Stephen G Waxman

List of Publications by Year
in descending order

594 papers	44,577 citations	1531 109 h-index	4741 175 g-index
606 all docs	606 docs citations	606 times ranked	23399 citing authors

#	Article	IF	Citations
1	Stem cell-derived sensory neurons modelling inherited erythromelalgia: normalization of excitability. Brain, 2023, 146, 359-371.	3.7	9
2	A Buthus martensii Karsch scorpion sting targets Nav1.7 in mice and mimics a phenotype of human chronic pain. Pain, 2022, 163, e202-e214.	2.0	4
3	iPSCs and DRGs: stepping stones to new pain therapies. Trends in Molecular Medicine, 2022, 28, 110-122.	3.5	15
4	Inhibition of sodium conductance by cannabigerol contributes to a reduction of dorsal root ganglion neuron excitability. British Journal of Pharmacology, 2022, 179, 4010-4030.	2.7	16
5	Depolarizing Na_V and Hyperpolarizing K_V Channels Are Co-Trafficked in Sensory Neurons. Journal of Neuroscience, 2022, 42, 4794-4811.	1.7	6
6	Peripheral Ion Channel Gene Screening in Painful- and Painless-Diabetic Neuropathy. International Journal of Molecular Sciences, 2022, 23, 7190.	1.8	9
7	Mini-review - Sodium channels and beyond in peripheral nerve disease: Modulation by cytokines and their effector protein kinases. Neuroscience Letters, 2021, 741, 135446.	1.0	12
8	Core principles for the implementation of the neurodata without borders data standard. Journal of Neuroscience Methods, 2021, 348, 108972.	1.3	3
9	KCNQ variants and pain modulation: a missense variant in Kv7.3 contributes to pain resilience. Brain Communications, 2021, 3, fcab212.	1.5	13
10	Non-extensitivity and criticality of atomic hydropathicity around a voltage-gated sodium channelâ€™s pore: a modeling study. Journal of Biological Physics, 2021, 47, 61-77.	0.7	3
11	Paclitaxel increases axonal localization and vesicular trafficking of Nav1.7. Brain, 2021, 144, 1727-1737.	3.7	35
12	Hydropathicity-based prediction of pain-causing NaV1.7 variants. BMC Bioinformatics, 2021, 22, 212.	1.2	5
13	Intravenous infusion of auto serum-expanded autologous mesenchymal stem cells in spinal cord injury patients: 13 case series. Clinical Neurology and Neurosurgery, 2021, 203, 106565.	0.6	42
14	Conditional RAC1 knockout in motor neurons restores H-reflex rate-dependent depression after spinal cord injury. Scientific Reports, 2021, 11, 7838.	1.6	6
15	Human cells and networks of pain: Transforming pain target identification and therapeutic development. Neuron, 2021, 109, 1426-1429.	3.8	47
16	A novel gain-of-function sodium channel Î²2 subunit mutation in idiopathic small fiber neuropathy. Journal of Neurophysiology, 2021, 126, 827-839.	0.9	5
17	Hominini-specific regulation of CBLN2 increases prefrontal spinogenesis. Nature, 2021, 598, 489-494.	13.7	37
18	Trigeminal Neuralgia TRPM8 Mutation. Neurology: Genetics, 2021, 7, e550.	0.9	10

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19	Congenital Insensitivity to Pain. Cornea, 2021, Publish Ahead of Print, 1610-1613.	0.9	1
20	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.	2.0	9
21	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.	1.6	5
22	Contributions of NaV1.8 and NaV1.9 to excitability in human induced pluripotent stem-cell derived somatosensory neurons. Scientific Reports, 2021, 11, 24283.	1.6	6
23	Measurement of axonal excitability: Consensus guidelines. Clinical Neurophysiology, 2020, 131, 308-323.	0.7	63
24	The small fiber neuropathy NaV1.7 I228M mutation: impaired neurite integrity via bioenergetic and mitotoxic mechanisms, and protection by dexpramipexole. Journal of Neurophysiology, 2020, 123, 645-657.	0.9	9
25	Dexpramipexole blocks Nav1.8 sodium channels and provides analgesia in multiple nociceptive and neuropathic pain models. Pain, 2020, 161, 831-841.	2.0	22
26	Exome Sequencing Implicates Impaired GABA Signaling and Neuronal Ion Transport in Trigeminal Neuralgia. IScience, 2020, 23, 101552.	1.9	32
27	Computational pipeline to probe NaV1.7 gain-of-function variants in neuropathic painful syndromes. Scientific Reports, 2020, 10, 17930.	1.6	3
28	Genomic analysis of 21 patients with corneal neuralgia after refractive surgery. Pain Reports, 2020, 5, e826.	1.4	11
29	Status of peripheral sodium channel blockers for non-addictive pain treatment. Nature Reviews Neurology, 2020, 16, 689-705.	4.9	82
30	Evaluation of molecular inversion probe versus TruSeqÂ® custom methods for targeted next-generation sequencing. PLoS ONE, 2020, 15, e0238467.	1.1	17
31	Sodium channel Nav1.6 in sensory neurons contributes to vincristine-induced allodynia. Brain, 2020, 143, 2421-2436.	3.7	20
32	Dendritic Spine Dynamics after Peripheral Nerve Injury: An Intravital Structural Study. Journal of Neuroscience, 2020, 40, 4297-4308.	1.7	12
33	Pharmacological characterization of a rat Nav1.7 loss-of-function model with insensitivity to pain. Pain, 2020, 161, 1350-1360.	2.0	14
34	Pharmacological activity and NMR solution structure of the leech peptide HSTX-I. Biochemical Pharmacology, 2020, 181, 114082.	2.0	2
35	Familial trigeminal neuralgia â€“ a systematic clinical study with a genomic screen of the neuronal electrogenisome. Cephalalgia, 2020, 40, 767-777.	1.8	35
36	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.	1.6	8

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37	Differential effect of lacosamide on Nav1.7 variants from responsive and non-responsive patients with small fibre neuropathy. Brain, 2020, 143, 771-782.	3.7	31
38	Resilience to Stress and Resilience to Pain: Lessons from Molecular Neurobiology and Genetics. Trends in Molecular Medicine, 2020, 26, 924-935.	3.5	13
39	Cumulative hydropathic topology of a voltageâ€gated sodium channel at atomic resolution. Proteins: Structure, Function and Bioinformatics, 2020, 88, 1319-1328.	1.5	3
40	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.	1.6	6
41	Rational Drug Design for Pain Medicine. Anesthesiology, 2020, 133, 497-499.	1.3	1
42	A Novel Gain-of-Function Nav1.9 Mutation in a Child With Episodic Pain. Frontiers in Neuroscience, 2019, 13, 918.	1.4	18
43	Building sensory axons: Delivery and distribution of Na _V 1.7 channels and effects of inflammatory mediators. Science Advances, 2019, 5, eaax4755.	4.7	46
44	Sodium Channels in Human Pain Disorders: Genetics and Pharmacogenomics. Annual Review of Neuroscience, 2019, 42, 87-106.	5.0	92
45	Fibroblast growth factor homologous factor 2 (FGF-13) associates with Nav1.7 in DRG neurons and alters its current properties in an isoform-dependent manner. Neurobiology of Pain (Cambridge, Mass) Tj ETQq1 1 0.784314 rgBT /Ov	1.7	25
46	Na_V1.6 regulates excitability of mechanosensitive sensory neurons. Journal of Physiology, 2019, 597, 3751-3768.	1.3	31
47	A gain-of-function sodium channelÎ²2-subunit mutation in painful diabetic neuropathy. Molecular Pain, 2019, 15, 174480691984980.	1.0	38
48	Restoration of brain circulation and cellular functions hours post-mortem. Nature, 2019, 568, 336-343.	13.7	175
49	The Two Sides of NaV1.7: Painful and Painless Channelopathies. Neuron, 2019, 101, 765-767.	3.8	10
50	The Role of Voltage-Gated Sodium Channels in Pain Signaling. Physiological Reviews, 2019, 99, 1079-1151.	13.1	408
51	Spinal cord motor neuron plasticity accompanies secondâ€degree burn injury and chronic pain. Physiological Reports, 2019, 7, e14288.	0.7	12
52	Pointer-kindreds and pain: big lessons from small families. Pain, 2019, 160, S49-S52.	2.0	0
53	Peripheral afferents and the pain experience. Pain, 2019, 160, 1487-1488.	2.0	7
54	Smallâ€fiber neuropathy: Expanding the clinical pain universe. Journal of the Peripheral Nervous System, 2019, 24, 19-33.	1.4	71

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55	Pediatric Erythromelalgia and SCN9A Mutations: Systematic Review and Single-Center Case Series. Journal of Pediatrics, 2019, 206, 217-224.e9.	0.9	18
56	Resilience to Pain: A Peripheral Component Identified Using Induced Pluripotent Stem Cells and Dynamic Clamp. Journal of Neuroscience, 2019, 39, 382-392.	1.7	66
57	Lacosamide in patients with Nav1.7 mutations-related small fibre neuropathy: a randomized controlled trial. Brain, 2019, 142, 263-275.	3.7	85
58	Expression of pathogenic SCN9A mutations in the zebrafish: A model to study small-fiber neuropathy. Experimental Neurology, 2019, 311, 257-264.	2.0	16
59	Yield of peripheral sodium channels gene screening in pure small fibre neuropathy. Journal of Neurology, Neurosurgery and Psychiatry, 2019, 90, 342-352.	0.9	47
60	Episodic Pain Syndrome Associated with a Novel Heterozygous Gain-of-Function SCN11A Missense Mutation. Neuropediatrics, 2019, 50, .	0.3	0
61	Conditional knockout of NaV1.6 in adult mice ameliorates neuropathic pain. Scientific Reports, 2018, 8, 3845.	1.6	66
62	Brain activity associated with pain in inherited erythromelalgia: stimulus-free pain engages brain areas involved in valuation and learning. Neurobiology of Pain (Cambridge, Mass), 2018, 3, 8-14.	1.0	2
63	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.	1.6	14
64	Na V 1.7 as a Pharmacogenomic Target for Pain: Moving Toward Precision Medicine. Trends in Pharmacological Sciences, 2018, 39, 258-275.	4.0	54
65	Reverse pharmacogenomics: carbamazepine normalizes activation and attenuates thermal hyperexcitability of sensory neurons due to Na_v1.7 mutation I234T. British Journal of Pharmacology, 2018, 175, 2261-2271.	2.7	29
66	Detection of local and remote cellular damage caused by spinal cord and peripheral nerve injury using a heat shock signaling reporter system. IBRO Reports, 2018, 5, 91-98.	0.3	9
67	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.	1.0	7
68	Nav1.5 in astrocytes plays a sexâ€specific role in clinical outcomes in a mouse model of multiple sclerosis. Glia, 2018, 66, 2174-2187.	2.5	10
69	Somatosensory Neurons Enter a State of Altered Excitability during Hibernation. Current Biology, 2018, 28, 2998-3004.e3.	1.8	12
70	Nav1.7 is phosphorylated by Fyn tyrosine kinase which modulates channel expression and gating in a cell type-dependent manner. Molecular Pain, 2018, 14, 174480691878222.	1.0	16
71	Nonmuscle myosin II isoforms interact with sodium channel alpha subunits. Molecular Pain, 2018, 14, 174480691878863.	1.0	7
72	Therapeutic potential of Pak1 inhibition for pain associated with cutaneous burn injury. Molecular Pain, 2018, 14, 174480691878864.	1.0	12

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73	Differential agingâ€related changes in neurophysiology and gene expression in IB4â€positive and IB4â€negative nociceptive neurons. Aging Cell, 2018, 17, e12795.	3.0	6
74	Loss-of-function mutations of SCN10A encoding NaV1.8 Î± subunit of voltage-gated sodium channel in patients with human kidney stone disease. Scientific Reports, 2018, 8, 10453.	1.6	7
75	Multiple myosin motors interact with sodium/potassium-ATPase alpha 1 subunits. Molecular Brain, 2018, 11, 45.	1.3	11
76	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.	1.0	24
77	Characterization of small fiber pathology in a mouse model of Fabry disease. ELife, 2018, 7, .	2.8	38
78	Alabama to Beijingâ€¦ and Back: The Search for a Pain Gene. Cerebrum: the Dana Forum on Brain Science, 2018, 2018, .	0.1	0
79	Pharmacological characterisation of the highly NaV1.7 selective spider venom peptide Pn3a. Scientific Reports, 2017, 7, 40883.	1.6	120
80	COL6A5variants in familial neuropathic chronic itch. Brain, 2017, 140, aww343.	3.7	25
81	Detection of vulnerable neurons damaged by environmental insults in utero. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 2367-2372.	3.3	17
82	Familial gain-of-function Na_v1.9 mutation in a painful channelopathy. Journal of Neurology, Neurosurgery and Psychiatry, 2017, 88, 233-240.	0.9	49
83	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.	4.9	144
84	Network topology of NaV1.7 mutations in sodium channel-related painful disorders. BMC Systems Biology, 2017, 11, 28.	3.0	29
85	Gain-of-function mutation of a voltage-gated sodium channel NaV1.7 associated with peripheral pain and impaired limb development. Journal of Biological Chemistry, 2017, 292, 9262-9272.	1.6	21
86	Sodium channels in pain disorders: pathophysiology and prospects for treatment. Pain, 2017, 158, S97-S107.	2.0	64
87	Nonlinear effects of hyperpolarizing shifts in activation of mutant Na_v1.7 channels on resting membrane potential. Journal of Neurophysiology, 2017, 117, 1702-1712.	0.9	6
88	Dendritic spine dysgenesis in superficial dorsal horn sensory neurons after spinal cord injury. Molecular Pain, 2017, 13, 174480691668801.	1.0	26
89	Mechanism of inhibition by chlorpromazine of the human pain threshold sodium channel, Nav1.7. Neuroscience Letters, 2017, 639, 1-7.	1.0	3
90	Ode to Glia: A Tribute to Bruce Ransom. Neurochemical Research, 2017, 42, 2442-2442.	1.6	0

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91	Between fire and ice: refractory hypothermia and warmth-induced pain in inherited erythromelalgia. BMJ Case Reports, 2017, 2017, bcr-2017-219486.	0.2	8
92	Sodium channel NaV1.9 mutations associated with insensitivity to pain dampen neuronal excitability. Journal of Clinical Investigation, 2017, 127, 2805-2814.	3.9	65
93	The AMPK Activator A769662 Blocks Voltage-Gated Sodium Channels: Discovery of a Novel Pharmacophore with Potential Utility for Analgesic Development. PLoS ONE, 2017, 12, e0169882.	1.1	16
94	A Gain-of-Function Mutation in Nav1.6 in a Case of Trigeminal Neuralgia. Molecular Medicine, 2016, 22, 338-348.	1.9	98
95	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.	1.1	152
96	Sodium channels in astroglia and microglia. Glia, 2016, 64, 1628-1645.	2.5	62
97	Dendritic spine remodeling following early and late Rac1 inhibition after spinal cord injury: evidence for a pain biomarker. Journal of Neurophysiology, 2016, 115, 2893-2910.	0.9	29
98	A SCN10A SNP biases human pain sensitivity. Molecular Pain, 2016, 12, 174480691666608.	1.0	40
99	Pharmacotherapy for Pain in a Family With Inherited Erythromelalgia Guided by Genomic Analysis and Functional Profiling. JAMA Neurology, 2016, 73, 659.	4.5	70
100	Pain Perception. JAMA Neurology, 2016, 73, 628.	4.5	14
101	Sodium Channels, Mitochondria, and Axonal Degeneration in Peripheral Neuropathy. Trends in Molecular Medicine, 2016, 22, 377-390.	3.5	46
102	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.	1.7	61
103	Pharmacological reversal of a pain phenotype in iPSC-derived sensory neurons and patients with inherited erythromelalgia. Science Translational Medicine, 2016, 8, 335ra56.	5.8	154
104	A painful neuropathy-associated Nav1.7 mutant leads to time-dependent degeneration of small-diameter axons associated with intracellular Ca²⁺dysregulation and decrease in ATP levels. Molecular Pain, 2016, 12, 174480691667447.	1.0	23
105	Inherited erythromelalgia due to mutations inSCN9A:natural history, clinical phenotype and somatosensory profile. Brain, 2016, 139, 1052-1065.	3.7	72
106	Sodium channel Na _v 1.8. Neurology, 2016, 86, 473-483.	1.5	83
107	The cerebellar channelopathy of multiple sclerosis. Neurology, 2016, 86, 406-407.	1.5	4
108	Voltage-Gated Ion Channels as Molecular Targets for Pain. , 2016, , 415-436.		1

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109	Sodium Channel Nav1.7 in Vascular Myocytes, Endothelium, and Innervating Axons in Human Skin. Molecular Pain, 2015, 11, s12990-015-0024.	1.0	28
110	Diversity of composition and function of sodium channels in peripheral sensory neurons. Pain, 2015, 156, 2406-2407.	2.0	22
111	Oral Administration of PF-01247324, a Subtype-Selective Nav1.8 Blocker, Reverses Cerebellar Deficits in a Mouse Model of Multiple Sclerosis. PLoS ONE, 2015, 10, e0119067.	1.1	18
112	Preferential Targeting of Nav1.6 Voltage-Gated Na+ Channels to the Axon Initial Segment during Development. PLoS ONE, 2015, 10, e0124397.	1.1	59
113	Contactin-1 and Neurofascin-155/-186 Are Not Targets of Auto-Antibodies in Multifocal Motor Neuropathy. PLoS ONE, 2015, 10, e0134274.	1.1	19
114	Virus-Mediated Knockdown of Nav1.3 in Dorsal Root Ganglia of STZ-Induced Diabetic Rats Alleviates Tactile Allodynia. Molecular Medicine, 2015, 21, 544-552.	1.9	62
115	De novo gain-of-function and loss-of-function mutations ofSCN8Ain patients with intellectual disabilities and epilepsy. Journal of Medical Genetics, 2015, 52, 330-337.	1.5	124
116	Painful peripheral neuropathy and sodium channel mutations. Neuroscience Letters, 2015, 596, 51-59.	1.0	66
117	Dendritic spine dysgenesis contributes to hyperreflexia after spinal cord injury. Journal of Neurophysiology, 2015, 113, 1598-1615.	0.9	42
118	NaV1.9: a sodium channel linked to human pain. Nature Reviews Neuroscience, 2015, 16, 511-519.	4.9	161
119	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.	0.9	89
120	The Domain II S4-S5 Linker in Nav1.9: A Missense Mutation Enhances Activation, Impairs Fast Inactivation, and Produces Human Painful Neuropathy. NeuroMolecular Medicine, 2015, 17, 158-169.	1.8	70
121	Destruction of paranodal architecture in inflammatory neuropathy with anti-contactin-1 autoantibodies. Journal of Neurology, Neurosurgery and Psychiatry, 2015, 86, 720-728.	0.9	152
122	Neurologyâ€”the next 10 years. Nature Reviews Neurology, 2015, 11, 658-664.	4.9	7
123	Dendritic spine dysgenesis in neuropathic pain. Neuroscience Letters, 2015, 601, 54-60.	1.0	25
124	Decreased Resting Functional Connectivity after Traumatic Brain Injury in the Rat. PLoS ONE, 2014, 9, e95280.	1.1	54
125	Translational pain research: Lessons from genetics and genomics. Science Translational Medicine, 2014, 6, 249sr4.	5.8	45
126	Depolarized Inactivation Overcomes Impaired Activation to Produce DRG Neuron Hyperexcitability in a Na_v1.7 Mutation in a Patient with Distal Limb Pain. Journal of Neuroscience, 2014, 34, 12328-12340.	1.7	18

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127	Painful neuropathies: the emerging role of sodium channelopathies. Journal of the Peripheral Nervous System, 2014, 19, 53-65.	1.4	82
128	Dynamics of sodium channel Nav1.5 expression in astrocytes in mouse models of multiple sclerosis. NeuroReport, 2014, 25, 1208-1215.	0.6	15
129	Regulating excitability of peripheral afferents: emerging ion channel targets. Nature Neuroscience, 2014, 17, 153-163.	7.1	361
130	Voltageâ€gated sodium channel Na_v1.5 contributes to astrogliosis in an in vitro model of glial injury via reverse Na⁺/Ca²⁺ exchange. Glia, 2014, 62, 1162-1175.	2.5	69
131	Contribution of sodium channels to lamellipodial protrusion and Rac1 and ERK1/2 activation in ATPâ€stimulated microglia. Glia, 2014, 62, 2080-2095.	2.5	30
132	Neuropathic pain in two-generation twins carrying the sodium channel Nav1.7 functional variant R1150W. Pain, 2014, 155, 2199-2203.	2.0	12
133	The Role of Sodium Channels in Painful Diabetic and Idiopathic Neuropathy. Current Diabetes Reports, 2014, 14, 538.	1.7	33
134	Sodium channel genes in pain-related disorders: phenotypeâ€“genotype associations and recommendations for clinical use. Lancet Neurology, The, 2014, 13, 1152-1160.	4.9	148
135	The G1662S NaV1.8 mutation in small fibre neuropathy: impaired inactivation underlying DRG neuron hyperexcitability. Journal of Neurology, Neurosurgery and Psychiatry, 2014, 85, 499-505.	0.9	80
136	Channelopathies, painful neuropathy, and diabetes: which way does the causal arrow point?. Trends in Molecular Medicine, 2014, 20, 544-550.	3.5	32
137	Gain-of-function mutations in sodium channel NaV1.9 in painful neuropathy. Brain, 2014, 137, 1627-1642.	3.7	242
138	Characterization of a de novo SCN8A mutation in a patient with epileptic encephalopathy. Epilepsy Research, 2014, 108, 1511-1518.	0.8	92
139	Paroxysmal itch caused by gain-of-function Nav1.7 mutation. Pain, 2014, 155, 1702-1707.	2.0	78
140	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.	2.1	96
141	Dynamic-clamp analysis of wild-type human Na_v1.7 and erythromelalgia mutant channel L858H. Journal of Neurophysiology, 2014, 111, 1429-1443.	0.9	59
142	Approach to Small Fiber Neuropathy. , 2014, , 507-517.		2
143	Altered Sodium Channel Gating as Molecular Basis for Pain: Contribution of Activation, Inactivation, and Resurgent Currents. Handbook of Experimental Pharmacology, 2014, 221, 91-110.	0.9	45
144	Painful Na-channelopathies: an expanding universe. Trends in Molecular Medicine, 2013, 19, 406-409.	3.5	60

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145	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.	1.7	61
146	Correlation of Nav1.8 and Nav1.9 Sodium Channel Expression with Neuropathic Pain in Human Subjects with Lingual Nerve Neuromas. Molecular Pain, 2013, 9, 1744-8069-9-52.	1.0	23
147	Nav1.7: Stress-Induced Changes in Immunoreactivity within Magnocellular Neurosecretory Neurons of the Supraoptic Nucleus. Molecular Pain, 2013, 9, 1744-8069-9-39.	1.0	24
148	Small-Fiber Neuropathy Nav1.8 Mutation Shifts Activation to Hyperpolarized Potentials and Increases Excitability of Dorsal Root Ganglion Neurons. Journal of Neuroscience, 2013, 33, 14087-14097.	1.7	107
149	The NaV1.7 sodium channel: from molecule to man. Nature Reviews Neuroscience, 2013, 14, 49-62.	4.9	474
150	Noncanonical Roles of Voltage-Gated Sodium Channels. Neuron, 2013, 80, 280-291.	3.8	171
151	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.	1.1	14
152	Wound-healing growth factor, basic FGF, induces Erk1/2-dependent mechanical hyperalgesia. Pain, 2013, 154, 2216-2226.	2.0	41
153	Burn injury-induced mechanical allodynia is maintained by Rac1-regulated dendritic spine dysgenesis. Experimental Neurology, 2013, 248, 509-519.	2.0	32
154	A new Nav1.7 mutation in an erythromelalgia patient. Biochemical and Biophysical Research Communications, 2013, 432, 99-104.	1.0	21
155	The response of Na_V1.3 sodium channels to ramp stimuli: multiple components and mechanisms. Journal of Neurophysiology, 2013, 109, 306-314.	0.9	36
156	Axonal Protection with Sodium Channel Blocking Agents in Models of Multiple Sclerosis. , 2013, , 179-201.		0
157	Nav1.5 sodium channels in macrophages in multiple sclerosis lesions. Multiple Sclerosis Journal, 2013, 19, 532-542.	1.4	30
158	Virus-mediated shRNA Knockdown of Nav1.3 in Rat Dorsal Root Ganglion Attenuates Nerve Injury-induced Neuropathic Pain. Molecular Therapy, 2013, 21, 49-56.	3.7	91
159	Neuropathyâ€associated Na_V1.7 variant I228M impairs integrity of dorsal root ganglion neuron axons. Annals of Neurology, 2013, 73, 140-145.	2.8	52
160	Multistate Structural Modeling and Voltage-Clamp Analysis of Epilepsy/Autism Mutation Kv10.2â€“R327H Demonstrate the Role of This Residue in Stabilizing the Channel Closed State. Journal of Neuroscience, 2013, 33, 16586-16593.	1.7	39
161	Molecular Architecture of a Sodium Channel S6 Helix. Journal of Biological Chemistry, 2013, 288, 13741-13747.	1.6	21
162	Screening Fluorescent Voltage Indicators with Spontaneously Spiking HEK Cells. PLoS ONE, 2013, 8, e85221.	1.1	77

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163	Opportunities in rehabilitation research. Journal of Rehabilitation Research and Development, 2013, 50, vii-xxxii.	1.6	7
164	Membrane properties and electrogenesis in the distal axons of small dorsal root ganglion neurons in vitro. Journal of Neurophysiology, 2012, 108, 729-740.	0.9	47
165	Extraterritorial temperature pain threshold abnormalities in subjects with healed thermal injury. Journal of Rehabilitation Research and Development, 2012, 49, 515.	1.6	12
166	Interaction of Voltage-gated Sodium Channel Nav1.6 (SCN8A) with Microtubule-associated Protein Map1b. Journal of Biological Chemistry, 2012, 287, 18459-18466.	1.6	32
167	Functional profiles of SCN9A variants in dorsal root ganglion neurons and superior cervical ganglion neurons correlate with autonomic symptoms in small fibre neuropathy. Brain, 2012, 135, 2613-2628.	3.7	90
168	Small nerve fibres, small hands and small feet: a new syndrome of pain, dysautonomia and acromesomelia in a kindred with a novel NaV1.7 mutation. Brain, 2012, 135, 345-358.	3.7	69
169	Interview: Pathways to discovery: making the translational leap. Pain Management, 2012, 2, 117-118.	0.7	0
170	Gain-of-function Na _v 1.8 mutations in painful neuropathy. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 19444-19449.	3.3	369
171	An AnkyrinG-Binding Motif Is Necessary and Sufficient for Targeting Na_v1.6 Sodium Channels to Axon Initial Segments and Nodes of Ranvier. Journal of Neuroscience, 2012, 32, 7232-7243.	1.7	115
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