Mark Estacion

List of Publications by Year
in descending order

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70 papers	4,347 citations	101543 36 h-index	114465 63 g-index
72 all docs	72 docs citations	72 times ranked	3949 citing authors

#	Article	IF	Citations
1	Inhibition of sodium conductance by cannabigerol contributes to a reduction of dorsal root ganglion neuron excitability. British Journal of Pharmacology, 2022, 179, 4010-4030.	5.4	16
2	Depolarizing Na_V and Hyperpolarizing K_V Channels Are Co-Trafficked in Sensory Neurons. Journal of Neuroscience, 2022, 42, 4794-4811.	3.6	6
3	KCNQ variants and pain modulation: a missense variant in Kv7.3 contributes to pain resilience. Brain Communications, 2021, 3, fcab212.	3.3	13
4	Two independent mouse lines carrying the Nav1.7 I228M gain-of-function variant display dorsal root ganglion neuron hyperexcitability but a minimal pain phenotype. Pain, 2021, 162, 1758-1770.	4.2	9
5	Lacosamide Inhibition of NaV1.7 Channels Depends on its Interaction With the Voltage Sensor Domain and the Channel Pore. Frontiers in Pharmacology, 2021, 12, 791740.	3.5	5
6	Contributions of NaV1.8 and NaV1.9 to excitability in human induced pluripotent stem-cell derived somatosensory neurons. Scientific Reports, 2021, 11, 24283.	3.3	6
7	Dexpramipexole blocks Nav1.8 sodium channels and provides analgesia in multiple nociceptive and neuropathic pain models. Pain, 2020, 161, 831-841.	4.2	22
8	A 49-residue sequence motif in the C terminus of Nav1.9 regulates trafficking of the channel to the plasma membrane. Journal of Biological Chemistry, 2020, 295, 1077-1090.	3.4	8
9	Differential effect of lacosamide on Nav1.7 variants from responsive and non-responsive patients with small fibre neuropathy. Brain, 2020, 143, 771-782.	7.6	31
10	A 49-residue sequence motif in the C terminus of Nav1.9 regulates trafficking of the channel to the plasma membrane. Journal of Biological Chemistry, 2020, 295, 1077-1090.	3.4	6
11	A Novel Gain-of-Function Nav1.9 Mutation in a Child With Episodic Pain. Frontiers in Neuroscience, 2019, 13, 918.	2.8	18
12	A gain-of-function sodium channelÎ²2-subunit mutation in painful diabetic neuropathy. Molecular Pain, 2019, 15, 174480691984980.	2.1	38
13	Atypical changes in DRG neuron excitability and complex pain phenotype associated with a Nav1.7 mutation that massively hyperpolarizes activation. Scientific Reports, 2018, 8, 1811.	3.3	14
14	Na V 1.7 as a Pharmacogenomic Target for Pain: Moving Toward Precision Medicine. Trends in Pharmacological Sciences, 2018, 39, 258-275.	8.7	54
15	A novel gain-of-function Na_v1.7 mutation in a carbamazepine-responsive patient with adult-onset painful peripheral neuropathy. Molecular Pain, 2018, 14, 174480691881500.	2.1	7
16	The Novel Activity of Carbamazepine as an Activation Modulator Extends from Na_V1.7 Mutations to the Na_V1.8-S242T Mutant Channel from a Patient with Painful Diabetic Neuropathy. Molecular Pharmacology, 2018, 94, 1256-1269.	2.3	24
17	Safety and efficacy of a Nav1.7 selective sodium channel blocker in patients with trigeminal neuralgia: a double-blind, placebo-controlled, randomised withdrawal phase 2a trial. Lancet Neurology, The, 2017, 16, 291-300.	10.2	144
18	Nonlinear effects of hyperpolarizing shifts in activation of mutant Na_v1.7 channels on resting membrane potential. Journal of Neurophysiology, 2017, 117, 1702-1712.	1.8	6

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19	Subtype-Selective Small Molecule Inhibitors Reveal a Fundamental Role for Nav1.7 in Nociceptor Electrogenesis, Axonal Conduction and Presynaptic Release. PLoS ONE, 2016, 11, e0152405.	2.5	152
20	Pharmacotherapy for Pain in a Family With Inherited Erythromelalgia Guided by Genomic Analysis and Functional Profiling. JAMA Neurology, 2016, 73, 659.	9.0	70
21	Sodium Channels, Mitochondria, and Axonal Degeneration in Peripheral Neuropathy. Trends in Molecular Medicine, 2016, 22, 377-390.	6.7	46
22	Nav1.7-A1632G Mutation from a Family with Inherited Erythromelalgia: Enhanced Firing of Dorsal Root Ganglia Neurons Evoked by Thermal Stimuli. Journal of Neuroscience, 2016, 36, 7511-7522.	3.6	61
23	Ca²⁺ toxicity due to reverse Na⁺/Ca²⁺ exchange contributes to degeneration of neurites of DRG neurons induced by a neuropathy-associated Nav1.7 mutation. Journal of Neurophysiology, 2015, 114, 1554-1564.	1.8	41
24	Human Na_v1.8: enhanced persistent and ramp currents contribute to distinct firing properties of human DRG neurons. Journal of Neurophysiology, 2015, 113, 3172-3185.	1.8	89
25	Contribution of sodium channels to lamellipodial protrusion and Rac1 and ERK1/2 activation in ATPâ€stimulated microglia. Glia, 2014, 62, 2080-2095.	4.9	30
26	Gain-of-function mutations in sodium channel NaV1.9 in painful neuropathy. Brain, 2014, 137, 1627-1642.	7.6	242
27	Characterization of a de novo SCN8A mutation in a patient with epileptic encephalopathy. Epilepsy Research, 2014, 108, 1511-1518.	1.6	92
28	A novel de novo mutation of SCN8A (Nav1.6) with enhanced channel activation in a child with epileptic encephalopathy. Neurobiology of Disease, 2014, 69, 117-123.	4.4	96
29	Sodium Channels Contribute to Degeneration of Dorsal Root Ganglion Neurites Induced by Mitochondrial Dysfunction in anIn VitroModel of Axonal Injury. Journal of Neuroscience, 2013, 33, 19250-19261.	3.6	61
30	Differential effect of D623N variant and wild-type Nav1.7 sodium channels on resting potential and interspike membrane potential of dorsal root ganglion neurons. Brain Research, 2013, 1529, 165-177.	2.2	14
31	A new Nav1.7 mutation in an erythromelalgia patient. Biochemical and Biophysical Research Communications, 2013, 432, 99-104.	2.1	21
32	The response of Na_V1.3 sodium channels to ramp stimuli: multiple components and mechanisms. Journal of Neurophysiology, 2013, 109, 306-314.	1.8	36
33	Molecular Architecture of a Sodium Channel S6 Helix. Journal of Biological Chemistry, 2013, 288, 13741-13747.	3.4	21
34	Structural modelling and mutant cycle analysis predict pharmacoresponsiveness of a Nav1.7 mutant channel. Nature Communications, 2012, 3, 1186.	12.8	88
35	Gain of function Na_V1.7 mutations in idiopathic small fiber neuropathy. Annals of Neurology, 2012, 71, 26-39.	5.3	518
36	Intra- and Interfamily Phenotypic Diversity in Pain Syndromes Associated with a Gain-of-Function Variant of Na_V1.7. Molecular Pain, 2011, 7, 1744-8069-7-92.	2.1	94

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37	Effects of Ranolazine on Wild-Type and Mutant hNa_v1.7 Channels and on DRG Neuron Excitability. Molecular Pain, 2010, 6, 1744-8069-6-35.	2.1	30
38	Can robots patch-clamp as well as humans? Characterization of a novel sodium channel mutation. Journal of Physiology, 2010, 588, 1915-1927.	2.9	33
39	A sodium channel mutation linked to epilepsy increases ramp and persistent current of Nav1.3 and induces hyperexcitability in hippocampal neurons. Experimental Neurology, 2010, 224, 362-368.	4.1	80
40	Maitotoxin converts the plasmalemmal Ca²⁺pump into a Ca²⁺-permeable nonselective cation channel. American Journal of Physiology - Cell Physiology, 2009, 297, C1533-C1543.	4.6	31
41	A novel Na_v1.7 mutation producing carbamazepineâ€responsive erythromelalgia. Annals of Neurology, 2009, 65, 733-741.	5.3	132
42	A sodium channel gene SCN9A polymorphism that increases nociceptor excitability. Annals of Neurology, 2009, 66, 862-866.	5.3	91
43	Voltage-clamp and current-clamp recordings from mammalian DRG neurons. Nature Protocols, 2009, 4, 1103-1112.	12.0	94
44	NaV1.7 Gain-of-function Mutations As A Continuum: A1632E Displays Physiological Changes Associated With Erythromelalgia And Paroxysmal Extreme Pain Disorder Mutations And Produces Symptoms Of Both Disorders. Biophysical Journal, 2009, 96, 12a.	0.5	4
45	Paroxysmal Extreme Pain Disorder M1627K Mutation in Human Na_v1.7 Renders DRG Neurons Hyperexcitable. Molecular Pain, 2008, 4, 1744-8069-4-37.	2.1	112
46	Na_V1.7 Gain-of-Function Mutations as a Continuum: A1632E Displays Physiological Changes Associated with Erythromelalgia and Paroxysmal Extreme Pain Disorder Mutations and Produces Symptoms of Both Disorders. Journal of Neuroscience, 2008, 28, 11079-11088.	3.6	148
47	Palytoxin-induced cell death cascade in bovine aortic endothelial cells. American Journal of Physiology - Cell Physiology, 2006, 291, C657-C667.	4.6	37
48	Human TRPC6 expressed in HEK 293 cells forms non-selective cation channels with limited Ca2+permeability. Journal of Physiology, 2006, 572, 359-377.	2.9	108
49	Identification and localization of TRPC channels in the rat kidney. American Journal of Physiology - Renal Physiology, 2006, 290, F1241-F1252.	2.7	122
50	Activation of Human TRPC6 Channels by Receptor Stimulation. Journal of Biological Chemistry, 2004, 279, 22047-22056.	3.4	84
51	Association of Immunophilins with Mammalian TRPC Channels. Journal of Biological Chemistry, 2004, 279, 34521-34529.	3.4	90
52	Maitotoxin-induced cell death cascade in bovine aortic endothelial cells: divalent cation specificity and selectivity. American Journal of Physiology - Cell Physiology, 2004, 287, C345-C356.	4.6	28
53	Maitotoxin Induces Biphasic Interleukin-1Î² Secretion and Membrane Blebbing in Murine Macrophages. Molecular Pharmacology, 2004, 66, 909-920.	2.3	45
54	P2X7 Receptor-Dependent Blebbing and the Activation of Rho-Effector Kinases, Caspases, and IL-1Î² Release. Journal of Immunology, 2003, 170, 5728-5738.	0.8	151

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55	Blockade of maitotoxin-induced endothelial cell lysis by glycine and<scp>l</scp>-alanine. American Journal of Physiology - Cell Physiology, 2003, 284, C1006-C1020.	4.6	76
56	Blockade of maitotoxin-induced oncotic cell death reveals zeiosis. , 2002, 2, 2.		21
57	Maitotoxin-induced membrane blebbing and cell death in bovine aortic endothelial cells. , 2001, 1, 2.		31
58	Regulation of Drosophila transient receptor potentialâ€like (TrpL) channels by phospholipase Câ€dependent mechanisms. Journal of Physiology, 2001, 530, 1-19.	2.9	129
59	Regulation of Drosophila TRPL Channels by Immunophilin FKBP59. Journal of Biological Chemistry, 2001, 276, 38762-38773.	3.4	58
60	Maitotoxin activates a nonselective cation channel and a P2Z/P2X₇-like cytolytic pore in human skin fibroblasts. American Journal of Physiology - Cell Physiology, 1999, 277, C755-C765.	4.6	65
61	Stimulation of Drosophila TrpL by capacitative Ca2+ entry. Biochemical Journal, 1999, 341, 41-49.	3.7	19
62	Stimulation of Drosophila TrpL by capacitative Ca2+ entry. Biochemical Journal, 1999, 341, 41.	3.7	8
63	Functional expression of TrpC1: a human homologue of the Drosophila Trp channel. Biochemical Journal, 1998, 331, 331-339.	3.7	112
64	Mutations Causing Achondroplasia and Thanatophoric Dysplasia Alter bFGF-Induced Calcium Signals in Human Diploid Fibroblasts. Human Molecular Genetics, 1997, 6, 681-688.	2.9	20
65	PDGF-Stimulated Calcium Influx Changes During In Vitro Cell Transformation. Cellular Signalling, 1997, 9, 363-366.	3.6	8
66	Competence induction by PDGF requires sustained calcium influx by a mechanism distinct from storage-dependent calcium influx. Cell Calcium, 1993, 14, 439-454.	2.4	45
67	Expression of voltage-gated calcium channels correlates with PDGF-stimulated calcium influx and depends upon cell density in C3H 10T12 mouse fibroblasts. Cell Calcium, 1993, 14, 161-171.	2.4	34
68	Acute electrophysiological responses of bradykininâ€stimulated human fibroblasts.. Journal of Physiology, 1991, 436, 603-620.	2.9	19
69	Characterization of ion channels seen in subconfluent human dermal fibroblasts.. Journal of Physiology, 1991, 436, 579-601.	2.9	67
70	Inhibition of voltage-dependent Na+ current in cell-fusion hybrids containing activated c-Ha-ras. Journal of Membrane Biology, 1990, 113, 169-175.	2.1	11