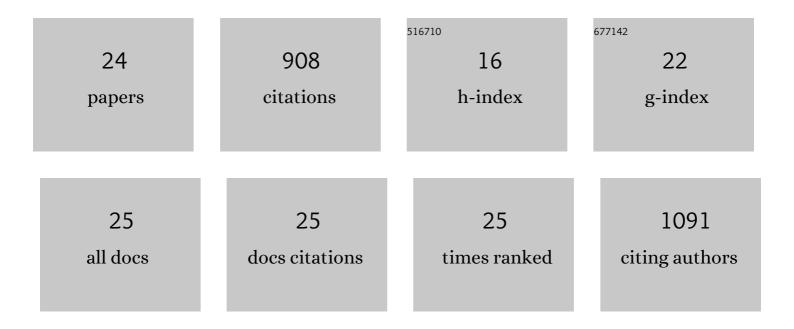
Conor McClenaghan

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	K2P channel gating mechanisms revealed by structures of TREK-2 and a complex with Prozac. Science, 2015, 347, 1256-1259.	12.6	255
2	Polymodal activation of the TREK-2 K2P channel produces structurally distinct open states. Journal of General Physiology, 2016, 147, 497-505.	1.9	65
3	Bilayer-Mediated Structural Transitions Control Mechanosensitivity of the TREK-2 K2P Channel. Structure, 2017, 25, 708-718.e2.	3.3	64
4	Loss-of-Function <i>ABCC8</i> Mutations in Pulmonary Arterial Hypertension. Circulation Genomic and Precision Medicine, 2018, 11, e002087.	3.6	62
5	Coronavirus Proteins as Ion Channels: Current and Potential Research. Frontiers in Immunology, 2020, 11, 573339.	4.8	56
6	Cantú syndrome: Findings from 74 patients in the International Cantú Syndrome Registry. American Journal of Medical Genetics, Part C: Seminars in Medical Genetics, 2019, 181, 658-681.	1.6	50
7	Cardiovascular consequences of KATP overactivity in Cantu syndrome. JCI Insight, 2018, 3, .	5.0	44
8	Glibenclamide reverses cardiovascular abnormalities of Cantu syndrome driven by KATP channel overactivity. Journal of Clinical Investigation, 2020, 130, 1116-1121.	8.2	40
9	Cantu syndrome–associated SUR2 (ABCC9) mutations in distinct structural domains result in KATP channel gain-of-function by differential mechanisms. Journal of Biological Chemistry, 2018, 293, 2041-2052.	3.4	34
10	Increased prokineticin 2 expression in gut inflammation: role in visceral pain and intestinal ion transport. Neurogastroenterology and Motility, 2012, 24, 65.	3.0	33
11	Conserved functional consequences of disease-associated mutations in the slide helix of Kir6.1 and Kir6.2 subunits of the ATP-sensitive potassium channel. Journal of Biological Chemistry, 2017, 292, 17387-17398.	3.4	31
12	ABCC9-related Intellectual disability Myopathy Syndrome is a KATP channelopathy with loss-of-function mutations in ABCC9. Nature Communications, 2019, 10, 4457.	12.8	31
13	Glibenclamide treatment in a Cantú syndrome patient with a pathogenic ABCC9 gainâ€ofâ€function variant: Initial experience. American Journal of Medical Genetics, Part A, 2019, 179, 1585-1590.	1.2	30
14	Pulmonary Hypertension and ATP-Sensitive Potassium Channels. Hypertension, 2019, 74, 14-22.	2.7	24
15	Pathophysiological Consequences of KATP Channel Overactivity and Pharmacological Response to Glibenclamide in Skeletal Muscle of a Murine Model of Cantù Syndrome. Frontiers in Pharmacology, 2020, 11, 604885.	3.5	19
16	The Mechanism of High-Output Cardiac Hypertrophy Arising From Potassium Channel Gain-of-Function in Cantú Syndrome. Function, 2020, 1, zqaa004.	2.3	18
17	Complex consequences of Cantu syndrome SUR2 variant R1154Q in genetically modified mice. JCI Insight, 2021, 6, .	5.0	11
18	TRPA1 Agonist Activity of Probenecid Desensitizes Channel Responses: Consequences for Screening. Assay and Drug Development Technologies, 2012, 10, 533-541.	1.2	10

#	Article	IF	CITATIONS
19	Consequences of SUR2[A478V] Mutation in Skeletal Muscle of Murine Model of Cantu Syndrome. Cells, 2021, 10, 1791.	4.1	10
20	Betaâ€cell excitability and excitabilityâ€driven diabetes in adult Zebrafish islets. Physiological Reports, 2019, 7, e14101.	1.7	8
21	GPR39 Is Coupled to TMEM16A in Intestinal Fibroblast-Like Cells. PLoS ONE, 2012, 7, e47686.	2.5	7
22	ATPâ€sensitive potassium channels in zebrafish cardiac and vascular smooth muscle. Journal of Physiology, 2022, 600, 299-312.	2.9	6
23	Bridging Personal and Population in Excitability Diseases: Will Studies of Rare Diseases Bring Generalizable Mechanisms From Monogenic Channelopathies?. Function, 2022, 3, zqab072.	2.3	0
24	Isolation of Cardiac and Vascular Smooth Muscle Cells from Adult, Juvenile, Larval and Embryonic Zebrafish for Electrophysiological Studies. Journal of Visualized Experiments, 2022, , .	0.3	0