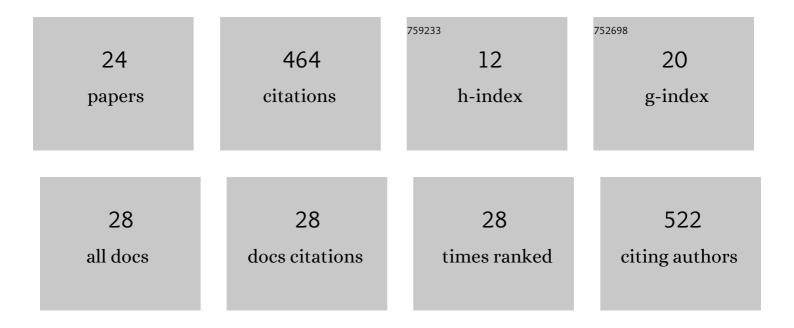
Colin H Peters

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Cardiac Pacemaker Activity and Aging. Annual Review of Physiology, 2020, 82, 21-43.	13.1	59
2	Extracellular Proton Modulation of the Cardiac Voltage-Gated Sodium Channel, NaV1.5. Biophysical Journal, 2011, 101, 2147-2156.	0.5	44
3	Acidosis Differentially Modulates Inactivation in NaV1.2, NaV1.4, and NaV1.5 Channels. Frontiers in Pharmacology, 2012, 3, 109.	3.5	40
4	Differential thermosensitivity in mixed syndrome cardiac sodium channel mutants. Journal of Physiology, 2015, 593, 4201-4223.	2.9	34
5	Triggers for arrhythmogenesis in the Brugada and long QT 3 syndromes. Progress in Biophysics and Molecular Biology, 2016, 120, 77-88.	2.9	31
6	Proton Sensors in the Pore Domain of the Cardiac Voltage-gated Sodium Channel. Journal of Biological Chemistry, 2013, 288, 4782-4791.	3.4	27
7	Effects of the antianginal drug, ranolazine, on the brain sodium channel <scp>N</scp> a _V 1.2 and its modulation by extracellular protons. British Journal of Pharmacology, 2013, 169, 704-716.	5.4	25
8	Introduction to Sodium Channels. Handbook of Experimental Pharmacology, 2014, 221, 1-6.	1.8	18
9	Proton-dependent inhibition of the cardiac sodium channel Nav1.5 by ranolazine. Frontiers in Pharmacology, 2013, 4, 78.	3.5	17
10	Effects of acidosis on neuronal voltage-gated sodium channels: Nav1.1 and Nav1.3. Channels, 2018, 12, 367-377.	2.8	17
11	Functional Genomics of Epilepsy and Associated Neurodevelopmental Disorders Using Simple Animal Models: From Genes, Molecules to Brain Networks. Frontiers in Cellular Neuroscience, 2019, 13, 556.	3.7	17
12	pH Modulation of Voltage-Gated Sodium Channels. Handbook of Experimental Pharmacology, 2018, 246, 147-160.	1.8	16
13	Bidirectional flow of the funny current (I _f) during the pacemaking cycle in murine sinoatrial node myocytes. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	7.1	14
14	Depolarization of the conductance-voltage relationship in the NaV1.5 mutant, E1784K, is due to altered fast inactivation. PLoS ONE, 2017, 12, e0184605.	2.5	14
15	Compound heterozygous <i>TRPV4</i> mutations in two siblings with a complex phenotype including severe intellectual disability and neuropathy. American Journal of Medical Genetics, Part A, 2017, 173, 3087-3092.	1.2	13
16	lsoform-specific regulation of HCN4 channels by a family of endoplasmic reticulum proteins. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 18079-18090.	7.1	13
17	Intracellular Na+ Modulates Pacemaking Activity in Murine Sinoatrial Node Myocytes: An In Silico Analysis. International Journal of Molecular Sciences, 2021, 22, 5645.	4.1	13
18	Persistent sodium currents in <i>SCN1A</i> developmental and degenerative epileptic dyskinetic encephalopathy. Brain Communications, 2021, 3, fcab235.	3.3	12

COLIN H PETERS

#	Article	IF	CITATIONS
19	A Mixed Periodic Paralysis & Myotonia Mutant, P1158S, Imparts pH-Sensitivity in Skeletal Muscle Voltage-gated Sodium Channels. Scientific Reports, 2018, 8, 6304.	3.3	11
20	Regulation of HCN Channels by Protein Interactions. Frontiers in Physiology, 0, 13, .	2.8	11
21	E1784K, the most common Brugada syndrome and long-QT syndrome type 3 mutant, disrupts sodium channel inactivation through two separate mechanisms. Journal of General Physiology, 2020, 152, .	1.9	10
22	The L1624Q Variant in SCN1A Causes Familial Epilepsy Through a Mixed Gain and Loss of Channel Function. Frontiers in Pharmacology, 2021, 12, 788192.	3.5	3
23	Arrhythmogenic triggers associated with Sudden Cardiac Death. Channels, 2018, 12, 76-77.	2.8	1
24	Case studies in neuroscience: a novel amino acid duplication in the NH ₂ -terminus of the brain sodium channel Na _V 1.1 underlying Dravet syndrome. Journal of Neurophysiology, 2019, 122, 1975-1980.	1.8	1