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	Sodium channel mutations in paramyotonia congenita uncouple inactivation from activation. Neuron, 1994, 12, 281-294.	8.1	341
2	SCN5A Polymorphism Restores Trafficking of a Brugada Syndrome Mutation on a Separate Gene. Circulation, 2006, 114, 368-376.	1.6	187
3	Expression and Intracellular Localization of anSCN5ADouble Mutant R1232W/T1620M Implicated in Brugada Syndrome. Circulation Research, 2002, 90, .	4.5	142
4	Novel Mechanism for Brugada Syndrome. Circulation Research, 2001, 88, E78-83.	4.5	116
5	Functional expression and properties of the human skeletal muscle sodium channel. Pflugers Archiv European Journal of Physiology, 1994, 427, 136-142.	2.8	114
6	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
7	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
8	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
9	Interleukin-6 inhibition of hERG underlies risk for acquired long QT in cardiac and systemic inflammation. PLoS ONE, 2018, 13, e0208321.	2.5	105
10	A Newly Characterized SCN5A Mutation Underlying Brugada Syndrome Unmasked by Hyperthermia. Journal of Cardiovascular Electrophysiology, 2003, 14, 407-411.	1.7	103
11	New Insights into Cardiac and Brain Sodium Channels Modulation by Beta Blockers. Frontiers in Pharmacology, 2011, 2, 1.	3.5	97
12	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
13	Gating Properties of Na_v1.7 and Na_v1.8 Peripheral Nerve Sodium Channels. Journal of Neuroscience, 2001, 21, 7909-7918.	3.6	88
14	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
15	A distinct de novo expression of Na_v1.5 sodium channels in human atrial fibroblasts differentiated into myofibroblasts. Journal of Physiology, 2012, 590, 4307-4319.	2.9	77
16	Biophysics, pathophysiology, and pharmacology of ion channel gating pores. Frontiers in Pharmacology, 2014, 5, 53.	3.5	74
17	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
18	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

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19	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
20	Biophysical phenotypes of SCN5A mutations causing long QT and Brugada syndromes. FEBS Letters, 2000, 487, 224-228.	2.8	69
21	Genetic associations of protein-coding variants in human disease. Nature, 2022, 603, 95-102.	27.8	67
22	Regulation of Nav channels in sensory neurons. Trends in Pharmacological Sciences, 2005, 26, 496-502.	8.7	66
23	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
24	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
25	Contribution of Long-QT Syndrome Genetic Variants in Sudden Infant Death Syndrome. Pediatric Cardiology, 2009, 30, 502-509.	1.3	62
26	Pathogenesis of the Novel Autoimmune-Associated Long-QT Syndrome. Circulation, 2015, 132, 230-240.	1.6	62
27	Congenital heart block: Identification of autoantibody binding site on the extracellular loop (domain) Tj ETQq1 1 0.784314 rgBT /Over	6.5	59
28	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
29	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
30	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
31	A recessive Na _v 1.4 mutation underlies congenital myasthenic syndrome with periodic paralysis. Neurology, 2016, 86, 161-169.	1.1	49
32	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
33	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
34	Nav1.5/R1193Q polymorphism is associated with both long QT and Brugada syndromes. Canadian Journal of Cardiology, 2006, 22, 309-313.	1.7	47
35	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
36	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

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37	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
38	Regulatory role of voltage-gated Na+ channel Î² subunits in sensory neurons. Frontiers in Pharmacology, 2011, 2, 70.	3.5	40
39	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
40	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
41	Regulation/Modulation of Sensory Neuron Sodium Channels. Handbook of Experimental Pharmacology, 2014, 221, 111-135.	1.8	35
42	Voltage-Gated Sodium Channels in Neurological Disorders. CNS and Neurological Disorders - Drug Targets, 2008, 7, 144-158.	1.4	34
43	Na_v1.5 mutations linked to dilated cardiomyopathy phenotypes. Channels, 2014, 8, 90-94.	2.8	33
44	Modulation of HERG potassium channel properties by external pH. Pflugers Archiv European Journal of Physiology, 1999, 438, 419-422.	2.8	32
45	Exome Sequencing Implicates Impaired GABA Signaling and Neuronal Ion Transport in Trigeminal Neuralgia. IScience, 2020, 23, 101552.	4.1	32
46	Differential Expression of Sodium Channel Î² Subunits in Dorsal Root Ganglion Sensory Neurons. Journal of Biological Chemistry, 2012, 287, 15044-15053.	3.4	31
47	Sudden Death of Cardiac Origin and Psychotropic Drugs. Frontiers in Pharmacology, 2012, 3, 76.	3.5	30
48	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
49	Mexiletine Differentially Restores the Trafficking Defects Caused by Two Brugada Syndrome Mutations. Frontiers in Pharmacology, 2012, 3, 62.	3.5	29
50	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
51	Expression and intracellular localization of an SCN5A double mutant R1232W/T1620M implicated in Brugada syndrome. Circulation Research, 2002, 90, E11-6.	4.5	29
52	A leaky voltage sensor domain of cardiac sodium channels causes arrhythmias associated with dilated cardiomyopathy. Scientific Reports, 2018, 8, 13804.	3.3	28
53	Acidic Residues on the Voltage-Sensor Domain Determine the Activation of the NaChBac Sodium Channel. Biophysical Journal, 2007, 92, 3513-3523.	0.5	27
54	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

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55	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
56	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
57	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
58	Molecular biology and biophysical properties of ion channel gating pores. Quarterly Reviews of Biophysics, 2014, 47, 364-388.	5.7	23
59	Myotonic dystrophy kinase modulates skeletal muscle but not cardiac voltage-gated sodium channels. FEBS Letters, 1997, 412, 621-624.	2.8	22
60	Perinatal and Postnatal Expression of Cav1.3 Î±1D Ca2+ Channel in the Rat Heart. Pediatric Research, 2011, 69, 479-484.	2.3	22
61	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
62	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
63	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
64	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
65	Antisense oligonucleotides as a potential treatment for brain deficits observed in myotonic dystrophy type 1. Gene Therapy, 2022, 29, 698-709.	4.5	20
66	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
67	The Brugada syndrome in Canada: A unique French-Canadian experience. Canadian Journal of Cardiology, 2007, 23, 71B-75B.	1.7	19
68	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
69	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
70	Characterization of the honeybee AmNaV1 channel and tools to assess the toxicity of insecticides. Scientific Reports, 2015, 5, 12475.	3.3	19
71	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
72	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

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73	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
74	Pyrethroids Differentially Alter Voltage-Gated Sodium Channels from the Honeybee Central Olfactory Neurons. PLoS ONE, 2014, 9, e112194.	2.5	19
75	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
76	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
77	Differential modulation of Nav1.7 and Nav1.8 channels by antidepressant drugs. European Journal of Pharmacology, 2015, 764, 395-403.	3.5	18
78	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
79	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
80	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
81	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
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	A novel mutation in the SCN5A gene is associated with Brugada syndrome. Life Sciences, 2007, 80, 716-724.	4.3	14
84	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
85	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
86	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
87	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
88	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
89	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
90	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

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91	Gating pore currents, a new pathological mechanism underlying cardiac arrhythmias associated with dilated cardiomyopathy. Channels, 2015, 9, 139-144.	2.8	12
92	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
93	Clinical aspects and physiopathology of Brugada syndrome: review of current concepts. Canadian Journal of Physiology and Pharmacology, 2006, 84, 795-802.	1.4	11
94	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
95	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
96	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
97	Editorial: Recent Advances in Voltage-Gated Sodium Channels, their Pharmacology and Related Diseases. Frontiers in Pharmacology, 2016, 7, 20.	3.5	11
98	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
99	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
100	Differentiation of lymphoblastoid-derived iPSCs into functional cardiomyocytes, neurons and myoblasts. Biochemical and Biophysical Research Communications, 2019, 516, 222-228.	2.1	9
101	Unmasked Brugada Pattern by Ajmaline Challenge in Patients with Myotonic Dystrophy Type 1. , 2015, 20, 28-36.		8
102	Induced pluripotent stem-cell-derived cardiomyocytes: cardiac applications, opportunities, and challenges. Canadian Journal of Physiology and Pharmacology, 2017, 95, 1108-1116.	1.4	8
103	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
104	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
105	The C-terminal region as a modulator of rNav1.7 and rNav1.8 expression levels. FEBS Letters, 2004, 559, 39-44.	2.8	7
106	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
107	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
108	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

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109	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
110	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
111	Deciphering the mechanisms underlying brain alterations and cognitive impairment in congenital myotonic dystrophy. Neurobiology of Disease, 2021, 160, 105532.	4.4	7
112	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
113	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
114	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
115	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
116	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
117	NPRL2 Inhibition of mTORC1 Controls Sodium Channel Expression and Brain Amino Acid Homeostasis. ENeuro, 2022, 9, ENEURO.0317-21.2022.	1.9	5
118	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
119	A wireless system for combined heart optogenetics and electrocardiography recording. , 2017, , .		4
120	Metaflumizone inhibits the honeybee Na_V1 channel by targeting recovery from slow inactivation. FEBS Letters, 2017, 591, 3842-3849.	2.8	4
121	Modulation of peripheral Na+ channels and neuronal firing by n-butyl-p-aminobenzoate. European Journal of Pharmacology, 2014, 727, 158-166.	3.5	3
122	MTSET modification of D4S6 cysteines stabilize the fast inactivated state of Nav1.5 sodium channels. Frontiers in Pharmacology, 2015, 6, 118.	3.5	3
123	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
124	Cardiac Metabolic State and Brugada Syndrome. Circulation Research, 2009, 105, 721-723.	4.5	2
125	Improving the characterization of calcium channel gating pore currents with Stac3. Journal of General Physiology, 2018, 150, 375-378.	1.9	2
126	Lymphoblastoid-derived human-induced pluripotent stem cells. , 2021, , 57-70.		2

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127	Recent advances in voltage-gated sodium channels, their pharmacology, and related diseases. Frontiers in Pharmacology, 2013, 4, 52.	3.5	1
128	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
129	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
130	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
131	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
132	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
133	Biophysical Characterization of the Honeybee's DSC1 Ortholog Highlights a New Voltage Dependant Calcium Channel Subfamily. Biophysical Journal, 2016, 110, 34a.	0.5	0
134	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
135	Biophysical and Molecular Characterization of Calcium Permeable Honeybee DSC1 (AmCaV4) Channel Expressed in Mammalian Cells. Biophysical Journal, 2019, 116, 390a.	0.5	0