Mohamed Chahine

List of Publications by Year
in descending order

135 papers	4,618 citations	94433 37 h-index	123424 61 g-index
140 all docs	140 docs citations	140 times ranked	4370 citing authors

#	Article	IF	Citations
1	Antisense oligonucleotides as a potential treatment for brain deficits observed in myotonic dystrophy type 1. Gene Therapy, 2022, 29, 698-709.	4.5	20
2	NPRL2 Inhibition of mTORC1 Controls Sodium Channel Expression and Brain Amino Acid Homeostasis. ENeuro, 2022, 9, ENEURO.0317-21.2022.	1.9	5
3	Genetic associations of protein-coding variants in human disease. Nature, 2022, 603, 95-102.	27.8	67
4	Racial Disparities in Ion Channelopathies and Inherited Cardiovascular Diseases Associated With Sudden Cardiac Death. Journal of the American Heart Association, 2022, 11, e023446.	3.7	6
5	SCN2A-related epilepsy of infancy with migrating focal seizures: report of a variant with apparent gain- and loss-of-function effects. Journal of Neurophysiology, 2022, 127, 1388-1397.	1.8	6
6	Novel G1481V and Q1491H SCN5A Mutations Linked to Long QT Syndrome Destabilize the Nav1.5 Inactivation State. CJC Open, 2021, 3, 256-266.	1.5	3
7	iPSC-derived cardiomyocytes from patients with myotonic dystrophy type 1 have abnormal ion channel functions and slower conduction velocities. Scientific Reports, 2021, 11, 2500.	3.3	17
8	Lymphoblastoid-derived human-induced pluripotent stem cells. , 2021, , 57-70.		2
9	R1617Q epilepsy mutation slows Na V 1.6Âsodium channel inactivation and increases the persistent current and neuronal firing. Journal of Physiology, 2021, 599, 1651-1664.	2.9	8
10	NaV1.5 knockout in iPSCs: a novel approach to study NaV1.5 variants in a human cardiomyocyte environment. Scientific Reports, 2021, 11, 17168.	3.3	8
11	The myocardial and neuronal infectivity of SARS-CoV-2 and detrimental outcomes. Canadian Journal of Physiology and Pharmacology, 2021, 99, 1128-1136.	1.4	1
12	Deciphering the mechanisms underlying brain alterations and cognitive impairment in congenital myotonic dystrophy. Neurobiology of Disease, 2021, 160, 105532.	4.4	7
13	Exome Sequencing Implicates Impaired GABA Signaling and Neuronal Ion Transport in Trigeminal Neuralgia. IScience, 2020, 23, 101552.	4.1	32
14	Novel re-expression of L-type calcium channel Cav1.3 in left ventricles of failing human heart. Heart Rhythm, 2020, 17, 1193-1197.	0.7	7
15	Differentiation of lymphoblastoid-derived iPSCs into functional cardiomyocytes, neurons and myoblasts. Biochemical and Biophysical Research Communications, 2019, 516, 222-228.	2.1	9
16	Biophysical and Molecular Characterization of Calcium Permeable Honeybee DSC1 (AmCaV4) Channel Expressed in Mammalian Cells. Biophysical Journal, 2019, 116, 390a.	0.5	0
17	Voltage-gated sodium channels from the bees Apis mellifera and Bombus terrestris are differentially modulated by pyrethroid insecticides. Scientific Reports, 2019, 9, 1078.	3.3	7
18	A Novel PITX2c Gain-of-Function Mutation, p.Met207Val, in Patients With Familial Atrial Fibrillation. American Journal of Cardiology, 2019, 123, 787-793.	1.6	18

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19	Substitutions of the S4DIV R2 residue (R1451) in NaV1.4 lead to complex forms of paramyotonia congenita and periodic paralyses. Scientific Reports, 2018, 8, 2041.	3.3	14
20	Interleukin-6 inhibition of hERG underlies risk for acquired long QT in cardiac and systemic inflammation. PLoS ONE, 2018, 13, e0208321.	2.5	105
21	A204E mutation in Nav1.4 DIS3 exerts gain- and loss-of-function effects that lead to periodic paralysis combining hyper- with hypo-kalaemic signs. Scientific Reports, 2018, 8, 16681.	3.3	5
22	A New Cardiac Channelopathy: From Clinical Phenotypes to Molecular Mechanisms Associated With Nav1.5 Gating Pores. Frontiers in Cardiovascular Medicine, 2018, 5, 139.	2.4	19
23	Biophysical Characterization of Two NaV1.4 Mutations Making a Clinical Overlap between the Myotonia-Hyperkalemic and Hypokalemic Periodic Paralysis Clusters of Disorders. Biophysical Journal, 2018, 114, 632a.	0.5	0
24	A leaky voltage sensor domain of cardiac sodium channels causes arrhythmias associated with dilated cardiomyopathy. Scientific Reports, 2018, 8, 13804.	3.3	28
25	Improving the characterization of calcium channel gating pore currents with Stac3. Journal of General Physiology, 2018, 150, 375-378.	1.9	2
26	Biophysical characterization of the Varroa destructor Na _V 1 sodium channel and its affinity for Ï„â€fluvalinate insecticide. FASEB Journal, 2017, 31, 3066-3071.	0.5	10
27	Induced pluripotent stem-cell-derived cardiomyocytes: cardiac applications, opportunities, and challenges. Canadian Journal of Physiology and Pharmacology, 2017, 95, 1108-1116.	1.4	8
28	Regulation of Cardiac Voltage-Gated Sodium Channel by Kinases: Roles of Protein Kinases A and C. Handbook of Experimental Pharmacology, 2017, 246, 161-184.	1.8	13
29	A wireless system for combined heart optogenetics and electrocardiography recording. , 2017, , .		4
30	Metaflumizone inhibits the honeybee Na_V1 channel by targeting recovery from slow inactivation. FEBS Letters, 2017, 591, 3842-3849.	2.8	4
31	Biophysical, Molecular, and Pharmacological Characterization of Voltage-Dependent Sodium Channels From Induced Pluripotent Stem Cell-Derived Cardiomyocytes. Canadian Journal of Cardiology, 2017, 33, 269-278.	1.7	19
32	Editorial: Recent Advances in Voltage-Gated Sodium Channels, their Pharmacology and Related Diseases. Frontiers in Pharmacology, 2016, 7, 20.	3.5	11
33	Biophysical Characterization of the Honeybee's DSC1 Ortholog Highlights a New Voltage Dependant Calcium Channel Subfamily. Biophysical Journal, 2016, 110, 34a.	0.5	0
34	Induction of autoimmune response to the extracellular loop of the HERG channel pore induces QTc prolongation in guineaâ€pigs. Journal of Physiology, 2016, 594, 6175-6187.	2.9	19
35	Biophysical characterization of the honeybee DSC1 orthologue reveals a novel voltage-dependent Ca2+ channel subfamily: CaV4. Journal of General Physiology, 2016, 148, 133-145.	1.9	13
36	A recessive Na _v 1.4 mutation underlies congenital myasthenic syndrome with periodic paralysis. Neurology, 2016, 86, 161-169.	1.1	49

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37	Characterization of the honeybee AmNaV1 channel and tools to assess the toxicity of insecticides. Scientific Reports, 2015, 5, 12475.	3.3	19
38	Unmasked Brugada Pattern by Ajmaline Challenge in Patients with Myotonic Dystrophy Type 1. , 2015, 20, 28-36.		8
39	MTSET modification of D4S6 cysteines stabilize the fast inactivated state of Nav1.5 sodium channels. Frontiers in Pharmacology, 2015, 6, 118.	3.5	3
40	Mutations in the Voltage Sensors of Domains I and II of Nav1.5 that are Associated with Arrhythmias and Dilated Cardiomyopathy Generate Gating Pore Currents. Frontiers in Pharmacology, 2015, 6, 301.	3.5	38
41	Effects of amlodipine and perindoprilate on the structure and function of mitochondria in ventricular cardiomyocytes during ischemiaâ€reperfusion in the pig. Fundamental and Clinical Pharmacology, 2015, 29, 21-30.	1.9	7
42	Pathogenesis of the Novel Autoimmune-Associated Long-QT Syndrome. Circulation, 2015, 132, 230-240.	1.6	62
43	Gating pore currents are defects in common with two Nav1.5 mutations in patients with mixed arrhythmias and dilated cardiomyopathy. Journal of General Physiology, 2015, 145, 93-106.	1.9	64
44	Molecular characterization and functional expression of the Apis mellifera voltage-dependent Ca2+ channels. Insect Biochemistry and Molecular Biology, 2015, 58, 12-27.	2.7	18
45	Differential modulation of Nav1.7 and Nav1.8 channels by antidepressant drugs. European Journal of Pharmacology, 2015, 764, 395-403.	3.5	18
46	Gating pore currents, a new pathological mechanism underlying cardiac arrhythmias associated with dilated cardiomyopathy. Channels, 2015, 9, 139-144.	2.8	12
47	Gating pore current is a novel biophysical defect of Nav1.5 mutations associated with unusual cardiac arrhythmias and dilation. Future Cardiology, 2015, 11, 287-291.	1.2	0
48	Biophysics, pathophysiology, and pharmacology of ion channel gating pores. Frontiers in Pharmacology, 2014, 5, 53.	3.5	74
49	Correlation of the electrophysiological profiles and sodium channel transcripts of individual rat dorsal root ganglia neurons. Frontiers in Cellular Neuroscience, 2014, 8, 285.	3.7	7
50	Na_v1.5 mutations linked to dilated cardiomyopathy phenotypes. Channels, 2014, 8, 90-94.	2.8	33
51	Fluoxetine Blocks Na_v1.5 Channels via a Mechanism Similar to That of Class 1 Antiarrhythmics. Molecular Pharmacology, 2014, 86, 378-389.	2.3	25
52	Molecular biology and biophysical properties of ion channel gating pores. Quarterly Reviews of Biophysics, 2014, 47, 364-388.	5.7	23
53	Pyridoxal-5â€²-phosphate (MC-1), a vitamin B6 derivative, inhibits expressed P2X receptors. Canadian Journal of Physiology and Pharmacology, 2014, 92, 189-196.	1.4	29
54	Modulation of peripheral Na+ channels and neuronal firing by n-butyl-p-aminobenzoate. European Journal of Pharmacology, 2014, 727, 158-166.	3.5	3

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55	The variant hERG/R148W associated with LQTS is a mutation that reduces current density on co-expression with the WT. Gene, 2014, 536, 348-356.	2.2	7
56	Myotonic dystrophy type 1 mimics and exacerbates Brugada phenotype induced by Nav1.5 sodium channel loss-of-function mutation. Heart Rhythm, 2014, 11, 1393-1400.	0.7	19
57	Regulation/Modulation of Sensory Neuron Sodium Channels. Handbook of Experimental Pharmacology, 2014, 221, 111-135.	1.8	35
58	Pyrethroids Differentially Alter Voltage-Gated Sodium Channels from the Honeybee Central Olfactory Neurons. PLoS ONE, 2014, 9, e112194.	2.5	19
59	Novel SCN5A mutations in two families with â€œBrugada-likeâ€•ST elevation in the inferior leads and conduction disturbances. Journal of Interventional Cardiac Electrophysiology, 2013, 37, 131-140.	1.3	11
60	Characterization of the first honeybee Ca2+ channel subunit reveals two novel species- and splicing-specific modes of regulation of channel inactivation. Pflugers Archiv European Journal of Physiology, 2013, 465, 985-996.	2.8	11
61	Investigating the Voltage Sensor Domains of Nav1.4, its Structural and Functional Properties via Histidine Scanning Mutagenesis. Biophysical Journal, 2013, 104, 133a.	0.5	0
62	Recent advances in voltage-gated sodium channels, their pharmacology, and related diseases. Frontiers in Pharmacology, 2013, 4, 52.	3.5	1
63	Sodium overload due to a persistent current that attenuates the arrhythmogenic potential of a novel LQT3 mutation. Frontiers in Pharmacology, 2013, 4, 126.	3.5	18
64	Differential Expression of Sodium Channel Î² Subunits in Dorsal Root Ganglion Sensory Neurons. Journal of Biological Chemistry, 2012, 287, 15044-15053.	3.4	31
65	A distinct de novo expression of Na<sub>v</sub1.5 sodium channels in human atrial fibroblasts differentiated into myofibroblasts. Journal of Physiology, 2012, 590, 4307-4319.	2.9	77
66	Gating pore currents and the resting state of Na _v 1.4 voltage sensor domains. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 19250-19255.	7.1	71
67	Biophysical characterization of M1476I, a sodium channel founder mutation associated with coldâ€induced myotonia in French Canadians. Journal of Physiology, 2012, 590, 2629-2644.	2.9	11
68	The Î²1-Subunit of Nav1.5 Cardiac Sodium Channel Is Required for a Dominant Negative Effect through Î±-Î± Interaction. PLoS ONE, 2012, 7, e48690.	2.5	29
69	Mexiletine Differentially Restores the Trafficking Defects Caused by Two Brugada Syndrome Mutations. Frontiers in Pharmacology, 2012, 3, 62.	3.5	29
70	Sudden Death of Cardiac Origin and Psychotropic Drugs. Frontiers in Pharmacology, 2012, 3, 76.	3.5	30
71	Coexisting mutations/polymorphisms of the long QT syndrome genes in patients with repaired Tetralogy of Fallot are associated with the risks of life-threatening events. Human Genetics, 2012, 131, 1295-1304.	3.8	19
72	A Proton Leak Current through the Cardiac Sodium Channel Is Linked to Mixed Arrhythmia and the Dilated Cardiomyopathy Phenotype. PLoS ONE, 2012, 7, e38331.	2.5	84

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73	New Insights into Cardiac and Brain Sodium Channels Modulation by Beta Blockers. Frontiers in Pharmacology, 2011, 2, 1.	3.5	97
74	Regulatory role of voltage-gated Na+ channel Î² subunits in sensory neurons. Frontiers in Pharmacology, 2011, 2, 70.	3.5	40
75	Regulation of Na_v1.6 and Na_v1.8 peripheral nerve Na⁺ channels by auxiliary Î²-subunits. Journal of Neurophysiology, 2011, 106, 608-619.	1.8	49
76	Perinatal and Postnatal Expression of Cav1.3 Î±1D Ca2+ Channel in the Rat Heart. Pediatric Research, 2011, 69, 479-484.	2.3	22
77	Y1767C, a novel SCN5A mutation, induces a persistent Na⁺ current and potentiates ranolazine inhibition of Na_v1.5 channels. American Journal of Physiology - Heart and Circulatory Physiology, 2011, 300, H288-H299.	3.2	42
78	Biophysical characterisation of the persistent sodium current of the Nav1.6 neuronal sodium channel: a single-channel analysis. Pflugers Archiv European Journal of Physiology, 2010, 460, 77-86.	2.8	47
79	Cell Membrane Expression of Cardiac Sodium Channel Na_v1.5 Is Modulated by Î±-Actinin-2 Interaction. Biochemistry, 2010, 49, 166-178.	2.5	57
80	Congenital heart block: Identification of autoantibody binding site on the extracellular loop (domain) Tj ETQq0 0 0 rgBT /Overlock 10 Tf	6.5	59
81	Phosphorylation of the Consensus Sites of Protein Kinase A on Î±1D L-type Calcium Channel. Journal of Biological Chemistry, 2009, 284, 5042-5049.	3.4	20
82	Biophysical characterization of a new SCN5A mutation S1333Y in a SIDS infant linked to long QT syndrome. FEBS Letters, 2009, 583, 890-896.	2.8	15
83	Contribution of Long-QT Syndrome Genetic Variants in Sudden Infant Death Syndrome. Pediatric Cardiology, 2009, 30, 502-509.	1.3	62
84	Gain-of-function mutation of Nav1.5 in atrial fibrillation enhances cellular excitability and lowers the threshold for action potential firing. Biochemical and Biophysical Research Communications, 2009, 380, 132-137.	2.1	105
85	Cardiac Metabolic State and Brugada Syndrome. Circulation Research, 2009, 105, 721-723.	4.5	2
86	Characterization of novel KCNH2 mutations in type 2 long QT syndrome manifesting as seizures. Canadian Journal of Cardiology, 2009, 25, 455-462.	1.7	26
87	Protein kinase C activation inhibits Î±1D L-type Ca channel: A single-channel analysis. Pflugers Archiv European Journal of Physiology, 2008, 455, 913-919.	2.8	12
88	Changes in action potentials and intracellular ionic homeostasis in a ventricular cell model related to a persistent sodium current in SCN5A mutations underlying LQT3. Progress in Biophysics and Molecular Biology, 2008, 96, 281-293.	2.9	6
89	A new C-terminal hERG mutation A915fs+47X associated with symptomatic LQT2 and auditory-trigger syncope. Heart Rhythm, 2008, 5, 1577-1586.	0.7	13
90	In utero onset of long QT syndrome with atrioventricular block and spontaneous or lidocaine-induced ventricular tachycardia: Compound effects of hERG pore region mutation and SCN5A N-terminus variant. Heart Rhythm, 2008, 5, 1567-1574.	0.7	20

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91	Enzyme Domain Affects the Movement of the Voltage Sensor in Ascidian and Zebrafish Voltage-sensing Phosphatases. Journal of Biological Chemistry, 2008, 283, 18248-18259.	3.4	108
92	Biophysical Properties of Human Na_v1.7 Splice Variants and Their Regulation by Protein Kinase A. Journal of Neurophysiology, 2008, 99, 2241-2250.	1.8	48
93	Voltage-Gated Sodium Channels in Neurological Disorders. CNS and Neurological Disorders - Drug Targets, 2008, 7, 144-158.	1.4	34
94	The occurrence of Brugada syndrome and isolated cardiac conductive disease in the same family could be due to a single SCN5A mutation or to the accidental association of both diseases. Europace, 2007, 10, 79-85.	1.7	13
95	Expression of skeletal muscle NaV1.4 Na channel isoform in canine cardiac Purkinje myocytes. Biochemical and Biophysical Research Communications, 2007, 355, 28-33.	2.1	16
96	A novel mutation in the SCN5A gene is associated with Brugada syndrome. Life Sciences, 2007, 80, 716-724.	4.3	14
97	The Brugada syndrome in Canada: A unique French-Canadian experience. Canadian Journal of Cardiology, 2007, 23, 71B-75B.	1.7	19
98	Acidic Residues on the Voltage-Sensor Domain Determine the Activation of the NaChBac Sodium Channel. Biophysical Journal, 2007, 92, 3513-3523.	0.5	27
99	Accessibility of Four Arginine Residues on the S4 Segment of the Bacillus halodurans Sodium Channel. Journal of Membrane Biology, 2007, 215, 169-180.	2.1	17
100	Lidocaine Promotes the Trafficking and Functional Expression of Nav1.8 Sodium Channels in Mammalian Cells. Journal of Neurophysiology, 2007, 98, 467-477.	1.8	20
101	Nav1.5/R1193Q polymorphism is associated with both long QT and Brugada syndromes. Canadian Journal of Cardiology, 2006, 22, 309-313.	1.7	47
102	A novel SCN5A mutation, F1344S, identified in a patient with Brugada syndrome and fever-induced ventricular fibrillation. Cardiovascular Research, 2006, 70, 521-529.	3.8	72
103	Protein kinase C activation inhibits Cav1.3 calcium channel at NH2-terminal serine 81 phosphorylation site. American Journal of Physiology - Heart and Circulatory Physiology, 2006, 291, H1614-H1622.	3.2	24
104	Clinical aspects and physiopathology of Brugada syndrome: review of current concepts. Canadian Journal of Physiology and Pharmacology, 2006, 84, 795-802.	1.4	11
105	SCN5A Polymorphism Restores Trafficking of a Brugada Syndrome Mutation on a Separate Gene. Circulation, 2006, 114, 368-376.	1.6	187
106	Regulation of Nav channels in sensory neurons. Trends in Pharmacological Sciences, 2005, 26, 496-502.	8.7	66
107	A novel nonsense mutation in the SCN5A gene leads to Brugada syndrome and a silent gene mutation carrier state. Canadian Journal of Cardiology, 2005, 21, 925-31.	1.7	22
108	A novel SCN5A mutation manifests as a malignant form of long QT syndrome with perinatal onset of tachycardia/bradycardia. Cardiovascular Research, 2004, 64, 268-278.	3.8	70

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109	Differential modulation of Nav 1.7 and Nav 1.8 peripheral nerve sodium channels by the local anesthetic lidocaine. British Journal of Pharmacology, 2004, 142, 576-584.	5.4	96
110	Genetic analysis of the cardiac sodium channel gene SCN5A in Koreans with Brugada syndrome. Journal of Human Genetics, 2004, 49, 573-578.	2.3	19
111	Role of Arginine Residues on the S4 Segment of the Bacillus halodurans Na+ Channel in Voltage-sensing. Journal of Membrane Biology, 2004, 201, 9-24.	2.1	35
112	Role of auxiliary $beta;1-, $beta;2-, and $beta;3-subunits and their interaction with Nav1.8 voltage-gated sodium channel*1. Biochemical and Biophysical Research Communications, 2004, 319, 531-531.	2.1	0
113	Role of auxiliary Î²1-, Î²2-, and Î²3-subunits and their interaction with Nav1.8 voltage-gated sodium channel. Biochemical and Biophysical Research Communications, 2004, 319, 531-540.	2.1	47
114	The C-terminal region as a modulator of rNav1.7 and rNav1.8 expression levels. FEBS Letters, 2004, 559, 39-44.	2.8	7
115	Modulation of Nav1.7 and Nav1.8 Peripheral Nerve Sodium Channels by Protein Kinase A and Protein Kinase C. Journal of Neurophysiology, 2004, 91, 1556-1569.	1.8	111
116	Loss of function associated with novel mutations of the SCN5A gene in patients with Brugada syndrome. Canadian Journal of Cardiology, 2004, 20, 425-30.	1.7	26
117	A Newly Characterized SCN5A Mutation Underlying Brugada Syndrome Unmasked by Hyperthermia. Journal of Cardiovascular Electrophysiology, 2003, 14, 407-411.	1.7	103
118	A novel mutation in SCN5A, delQKP 1507â€“1509, causing long QT syndrome: Role of Q1507 residue in sodium channel inactivation. Journal of Molecular and Cellular Cardiology, 2003, 35, 1513-1521.	1.9	42
119	Biophysical characteristics of a new mutation on the KCNQ1 potassium channel (L251P) causing long QT syndrome. Canadian Journal of Physiology and Pharmacology, 2003, 81, 129-134.	1.4	10
120	Modulation of L-type Ca2+ channels in neonatal rat heart by a novel Ca2+ channel agonist. Canadian Journal of Physiology and Pharmacology, 2003, 81, 135-141.	1.4	0
121	Expression and Intracellular Localization of anSCN5ADouble Mutant R1232W/T1620M Implicated in Brugada Syndrome. Circulation Research, 2002, 90, .	4.5	142
122	A tryptophan residue (W736) in the amino-terminus of the P-segment of domain II is involved in pore formation in Na ν 1.4 voltage-gated sodium channels. Pflugers Archiv European Journal of Physiology, 2002, 445, 18-24.	2.8	6
123	Expression and intracellular localization of an SCN5A double mutant R1232W/T1620M implicated in Brugada syndrome. Circulation Research, 2002, 90, E11-6.	4.5	29
124	Gating Properties of Na_v1.7 and Na_v1.8 Peripheral Nerve Sodium Channels. Journal of Neuroscience, 2001, 21, 7909-7918.	3.6	88
125	Ethanol delays and reverses lysophosphatidylcholine-induced calcium overload in neonatal rat heart cells. Pflugers Archiv European Journal of Physiology, 2001, 443, 48-53.	2.8	4
126	Novel Mechanism for Brugada Syndrome. Circulation Research, 2001, 88, E78-83.	4.5	116

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127	SCN5A mutation (T1620M) causing Brugada syndrome exhibits different phenotypes when expressed in Xenopus oocytes and mammalian cells. FEBS Letters, 2000, 467, 12-16.	2.8	65
128	Biophysical phenotypes of SCN5A mutations causing long QT and Brugada syndromes. FEBS Letters, 2000, 487, 224-228.	2.8	69
129	Cysteine scanning analysis of the IFM cluster in the inactivation gate of a human heart sodium channel. Cardiovascular Research, 1999, 42, 521-529.	3.8	12
130	Modulation of HERG potassium channel properties by external pH. Pflugers Archiv European Journal of Physiology, 1999, 438, 419-422.	2.8	32
131	Extrapore Residues of the S5-S6 Loop of Domain 2 of the Voltage-Gated Skeletal Muscle Sodium Channel (rSkM1) Contribute to the Î¼-Conotoxin GIIIA Binding Site. Biophysical Journal, 1998, 75, 236-246.	0.5	54
132	Restoration of Fast Inactivation in an Inactivation-Defective Human Heart Sodium Channel by the Cysteine Modifying Reagent Benzyl-MTS: Analysis of IFM-ICM Mutation. Biochemical and Biophysical Research Communications, 1997, 233, 606-610.	2.1	11
133	Myotonic dystrophy kinase modulates skeletal muscle but not cardiac voltage-gated sodium channels. FEBS Letters, 1997, 412, 621-624.	2.8	22
134	Functional expression and properties of the human skeletal muscle sodium channel. Pflugers Archiv European Journal of Physiology, 1994, 427, 136-142.	2.8	114
135	Sodium channel mutations in paramyotonia congenita uncouple inactivation from activation. Neuron, 1994, 12, 281-294.	8.1	341