Sulayman D Dib-Hajj

List of Publications by Year
in descending order

171 papers	15,493 citations	11639 70 h-index	19726 117 g-index
173 all docs	173 docs citations	173 times ranked	8272 citing authors

#	Article	IF	Citations
1	Sodium Channels in Normal and Pathological Pain. Annual Review of Neuroscience, 2010, 33, 325-347.	5.0	529
2	Gain of function Na_V1.7 mutations in idiopathic small fiber neuropathy. Annals of Neurology, 2012, 71, 26-39.	2.8	518
3	The NaV1.7 sodium channel: from molecule to man. Nature Reviews Neuroscience, 2013, 14, 49-62.	4.9	474
4	The Role of Voltage-Gated Sodium Channels in Pain Signaling. Physiological Reviews, 2019, 99, 1079-1151.	13.1	408
5	A Novel Persistent Tetrodotoxin-Resistant Sodium Current In SNS-Null And Wild-Type Small Primary Sensory Neurons. Journal of Neuroscience, 1999, 19, RC43-RC43.	1.7	396
6	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
7	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.	2.6	365
8	Electrophysiological Properties of Mutant Nav1.7 Sodium Channels in a Painful Inherited Neuropathy. Journal of Neuroscience, 2004, 24, 8232-8236.	1.7	353
9	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.	3.3	350
10	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.	1.7	287
11	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.	1.3	280
12	Plasticity of sodium channel expression in DRG neurons in the chronic constriction injury model of neuropathic pain. Pain, 1999, 83, 591-600.	2.0	249
13	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.	1.3	244
14	Gain-of-function mutations in sodium channel NaV1.9 in painful neuropathy. Brain, 2014, 137, 1627-1642.	3.7	242
15	NaN/Nav1.9: a sodium channel with unique properties. Trends in Neurosciences, 2002, 25, 253-259.	4.2	232
16	From genes to pain: Nav1.7 and human pain disorders. Trends in Neurosciences, 2007, 30, 555-563.	4.2	231
17	Inherited Neuronal Ion Channelopathies: New Windows on Complex Neurological Diseases. Journal of Neuroscience, 2008, 28, 11768-11777.	1.7	225
18	Nav1.8 expression is not restricted to nociceptors in mouse peripheral nervous system. Pain, 2012, 153, 2017-2030.	2.0	223

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19	SNS Na+ channel expression increases in dorsal root ganglion neurons in the carrageenan inflammatory pain model. NeuroReport, 1998, 9, 967-972.	0.6	219
20	Voltage-Gated Sodium Channels: Therapeutic Targets for Pain. Pain Medicine, 2009, 10, 1260-1269.	0.9	200
21	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.	1.7	193
22	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.	2.5	192
23	Sodium channels, excitability of primary sensory neurons, and the molecular basis of pain. , 1999, 22, 1177-1187.		180
24	NaV1.9: a sodium channel linked to human pain. Nature Reviews Neuroscience, 2015, 16, 511-519.	4.9	161
25	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.	1.3	158
26	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.	1.7	156
27	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.	1.0	156
28	Voltage-gated sodium channel expression in rat and human epidermal keratinocytes: Evidence for a role in pain. Pain, 2008, 139, 90-105.	2.0	153
29	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
30	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
31	Sporadic onset of erythermalgia: A gain-of-function mutation in Nav1.7. Annals of Neurology, 2006, 59, 553-558.	2.8	150
32	ERK1/2 Mitogen-Activated Protein Kinase Phosphorylates Sodium Channel Na_v1.7 and Alters Its Gating Properties. Journal of Neuroscience, 2010, 30, 1637-1647.	1.7	149
33	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
34	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.	1.7	142
35	Modulation of the Cardiac Sodium Channel Nav1.5 by Fibroblast Growth Factor Homologous Factor 1B. Journal of Biological Chemistry, 2003, 278, 1029-1036.	1.6	140
36	Nav1.7 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.	1.7	136

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37	A novel Na_v1.7 mutation producing carbamazepineâ€responsive erythromelalgia. Annals of Neurology, 2009, 65, 733-741.	2.8	132
38	Voltage-gated sodium channels in pain states: Role in pathophysiology and targets for treatment. Brain Research Reviews, 2009, 60, 65-83.	9.1	130
39	Role of hippocampal sodium channel Nav1.6 in kindling epileptogenesis. Epilepsia, 2009, 50, 44-55.	2.6	129
40	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.	1.7	124
41	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
42	Erythermalgia: molecular basis for an inherited pain syndrome. Trends in Molecular Medicine, 2005, 11, 555-562.	3.5	121
43	Pharmacological characterisation of the highly NaV1.7 selective spider venom peptide Pn3a. Scientific Reports, 2017, 7, 40883.	1.6	120
44	Early- and late-onset inherited erythromelalgia: genotypeâ€“phenotype correlation. Brain, 2009, 132, 1711-1722.	3.7	117
45	FGF14 N-terminal splice variants differentially modulate Nav1.2 and Nav1.6-encoded sodium channels. Molecular and Cellular Neurosciences, 2009, 42, 90-101.	1.0	117
46	Paroxysmal Extreme Pain Disorder M1627K Mutation in Human Na_v1.7 Renders DRG Neurons Hyperexcitable. Molecular Pain, 2008, 4, 1744-8069-4-37.	1.0	112
47	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.	1.6	111
48	Transfection of rat or mouse neurons by biolistics or electroporation. Nature Protocols, 2009, 4, 1118-1127.	5.5	110
49	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
50	Voltage-Gated Sodium Channel Nav1.6 Is Modulated by p38 Mitogen-Activated Protein Kinase. Journal of Neuroscience, 2005, 25, 6621-6630.	1.7	105
51	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.	1.0	101
52	Nav 1.6 channels generate resurgent sodium currents in spinal sensory neurons. FEBS Letters, 2005, 579, 2166-2170.	1.3	98
53	A Gain-of-Function Mutation in Nav1.6 in a Case of Trigeminal Neuralgia. Molecular Medicine, 2016, 22, 338-348.	1.9	98
54	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

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55	Voltage-clamp and current-clamp recordings from mammalian DRG neurons. Nature Protocols, 2009, 4, 1103-1112.	5.5	94
56	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.	1.0	94
57	Characterization of a de novo SCN8A mutation in a patient with epileptic encephalopathy. Epilepsy Research, 2014, 108, 1511-1518.	0.8	92
58	Sodium Channels in Human Pain Disorders: Genetics and Pharmacogenomics. Annual Review of Neuroscience, 2019, 42, 87-106.	5.0	92
59	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
60	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
61	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
62	Sodium Channel Na_v1.7 Is Essential for Lowering Heat Pain Threshold after Burn Injury. Journal of Neuroscience, 2012, 32, 10819-10832.	1.7	88
63	Structural modelling and mutant cycle analysis predict pharmacoresponsiveness of a Nav1.7 mutant channel. Nature Communications, 2012, 3, 1186.	5.8	88
64	Inherited erythermalgia: Limb pain from an S4 charge-neutral Na channelopathy. Neurology, 2006, 67, 1563-1567.	1.5	86
65	Lacosamide in patients with Nav1.7 mutations-related small fibre neuropathy: a randomized controlled trial. Brain, 2019, 142, 263-275.	3.7	85
66	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.	2.0	80
67	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
68	Size Matters: Erythromelalgia Mutation S241T in Nav1.7 Alters Channel Gating. Journal of Biological Chemistry, 2006, 281, 36029-36035.	1.6	78
69	Screening Fluorescent Voltage Indicators with Spontaneously Spiking HEK Cells. PLoS ONE, 2013, 8, e85221.	1.1	77
70	Direct Interaction with Contactin Targets Voltage-gated Sodium Channel Nav1.9/NaN to the Cell Membrane. Journal of Biological Chemistry, 2001, 276, 46553-46561.	1.6	76
71	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.	1.2	75
72	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.	0.9	75

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73	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.	1.2	73
74	Mexiletine-responsive erythromelalgia due to a new Nav1.7 mutation showing use-dependent current fall-off. Experimental Neurology, 2009, 216, 383-389.	2.0	73
75	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.	1.3	72
76	Inherited erythromelalgia due to mutations inSCN9A:natural history, clinical phenotype and somatosensory profile. Brain, 2016, 139, 1052-1065.	3.7	72
77	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
78	Pharmacotherapy for Pain in a Family With Inherited Erythromelalgia Guided by Genomic Analysis and Functional Profiling. JAMA Neurology, 2016, 73, 659.	4.5	70
79	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
80	Deletion mutation of sodium channel NaV1.7 in inherited erythromelalgia: enhanced slow inactivation modulates dorsal root ganglion neuron hyperexcitability. Brain, 2011, 134, 1972-1986.	3.7	66
81	Conditional knockout of NaV1.6 in adult mice ameliorates neuropathic pain. Scientific Reports, 2018, 8, 3845.	1.6	66
82	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
83	Sodium channel NaV1.9 mutations associated with insensitivity to pain dampen neuronal excitability. Journal of Clinical Investigation, 2017, 127, 2805-2814.	3.9	65
84	Sodium channels in pain disorders: pathophysiology and prospects for treatment. Pain, 2017, 158, S97-S107.	2.0	64
85	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
86	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
87	Chapter 4 Genetics and Molecular Pathophysiology of Nav1.7â€Related Pain Syndromes. Advances in Genetics, 2008, 63, 85-110.	0.8	60
88	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
89	Preferential Targeting of Nav1.6 Voltage-Gated Na+ Channels to the Axon Initial Segment during Development. PLoS ONE, 2015, 10, e0124397.	1.1	59
90	Paroxysmal extreme pain disorder: a molecular lesion of peripheral neurons. Nature Reviews Neurology, 2011, 7, 51-55.	4.9	57

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91	Alternative splicing may contribute to time-dependent manifestation of inherited erythromelalgia. Brain, 2010, 133, 1823-1835.	3.7	56
92	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
93	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.	1.7	52
94	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
95	Two Nedd4-binding Motifs Underlie Modulation of Sodium Channel Nav1.6 by p38 MAPK. Journal of Biological Chemistry, 2010, 285, 26149-26161.	1.6	47
96	Human cells and networks of pain: Transforming pain target identification and therapeutic development. Neuron, 2021, 109, 1426-1429.	3.8	47
97	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
98	Translational pain research: Lessons from genetics and genomics. Science Translational Medicine, 2014, 6, 249sr4.	5.8	45
99	Calmodulin Regulates Current Density and Frequency-Dependent Inhibition of Sodium Channel Nav1.8 in DRG Neurons. Journal of Neurophysiology, 2006, 96, 97-108.	0.9	44
100	Theataxia3Mutation in the N-Terminal Cytoplasmic Domain of Sodium Channel Na_v1.6 Disrupts Intracellular Trafficking. Journal of Neuroscience, 2009, 29, 2733-2741.	1.7	43
101	Critical Molecular Determinants of Voltage-Gated Sodium Channel Sensitivity to Î¼-Conotoxins GIIIA/B. Molecular Pharmacology, 2002, 61, 1192-1201.	1.0	42
102	A new Na_v1.7 sodium channel mutation I234T in a child with severe pain. European Journal of Pain, 2010, 14, 944-950.	1.4	42
103	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.	3.9	41
104	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.	1.6	40
105	A SCN10A SNP biases human pain sensitivity. Molecular Pain, 2016, 12, 174480691666608.	1.0	40
106	Temperature Dependence of Erythromelalgia Mutation L858F in Sodium Channel Nav1.7. Molecular Pain, 2007, 3, 1744-8069-3-3.	1.0	39
107	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
108	A gain-of-function sodium channelÎ²2-subunit mutation in painful diabetic neuropathy. Molecular Pain, 2019, 15, 174480691984980.	1.0	38

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109	Characterization of small fiber pathology in a mouse model of Fabry disease. ELife, 2018, 7, .	2.8	38
110	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.	1.0	35
111	Familial trigeminal neuralgia â€“ a systematic clinical study with a genomic screen of the neuronal electrogenisome. Cephalalgia, 2020, 40, 767-777.	1.8	35
112	Paclitaxel increases axonal localization and vesicular trafficking of Nav1.7. Brain, 2021, 144, 1727-1737.	3.7	35
113	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.	1.0	33
114	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
115	Na_V1.6 regulates excitability of mechanosensitive sensory neurons. Journal of Physiology, 2019, 597, 3751-3768.	1.3	31
116	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
117	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.	1.0	30
118	Network topology of NaV1.7 mutations in sodium channel-related painful disorders. BMC Systems Biology, 2017, 11, 28.	3.0	29
119	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
120	Isoform-specific and pan-channel partners regulate trafficking and plasma membrane stability; and alter sodium channel gating properties. Neuroscience Letters, 2010, 486, 84-91.	1.0	25
121	COL6A5variants in familial neuropathic chronic itch. Brain, 2017, 140, aww343.	3.7	25
122	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	1.0	25
123	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
124	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.	2.1	23
125	Diversity of composition and function of sodium channels in peripheral sensory neurons. Pain, 2015, 156, 2406-2407.	2.0	22
126	Dexpramipexole blocks Nav1.8 sodium channels and provides analgesia in multiple nociceptive and neuropathic pain models. Pain, 2020, 161, 831-841.	2.0	22

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127	Diverse Functions and Dynamic Expression of Neuronal Sodium Channels. Novartis Foundation Symposium, 2008, , 34-60.	1.2	21
128	A new Nav1.7 mutation in an erythromelalgia patient. Biochemical and Biophysical Research Communications, 2013, 432, 99-104.	1.0	21
129	Molecular Architecture of a Sodium Channel S6 Helix. Journal of Biological Chemistry, 2013, 288, 13741-13747.	1.6	21
130	Functional role of the C-terminus of voltage-gated sodium channel Nav1.8. FEBS Letters, 2004, 572, 256-260.	1.3	20
131	Sodium channel Nav1.6 in sensory neurons contributes to vincristine-induced allodynia. Brain, 2020, 143, 2421-2436.	3.7	20
132	Contactin-1 and Neurofascin-155/-186 Are Not Targets of Auto-Antibodies in Multifocal Motor Neuropathy. PLoS ONE, 2015, 10, e0134274.	1.1	19
133	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
134	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
135	A Novel Gain-of-Function Nav1.9 Mutation in a Child With Episodic Pain. Frontiers in Neuroscience, 2019, 13, 918.	1.4	18
136	Pediatric Erythromelalgia and SCN9A Mutations: Systematic Review and Single-Center Case Series. Journal of Pediatrics, 2019, 206, 217-224.e9.	0.9	18
137	Evaluation of molecular inversion probe versus TruSeqÂ® custom methods for targeted next-generation sequencing. PLoS ONE, 2020, 15, e0238467.	1.1	17
138	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
139	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
140	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
141	Mutations in the sodium channel Nav1.7 underlie inherited erythromelalgia. Drug Discovery Today Disease Mechanisms, 2006, 3, 343-350.	0.8	14
142	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
143	Pharmacological characterization of a rat Nav1.7 loss-of-function model with insensitivity to pain. Pain, 2020, 161, 1350-1360.	2.0	14
144	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.	1.9	13

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145	KCNQ variants and pain modulation: a missense variant in Kv7.3 contributes to pain resilience. Brain Communications, 2021, 3, fcab212.	1.5	13
146	Neuropathic pain in two-generation twins carrying the sodium channel Nav1.7 functional variant R1150W. Pain, 2014, 155, 2199-2203.	2.0	12
147	Somatosensory Neurons Enter a State of Altered Excitability during Hibernation. Current Biology, 2018, 28, 2998-3004.e3.	1.8	12
148	Therapeutic potential of Pak1 inhibition for pain associated with cutaneous burn injury. Molecular Pain, 2018, 14, 174480691878864.	1.0	12
149	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
150	Multiple myosin motors interact with sodium/potassium-ATPase alpha 1 subunits. Molecular Brain, 2018, 11, 45.	1.3	11
151	The Two Sides of NaV1.7: Painful and Painless Channelopathies. Neuron, 2019, 101, 765-767.	3.8	10
152	Trigeminal Neuralgia TRPM8 Mutation. Neurology: Genetics, 2021, 7, e550.	0.9	10
153	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
154	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
155	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
156	Nonmuscle myosin II isoforms interact with sodium channel alpha subunits. Molecular Pain, 2018, 14, 174480691878863.	1.0	7
157	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
158	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
159	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
160	Depolarizing Na_V and Hyperpolarizing K_V Channels Are Co-Trafficked in Sensory Neurons. Journal of Neuroscience, 2022, 42, 4794-4811.	1.7	6
161	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
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