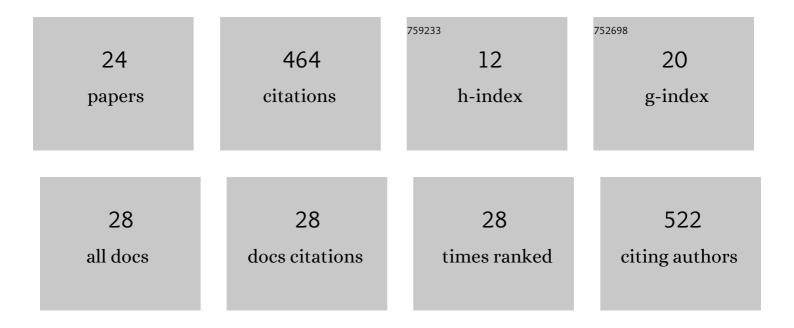
Colin H Peters

List of Publications by Year in descending order

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COLIN H DETEDS

#	Article	IF	CITATIONS
1	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
2	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
3	Persistent sodium currents in <i>SCN1A</i> developmental and degenerative epileptic dyskinetic encephalopathy. Brain Communications, 2021, 3, fcab235.	3.3	12
4	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
5	Cardiac Pacemaker Activity and Aging. Annual Review of Physiology, 2020, 82, 21-43.	13.1	59
6	Isoform-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
7	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
8	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
9	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
10	pH Modulation of Voltage-Gated Sodium Channels. Handbook of Experimental Pharmacology, 2018, 246, 147-160.	1.8	16
11	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
12	Arrhythmogenic triggers associated with Sudden Cardiac Death. Channels, 2018, 12, 76-77.	2.8	1
13	Effects of acidosis on neuronal voltage-gated sodium channels: Nav1.1 and Nav1.3. Channels, 2018, 12, 367-377.	2.8	17
14	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
15	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
16	Triggers for arrhythmogenesis in the Brugada and long QT 3 syndromes. Progress in Biophysics and Molecular Biology, 2016, 120, 77-88.	2.9	31
17	Differential thermosensitivity in mixed syndrome cardiac sodium channel mutants. Journal of Physiology, 2015, 593, 4201-4223.	2.9	34
18	Introduction to Sodium Channels. Handbook of Experimental Pharmacology, 2014, 221, 1-6.	1.8	18

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#	Article	IF	CITATIONS
19	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
20	Proton Sensors in the Pore Domain of the Cardiac Voltage-gated Sodium Channel. Journal of Biological Chemistry, 2013, 288, 4782-4791.	3.4	27
21	Proton-dependent inhibition of the cardiac sodium channel Nav1.5 by ranolazine. Frontiers in Pharmacology, 2013, 4, 78.	3.5	17
22	Acidosis Differentially Modulates Inactivation in NaV1.2, NaV1.4, and NaV1.5 Channels. Frontiers in Pharmacology, 2012, 3, 109.	3.5	40
23	Extracellular Proton Modulation of the Cardiac Voltage-Gated Sodium Channel, NaV1.5. Biophysical Journal, 2011, 101, 2147-2156.	0.5	44
24	Regulation of HCN Channels by Protein Interactions. Frontiers in Physiology, 0, 13, .	2.8	11