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	Gain of function Na_V1.7 mutations in idiopathic small fiber neuropathy. Annals of Neurology, 2012, 71, 26-39.	5.3	518
2	Gain-of-function mutations in sodium channel NaV1.9 in painful neuropathy. Brain, 2014, 137, 1627-1642.	7.6	242
3	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
4	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
5	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
6	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
7	A novel Na_v1.7 mutation producing carbamazepineâ€responsive erythromelalgia. Annals of Neurology, 2009, 65, 733-741.	5.3	132
8	Regulation of Drosophila transient receptor potentialâ€like (TrpL) channels by phospholipase Câ€dependent mechanisms. Journal of Physiology, 2001, 530, 1-19.	2.9	129
9	Identification and localization of TRPC channels in the rat kidney. American Journal of Physiology - Renal Physiology, 2006, 290, F1241-F1252.	2.7	122
10	Functional expression of TrpC1: a human homologue of the Drosophila Trp channel. Biochemical Journal, 1998, 331, 331-339.	3.7	112
11	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
12	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
13	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
14	Voltage-clamp and current-clamp recordings from mammalian DRG neurons. Nature Protocols, 2009, 4, 1103-1112.	12.0	94
15	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
16	Characterization of a de novo SCN8A mutation in a patient with epileptic encephalopathy. Epilepsy Research, 2014, 108, 1511-1518.	1.6	92
17	A sodium channel gene SCN9A polymorphism that increases nociceptor excitability. Annals of Neurology, 2009, 66, 862-866.	5.3	91
18	Association of Immunophilins with Mammalian TRPC Channels. Journal of Biological Chemistry, 2004, 279, 34521-34529.	3.4	90

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19	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
20	Structural modelling and mutant cycle analysis predict pharmacoresponsiveness of a Nav1.7 mutant channel. Nature Communications, 2012, 3, 1186.	12.8	88
21	Activation of Human TRPC6 Channels by Receptor Stimulation. Journal of Biological Chemistry, 2004, 279, 22047-22056.	3.4	84
22	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
23	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
24	Pharmacotherapy for Pain in a Family With Inherited Erythromelalgia Guided by Genomic Analysis and Functional Profiling. JAMA Neurology, 2016, 73, 659.	9.0	70
25	Characterization of ion channels seen in subconfluent human dermal fibroblasts.. Journal of Physiology, 1991, 436, 579-601.	2.9	67
26	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
27	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
28	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
29	Regulation of Drosophila TRPL Channels by Immunophilin FKBP59. Journal of Biological Chemistry, 2001, 276, 38762-38773.	3.4	58
30	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
31	Sodium Channels, Mitochondria, and Axonal Degeneration in Peripheral Neuropathy. Trends in Molecular Medicine, 2016, 22, 377-390.	6.7	46
32	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
33	Maitotoxin Induces Biphasic Interleukin-1Î² Secretion and Membrane Blebbing in Murine Macrophages. Molecular Pharmacology, 2004, 66, 909-920.	2.3	45
34	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
35	A gain-of-function sodium channelÎ²2-subunit mutation in painful diabetic neuropathy. Molecular Pain, 2019, 15, 174480691984980.	2.1	38
36	Palytoxin-induced cell death cascade in bovine aortic endothelial cells. American Journal of Physiology - Cell Physiology, 2006, 291, C657-C667.	4.6	37

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37	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
38	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
39	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
40	Maitotoxin-induced membrane blebbing and cell death in bovine aortic endothelial cells. , 2001, 1, 2.		31
41	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
42	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
43	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
44	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
45	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
46	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
47	Dexpramipexole blocks Nav1.8 sodium channels and provides analgesia in multiple nociceptive and neuropathic pain models. Pain, 2020, 161, 831-841.	4.2	22
48	Blockade of maitotoxin-induced oncotic cell death reveals zeiosis. , 2002, 2, 2.		21
49	A new Nav1.7 mutation in an erythromelalgia patient. Biochemical and Biophysical Research Communications, 2013, 432, 99-104.	2.1	21
50	Molecular Architecture of a Sodium Channel S6 Helix. Journal of Biological Chemistry, 2013, 288, 13741-13747.	3.4	21
51	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
52	Acute electrophysiological responses of bradykininâ€stimulated human fibroblasts.. Journal of Physiology, 1991, 436, 603-620.	2.9	19
53	Stimulation of Drosophila TrpL by capacitative Ca2+ entry. Biochemical Journal, 1999, 341, 41-49.	3.7	19
54	A Novel Gain-of-Function Nav1.9 Mutation in a Child With Episodic Pain. Frontiers in Neuroscience, 2019, 13, 918.	2.8	18

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55	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
56	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
57	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
58	KCNQ variants and pain modulation: a missense variant in Kv7.3 contributes to pain resilience. Brain Communications, 2021, 3, fcab212.	3.3	13
59	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
60	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
61	PDGF-Stimulated Calcium Influx Changes During In Vitro Cell Transformation. Cellular Signalling, 1997, 9, 363-366.	3.6	8
62	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
63	Stimulation of Drosophila TrpL by capacitative Ca2+ entry. Biochemical Journal, 1999, 341, 41.	3.7	8
64	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
65	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
66	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
67	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
68	Depolarizing Na_V and Hyperpolarizing K_V Channels Are Co-Trafficked in Sensory Neurons. Journal of Neuroscience, 2022, 42, 4794-4811.	3.6	6
69	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
70	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