Sulayman D Dib-Hajj

List of Publications by Year
in descending order

171 papers	15,493 citations	11651 70 h-index	19749 117 g-index
173 all docs	173 docs citations	173 times ranked	8272 citing authors

#	Article	IF	Citations
1	A Buthus martensii Karsch scorpion sting targets Nav1.7 in mice and mimics a phenotype of human chronic pain. Pain, 2022, 163, e202-e214.	4.2	4
2	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
3	Depolarizing Na_V and Hyperpolarizing K_V Channels Are Co-Trafficked in Sensory Neurons. Journal of Neuroscience, 2022, 42, 4794-4811.	3.6	6
4	Mini-review - Sodium channels and beyond in peripheral nerve disease: Modulation by cytokines and their effector protein kinases. Neuroscience Letters, 2021, 741, 135446.	2.1	12
5	KCNQ variants and pain modulation: a missense variant in Kv7.3 contributes to pain resilience. Brain Communications, 2021, 3, fcab212.	3.3	13
6	Paclitaxel increases axonal localization and vesicular trafficking of Nav1.7. Brain, 2021, 144, 1727-1737.	7.6	35
7	Human cells and networks of pain: Transforming pain target identification and therapeutic development. Neuron, 2021, 109, 1426-1429.	8.1	47
8	A novel gain-of-function sodium channel Î²2 subunit mutation in idiopathic small fiber neuropathy. Journal of Neurophysiology, 2021, 126, 827-839.	1.8	5
9	Trigeminal Neuralgia TRPM8 Mutation. Neurology: Genetics, 2021, 7, e550.	1.9	10
10	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
11	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
12	Dexpramipexole blocks Nav1.8 sodium channels and provides analgesia in multiple nociceptive and neuropathic pain models. Pain, 2020, 161, 831-841.	4.2	22
13	Evaluation of molecular inversion probe versus TruSeqÂ® custom methods for targeted next-generation sequencing. PLoS ONE, 2020, 15, e0238467.	2.5	17
14	Sodium channel Nav1.6 in sensory neurons contributes to vincristine-induced allodynia. Brain, 2020, 143, 2421-2436.	7.6	20
15	Pharmacological characterization of a rat Nav1.7 loss-of-function model with insensitivity to pain. Pain, 2020, 161, 1350-1360.	4.2	14
16	Pharmacological activity and NMR solution structure of the leech peptide HSTX-I. Biochemical Pharmacology, 2020, 181, 114082.	4.4	2
17	Familial trigeminal neuralgia â€“ a systematic clinical study with a genomic screen of the neuronal electrogenisome. Cephalalgia, 2020, 40, 767-777.	3.9	35
18	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

#	Article	IF	Citations
19	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
20	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
21	A Novel Gain-of-Function Nav1.9 Mutation in a Child With Episodic Pain. Frontiers in Neuroscience, 2019, 13, 918.	2.8	18
22	Building sensory axons: Delivery and distribution of Na _V 1.7 channels and effects of inflammatory mediators. Science Advances, 2019, 5, eaax4755.	10.3	46
23	Sodium Channels in Human Pain Disorders: Genetics and Pharmacogenomics. Annual Review of Neuroscience, 2019, 42, 87-106.	10.7	92
24	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 ETQq0 0 0 rgBT /Overlock 10 T	2.5	28
25	Na_V1.6 regulates excitability of mechanosensitive sensory neurons. Journal of Physiology, 2019, 597, 3751-3768.	2.9	31
26	A gain-of-function sodium channelÎ²2-subunit mutation in painful diabetic neuropathy. Molecular Pain, 2019, 15, 174480691984980.	2.1	38
27	The Two Sides of NaV1.7: Painful and Painless Channelopathies. Neuron, 2019, 101, 765-767.	8.1	10
28	The Role of Voltage-Gated Sodium Channels in Pain Signaling. Physiological Reviews, 2019, 99, 1079-1151.	28.8	408
29	Pediatric Erythromelalgia and SCN9A Mutations: Systematic Review and Single-Center Case Series. Journal of Pediatrics, 2019, 206, 217-224.e9.	1.8	18
30	Resilience to Pain: A Peripheral Component Identified Using Induced Pluripotent Stem Cells and Dynamic Clamp. Journal of Neuroscience, 2019, 39, 382-392.	3.6	66
31	Lacosamide in patients with Nav1.7 mutations-related small fibre neuropathy: a randomized controlled trial. Brain, 2019, 142, 263-275.	7.6	85
32	Episodic Pain Syndrome Associated with a Novel Heterozygous Gain-of-Function SCN11A Missense Mutation. Neuropediatrics, 2019, 50, .	0.6	0
33	Conditional knockout of NaV1.6 in adult mice ameliorates neuropathic pain. Scientific Reports, 2018, 8, 3845.	3.3	66
34	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.	2.5	2
35	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
36	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

#	Article	IF	Citations
37	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.	5.4	29
38	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
39	Somatosensory Neurons Enter a State of Altered Excitability during Hibernation. Current Biology, 2018, 28, 2998-3004.e3.	3.9	12
40	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.	2.1	16
41	Nonmuscle myosin II isoforms interact with sodium channel alpha subunits. Molecular Pain, 2018, 14, 174480691878863.	2.1	7
42	Therapeutic potential of Pak1 inhibition for pain associated with cutaneous burn injury. Molecular Pain, 2018, 14, 174480691878864.	2.1	12
43	Differential agingâ€related changes in neurophysiology and gene expression in IB4â€positive and IB4â€negative nociceptive neurons. Aging Cell, 2018, 17, e12795.	6.7	6
44	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.	3.3	7
45	Multiple myosin motors interact with sodium/potassium-ATPase alpha 1 subunits. Molecular Brain, 2018, 11, 45.	2.6	11
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	Characterization of small fiber pathology in a mouse model of Fabry disease. ELife, 2018, 7, .	6.0	38
48	Pharmacological characterisation of the highly NaV1.7 selective spider venom peptide Pn3a. Scientific Reports, 2017, 7, 40883.	3.3	120
49	COL6A5variants in familial neuropathic chronic itch. Brain, 2017, 140, aww343.	7.6	25
50	Familial gain-of-function Na_v1.9 mutation in a painful channelopathy. Journal of Neurology, Neurosurgery and Psychiatry, 2017, 88, 233-240.	1.9	49
51	Network topology of NaV1.7 mutations in sodium channel-related painful disorders. BMC Systems Biology, 2017, 11, 28.	3.0	29
52	Sodium channels in pain disorders: pathophysiology and prospects for treatment. Pain, 2017, 158, S97-S107.	4.2	64
53	Sodium channel NaV1.9 mutations associated with insensitivity to pain dampen neuronal excitability. Journal of Clinical Investigation, 2017, 127, 2805-2814.	8.2	65
54	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.	2.5	16

#	Article	IF	Citations
55	A Gain-of-Function Mutation in Nav1.6 in a Case of Trigeminal Neuralgia. Molecular Medicine, 2016, 22, 338-348.	4.4	98
56	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
57	A SCN10A SNP biases human pain sensitivity. Molecular Pain, 2016, 12, 174480691666608.	2.1	40
58	Pharmacotherapy for Pain in a Family With Inherited Erythromelalgia Guided by Genomic Analysis and Functional Profiling. JAMA Neurology, 2016, 73, 659.	9.0	70
59	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
60	iFGF14-Na_vs: A monogamous partnership?. Channels, 2016, 10, 435-436.	2.8	1
61	Inherited erythromelalgia due to mutations inSCN9A:natural history, clinical phenotype and somatosensory profile. Brain, 2016, 139, 1052-1065.	7.6	72
62	Single amino acid deletion in transmembrane segment D4S6 of sodium channel Scn8a (Nav1.6) in a mouse mutant with a chronic movement disorder. Neurobiology of Disease, 2016, 89, 36-45.	4.4	23
63	Diversity of composition and function of sodium channels in peripheral sensory neurons. Pain, 2015, 156, 2406-2407.	4.2	22
64	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.	2.5	18
65	Preferential Targeting of Nav1.6 Voltage-Gated Na+ Channels to the Axon Initial Segment during Development. PLoS ONE, 2015, 10, e0124397.	2.5	59
66	Contactin-1 and Neurofascin-155/-186 Are Not Targets of Auto-Antibodies in Multifocal Motor Neuropathy. PLoS ONE, 2015, 10, e0134274.	2.5	19
67	Virus-Mediated Knockdown of Nav1.3 in Dorsal Root Ganglia of STZ-Induced Diabetic Rats Alleviates Tactile Allodynia. Molecular Medicine, 2015, 21, 544-552.	4.4	62
68	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.	3.2	124
69	NaV1.9: a sodium channel linked to human pain. Nature Reviews Neuroscience, 2015, 16, 511-519.	10.2	161
70	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
71	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.	3.4	70
72	Destruction of paranodal architecture in inflammatory neuropathy with anti-contactin-1 autoantibodies. Journal of Neurology, Neurosurgery and Psychiatry, 2015, 86, 720-728.	1.9	152

#	Article	IF	Citations
73	Translational pain research: Lessons from genetics and genomics. Science Translational Medicine, 2014, 6, 249sr4.	12.4	45
74	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.	3.6	18
75	Neuropathic pain in two-generation twins carrying the sodium channel Nav1.7 functional variant R1150W. Pain, 2014, 155, 2199-2203.	4.2	12
76	Sodium channel genes in pain-related disorders: phenotypeâ€“genotype associations and recommendations for clinical use. Lancet Neurology, The, 2014, 13, 1152-1160.	10.2	148
77	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.	1.9	80
78	Human pain in a dish: Native DRG neurons and differentiated pluripotent stem cells. Pain, 2014, 155, 1681-1682.	4.2	4
79	Gain-of-function mutations in sodium channel NaV1.9 in painful neuropathy. Brain, 2014, 137, 1627-1642.	7.6	242
80	Characterization of a de novo SCN8A mutation in a patient with epileptic encephalopathy. Epilepsy Research, 2014, 108, 1511-1518.	1.6	92
81	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
82	Dynamic-clamp analysis of wild-type human Na_v1.7 and erythromelalgia mutant channel L858H. Journal of Neurophysiology, 2014, 111, 1429-1443.	1.8	59
83	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.	3.6	107
84	The NaV1.7 sodium channel: from molecule to man. Nature Reviews Neuroscience, 2013, 14, 49-62.	10.2	474
85	A new Nav1.7 mutation in an erythromelalgia patient. Biochemical and Biophysical Research Communications, 2013, 432, 99-104.	2.1	21
86	Virus-mediated shRNA Knockdown of Nav1.3 in Rat Dorsal Root Ganglion Attenuates Nerve Injury-induced Neuropathic Pain. Molecular Therapy, 2013, 21, 49-56.	8.2	91
87	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.	3.6	39
88	Molecular Architecture of a Sodium Channel S6 Helix. Journal of Biological Chemistry, 2013, 288, 13741-13747.	3.4	21
89	Screening Fluorescent Voltage Indicators with Spontaneously Spiking HEK Cells. PLoS ONE, 2013, 8, e85221.	2.5	77
90	Interaction of Voltage-gated Sodium Channel Nav1.6 (SCN8A) with Microtubule-associated Protein Map1b. Journal of Biological Chemistry, 2012, 287, 18459-18466.	3.4	32

#	Article	IF	Citations
91	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.	7.6	90
92	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.	7.6	69
93	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.	7.1	369
94	Sodium Channel Na_v1.7 Is Essential for Lowering Heat Pain Threshold after Burn Injury. Journal of Neuroscience, 2012, 32, 10819-10832.	3.6	88
95	Structural modelling and mutant cycle analysis predict pharmacoresponsiveness of a Nav1.7 mutant channel. Nature Communications, 2012, 3, 1186.	12.8	88
96	Nav1.8 expression is not restricted to nociceptors in mouse peripheral nervous system. Pain, 2012, 153, 2017-2030.	4.2	223
97	Expression of Nav1.7 in DRG Neurons Extends from Peripheral Terminals in the Skin to Central Preterminal Branches and Terminals in the Dorsal Horn. Molecular Pain, 2012, 8, 1744-8069-8-82.	2.1	156
98	De Novo Pathogenic SCN8A Mutation Identified by Whole-Genome Sequencing of a Family Quartet Affected by Infantile Epileptic Encephalopathy and SUDEP. American Journal of Human Genetics, 2012, 90, 502-510.	6.2	365
99	Gain of function Na_V1.7 mutations in idiopathic small fiber neuropathy. Annals of Neurology, 2012, 71, 26-39.	5.3	518
100	PKCÎµ phosphorylation of the sodium channel NaV1.8 increases channel function and produces mechanical hyperalgesia in mice. Journal of Clinical Investigation, 2012, 122, 1306-1315.	8.2	41
101	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
102	Deletion mutation of sodium channel NaV1.7 in inherited erythromelalgia: enhanced slow inactivation modulates dorsal root ganglion neuron hyperexcitability. Brain, 2011, 134, 1972-1986.	7.6	66
103	Paroxysmal extreme pain disorder: a molecular lesion of peripheral neurons. Nature Reviews Neurology, 2011, 7, 51-55.	10.1	57
104	Mutations at opposite Ends of the DIII/S4-S5 Linker of Sodium Channel NaV1.7 Produce Distinct Pain Disorders. Molecular Pain, 2010, 6, 1744-8069-6-24.	2.1	33
105	A new Na_v1.7 sodium channel mutation I234T in a child with severe pain. European Journal of Pain, 2010, 14, 944-950.	2.8	42
106	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
107	ERK1/2 Mitogen-Activated Protein Kinase Phosphorylates Sodium Channel Na_v1.7 and Alters Its Gating Properties. Journal of Neuroscience, 2010, 30, 1637-1647.	3.6	149
108	Alternative splicing may contribute to time-dependent manifestation of inherited erythromelalgia. Brain, 2010, 133, 1823-1835.	7.6	56

#	Article	IF	Citations
109	Two Nedd4-binding Motifs Underlie Modulation of Sodium Channel Nav1.6 by p38 MAPK. Journal of Biological Chemistry, 2010, 285, 26149-26161.	3.4	47
110	Isoform-specific and pan-channel partners regulate trafficking and plasma membrane stability; and alter sodium channel gating properties. Neuroscience Letters, 2010, 486, 84-91.	2.1	25
111	Proteomics of voltage-gated ion channels. Neuroscience Letters, 2010, 486, 51-52.	2.1	0
112	Sodium Channels in Normal and Pathological Pain. Annual Review of Neuroscience, 2010, 33, 325-347.	10.7	529
113	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
114	Early- and late-onset inherited erythromelalgia: genotypeâ€“phenotype correlation. Brain, 2009, 132, 1711-1722.	7.6	117
115	Theataxia3Mutation in the N-Terminal Cytoplasmic Domain of Sodium Channel Na_v1.6 Disrupts Intracellular Trafficking. Journal of Neuroscience, 2009, 29, 2733-2741.	3.6	43
116	Voltage-gated sodium channels in pain states: Role in pathophysiology and targets for treatment. Brain Research Reviews, 2009, 60, 65-83.	9.0	130
117	A novel Na_v1.7 mutation producing carbamazepineâ€responsive erythromelalgia. Annals of Neurology, 2009, 65, 733-741.	5.3	132
118	Transfection of rat or mouse neurons by biolistics or electroporation. Nature Protocols, 2009, 4, 1118-1127.	12.0	110
119	Voltage-clamp and current-clamp recordings from mammalian DRG neurons. Nature Protocols, 2009, 4, 1103-1112.	12.0	94
120	Voltage-Gated Sodium Channels: Therapeutic Targets for Pain. Pain Medicine, 2009, 10, 1260-1269.	1.9	200
121	Role of hippocampal sodium channel Nav1.6 in kindling epileptogenesis. Epilepsia, 2009, 50, 44-55.	5.1	129
122	Mexiletine-responsive erythromelalgia due to a new Nav1.7 mutation showing use-dependent current fall-off. Experimental Neurology, 2009, 216, 383-389.	4.1	73
123	FGF14 N-terminal splice variants differentially modulate Nav1.2 and Nav1.6-encoded sodium channels. Molecular and Cellular Neurosciences, 2009, 42, 90-101.	2.2	117
124	Inherited Neuronal Ion Channelopathies: New Windows on Complex Neurological Diseases. Journal of Neuroscience, 2008, 28, 11768-11777.	3.6	225
125	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
126	Mutation I136V Alters Electrophysiological Properties of the Na_V1.7 Channel in a Family with Onset of Erythromelalgia in the Second Decade. Molecular Pain, 2008, 4, 1744-8069-4-1.	2.1	101

#	Article	IF	Citations
127	Voltage-gated sodium channel expression in rat and human epidermal keratinocytes: Evidence for a role in pain. Pain, 2008, 139, 90-105.	4.2	153
128	A Pore-blocking Hydrophobic Motif at the Cytoplasmic Aperture of the Closed-state Nav1.7 Channel Is Disrupted by the Erythromelalgia-associated F1449V Mutation. Journal of Biological Chemistry, 2008, 283, 24118-24127.	3.4	40
129	Phosphorylation of Sodium Channel Na_v1.8 by p38 Mitogen-Activated Protein Kinase Increases Current Density in Dorsal Root Ganglion Neurons. Journal of Neuroscience, 2008, 28, 3190-3201.	3.6	156
130	Chapter 4 Genetics and Molecular Pathophysiology of Nav1.7â€Related Pain Syndromes. Advances in Genetics, 2008, 63, 85-110.	1.8	60
131	Diverse Functions and Dynamic Expression of Neuronal Sodium Channels. Novartis Foundation Symposium, 2008, , 34-60.	1.1	21
132	Differential Slow Inactivation and Use-Dependent Inhibition of Nav1.8 Channels Contribute to Distinct Firing Properties in IB4+ and IB4â¯ DRG Neurons. Journal of Neurophysiology, 2007, 97, 1258-1265.	1.8	75
133	From genes to pain: Nav1.7 and human pain disorders. Trends in Neurosciences, 2007, 30, 555-563.	8.6	231
134	Temperature Dependence of Erythromelalgia Mutation L858F in Sodium Channel Nav1.7. Molecular Pain, 2007, 3, 1744-8069-3-3.	2.1	39
135	A Nav1.7 channel mutation associated with hereditary erythromelalgia contributes to neuronal hyperexcitability and displays reduced lidocaine sensitivity. Journal of Physiology, 2007, 581, 1019-1031.	2.9	158
136	Mutations in the sodium channel Nav1.7 underlie inherited erythromelalgia. Drug Discovery Today Disease Mechanisms, 2006, 3, 343-350.	0.8	14
137	Calmodulin Regulates Current Density and Frequency-Dependent Inhibition of Sodium Channel Nav1.8 in DRG Neurons. Journal of Neurophysiology, 2006, 96, 97-108.	1.8	44
138	Differential modulation of sodium channel Nav1.6 by two members of the fibroblast growth factor homologous factorâ€ƒ2 subfamily. European Journal of Neuroscience, 2006, 23, 2551-2562.	2.6	73
139	Sporadic onset of erythermalgia: A gainâ€ofâ€function mutation in Na_v1.7. Annals of Neurology, 2006, 59, 553-558.	5.3	150
140	Inherited erythermalgia: Limb pain from an S4 charge-neutral Na channelopathy. Neurology, 2006, 67, 1563-1567.	1.1	86
141	Size Matters: Erythromelalgia Mutation S241T in Nav1.7 Alters Channel Gating. Journal of Biological Chemistry, 2006, 281, 36029-36035.	3.4	78
142	Na_v1.7 Mutant A863P in Erythromelalgia: Effects of Altered Activation and Steady-State Inactivation on Excitability of Nociceptive Dorsal Root Ganglion Neurons. Journal of Neuroscience, 2006, 26, 12566-12575.	3.6	136
143	A single sodium channel mutation produces hyper- or hypoexcitability in different types of neurons. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 8245-8250.	7.1	350
144	Contactin regulates the current density and axonal expression of tetrodotoxin-resistant but not tetrodotoxin-sensitive sodium channels in DRG neurons. European Journal of Neuroscience, 2005, 22, 39-49.	2.6	75

#	Article	IF	Citations
145	Electrophysiological properties of two axonal sodium channels, Nav1.2 and Nav1.6, expressed in mouse spinal sensory neurones. Journal of Physiology, 2005, 564, 803-815.	2.9	244
146	Pharmacological properties of neuronal TTX-resistant sodium channels and the role of a critical serine pore residue. Pflugers Archiv European Journal of Physiology, 2005, 451, 454-463.	2.8	72
147	Voltage-Gated Sodium Channel Na_v1.6 Is Modulated by p38 Mitogen-Activated Protein Kinase. Journal of Neuroscience, 2005, 25, 6621-6630.	3.6	105
148	Erythermalgia: molecular basis for an inherited pain syndrome. Trends in Molecular Medicine, 2005, 11, 555-562.	6.7	121
149	CAP-1A is a novel linker that binds clathrin and the voltage-gated sodium channel Nav1.8. Molecular and Cellular Neurosciences, 2005, 28, 636-649.	2.2	35
150	Nav 1.6 channels generate resurgent sodium currents in spinal sensory neurons. FEBS Letters, 2005, 579, 2166-2170.	2.8	98
151	Electrophysiological Properties of Mutant Nav1.7 Sodium Channels in a Painful Inherited Neuropathy. Journal of Neuroscience, 2004, 24, 8232-8236.	3.6	353
152	Fibroblast Growth Factor Homologous Factor 2B: Association with Nav1.6 and Selective Colocalization at Nodes of Ranvier of Dorsal Root Axons. Journal of Neuroscience, 2004, 24, 6765-6775.	3.6	124
153	Contactin Associates with Sodium Channel Nav1.3 in Native Tissues and Increases Channel Density at the Cell Surface. Journal of Neuroscience, 2004, 24, 7387-7399.	3.6	52
154	Functional role of the C-terminus of voltage-gated sodium channel Nav1.8. FEBS Letters, 2004, 572, 256-260.	2.8	20
155	Modulation of the Cardiac Sodium Channel Nav1.5 by Fibroblast Growth Factor Homologous Factor 1B. Journal of Biological Chemistry, 2003, 278, 1029-1036.	3.4	140
156	Distinct repriming and closed-state inactivation kinetics of Nav1.6 and Nav1.7 sodium channels in mouse spinal sensory neurons. Journal of Physiology, 2003, 551, 741-750.	2.9	280
157	Critical Molecular Determinants of Voltage-Gated Sodium Channel Sensitivity to Î¼-Conotoxins GIIIA/B. Molecular Pharmacology, 2002, 61, 1192-1201.	2.3	42
158	NaN/Nav1.9: a sodium channel with unique properties. Trends in Neurosciences, 2002, 25, 253-259.	8.6	232
159	Structure of the Sodium Channel Gene SCN11A: Evidence for Intron-to-Exon Conversion Model and Implications for Gene Evolution. Molecular Neurobiology, 2002, 26, 235-250.	4.0	13
160	Nav1.3 Sodium Channels: Rapid Repriming and Slow Closed-State Inactivation Display Quantitative Differences after Expression in a Mammalian Cell Line and in Spinal Sensory Neurons. Journal of Neuroscience, 2001, 21, 5952-5961.	3.6	287
161	Direct Interaction with Contactin Targets Voltage-gated Sodium Channel Nav1.9/NaN to the Cell Membrane. Journal of Biological Chemistry, 2001, 276, 46553-46561.	3.4	76
162	Fibroblast Growth Factor Homologous Factor 1B Binds to the C Terminus of the Tetrodotoxin-resistant Sodium Channel rNav1.9a (NaN). Journal of Biological Chemistry, 2001, 276, 18925-18933.	3.4	111

#	Article	IF	Citations
163	Glial-Derived Neurotrophic Factor Upregulates Expression of Functional SNS and NaN Sodium Channels and Their Currents in Axotomized Dorsal Root Ganglion Neurons. Journal of Neuroscience, 2000, 20, 8754-8761.	3.6	142
164	Changes in Expression of Two Tetrodotoxin-Resistant Sodium Channels and Their Currents in Dorsal Root Ganglion Neurons after Sciatic Nerve Injury But Not Rhizotomy. Journal of Neuroscience, 2000, 20, 7279-7289.	3.6	193
165	A Novel Persistent Tetrodotoxin-Resistant Sodium Current In SNS-Null And Wild-Type Small Primary Sensory Neurons. Journal of Neuroscience, 1999, 19, RC43-RC43.	3.6	396
166	Sodium channels, excitability of primary sensory neurons, and the molecular basis of pain. , 1999, 22, 1177-1187.		180
167	Plasticity of sodium channel expression in DRG neurons in the chronic constriction injury model of neuropathic pain. Pain, 1999, 83, 591-600.	4.2	249
168	Differential role of GDNF and NGF in the maintenance of two TTX-resistant sodium channels in adult DRG neurons. Molecular Brain Research, 1999, 67, 267-282.	2.3	192
169	Sodium channels, excitability of primary sensory neurons, and the molecular basis of pain. Muscle and Nerve, 1999, 22, 1177-1187.	2.2	2
170	SNS Na+ channel expression increases in dorsal root ganglion neurons in the carrageenan inflammatory pain model. NeuroReport, 1998, 9, 967-972.	1.2	219
171	Voltage-Gated Sodium Channels: Multiple Roles in the Pathophysiology of Pain. , 0, , 67-104.		0