3	13
65	Calcium channels coupled to neurotransmitter release at neonatal rat neuromuscular junctions. Journal of Physiology, 1999, 514, 533-540.	1.3	102
66	L-Type calcium channels unmasked by cell-permeant Ca 2+ buffer at mouse motor nerve terminals. Pflugers Archiv European Journal of Physiology, 1999, 437, 523-528.	1.3	34
67	Toxins affecting calcium channels in neurons. Toxicon, 1997, 35, 1161-1191.	0.8	64
68	Effects of Ca2+ channel blocker neurotoxins on transmitter release and presynaptic currents at the mouse neuromuscular junction. British Journal of Pharmacology, 1997, 121, 1531-1540.	2.7	51
69	Evaluation of antioxidants, protein, and lipid oxidation products in blood from sporadic amyotrophic lateral sclerosis patients. Neurochemical Research, 1997, 22, 535-539.	1.6	80
70	P/Q-type calcium channels activate neighboring calcium-dependent potassium channels in mouse motor nerve terminals. Pflugers Archiv European Journal of Physiology, 1997, 434, 406-412.	1.3	26
71	Calcium Channel Diversity at the Vertebrate Neuromuscular Junction. , 1997, , 37-46.		O
72	Uptake of immunoglobulin G from amyotrophic lateral sclerosis patients by motor nerve terminals in mice. Journal of the Neurological Sciences, 1996, 137, 97-102.	0.3	12

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73	Different calcium channels mediate transmitter release evoked by transient or sustained depolarization at mammalian symphatetic ganglia. Neuroscience, 1995, 64, 117-123.	1.1	40
74	Pharmacological Characterization of the Voltageâ€Dependent Ca ²⁺ Channels Present in Synaptosomes from Rat and Chicken Central Nervous System. Journal of Neurochemistry, 1995, 64, 2544-2551.	2.1	33
75	Potassium channels from normal and denervated mouse skeletal muscle fibers. Muscle and Nerve, 1993, 16, 579-586.	1.0	10
76	Congenital myasthenic syndromes: II. Syndrome attributed to abnormal interaction of acetylcholine with its receptor. Muscle and Nerve, 1993, 16, 1293-1301.	1.0	68
77	Newly recognized congenital myasthenic syndrome associated with high conductance and fast closure of the acetylcholine receptor channel. Annals of Neurology, 1993, 34, 38-47.	2.8	39
78	Mammalian Neuromuscular Transmission Blocked by Funnel Web Toxin. Annals of the New York Academy of Sciences, 1993, 681, 405-407.	1.8	18
79	Congenital Myasthenic Syndrome Attributed to an Abnormal Interaction of Acetylcholine with Its Receptor. Annals of the New York Academy of Sciences, 1993, 681, 487-495.	1.8	7
80	Transmitter release and presynaptic Ca2+ currents blocked by the spider toxin ω-Aga-IVA. NeuroReport, 1993, 5, 333-336.	0.6	114
81	P-type voltage-dependent calcium channel mediates presynaptic calcium influx and transmitter release in mammalian synapses Proceedings of the National Academy of Sciences of the United States of America, 1992, 89, 3330-3333.	3.3	369
82	Effect of I‰-conotoxin GVIA on neurotransmitter release at the mouse neuromuscular junction. Brain Research, 1991, 557, 336-339.	1.1	51
83	Chapter 14 Newly recognized congenital myasthenic syndromes: I. Congenital paucity of synaptic vesicles and reduced quantal release. Progress in Brain Research, 1990, , 125-137.	0.9	36
84	Ca-dependent slow action potentials in neuromuscular diseases. Journal of Cellular Physiology, 1990, 143, 590-595.	2.0	0
85	Ca2+ role on the effect of phorbol esters on the spontaneous quantal release of neurotransmitter at the mouse neuromuscular junction. Brain Research, 1990, 525, 280-284.	1.1	2
86	Neuronal control of extrajunctional acetylcholine receptor-channels induced by injury in frog skeletal muscle fibres. Pflugers Archiv European Journal of Physiology, 1989, 414, 113-117.	1.3	1
87	Electrical properties of normal, denervated and organ-cultured slow fibres of toad cruralis muscles. Pflugers Archiv European Journal of Physiology, 1989, 414, 584-588.	1.3	2
88	Ca-dependent slow action potentials in human skeletal muscle. Journal of Cellular Physiology, 1988, 137, 448-454.	2.0	2
89	Immunoglobulins from amyotrophic lateral sclerosis patients enhance spontaneous transmitter release from motor-nerve terminals Proceedings of the National Academy of Sciences of the United States of America, 1988, 85, 7371-7374.	3.3	55
90	Electrophysiologic denervation changes of human muscle fibers in motoneuron diseases. Muscle and Nerve, 1986, 9, 748-755.	1.0	9

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91	On the appearance of acetylcholine receptors in denervated rat diaphragm, and its dependence on nerve stump length. Brain Research, 1978, 153, 539-548.	1.1	25
92	Nonacceptance of innervation by innervated neonatal rat muscle. Developmental Biology, 1977, 61, 166-176.	0.9	10
93	Potassium and calcium conductance in slow muscle fibres of the toad Journal of Physiology, 1976, 255, 435-448.	1.3	17
94	Reversible inhibition of potassium contractures by optical isomers of verapamil and D 600 on slow muscle fibres of the frog. Naunyn-Schmiedeberg's Archives of Pharmacology, 1976, 292, 21-27.	1.4	27