

Peter Proks

List of Publications by Year in descending order

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32
papers

3,481
citations

331259

21
h-index

454577

30
g-index

36
all docs

36
docs citations

36
times ranked

3108
citing authors

#	ARTICLE	IF	CITATIONS
1	The <i>KCNJ11-E23K</i> Gene Variant Hastens Diabetes Progression by Impairing Glucose-Induced Insulin Secretion. <i>Diabetes</i> , 2021, 70, 1145-1156.	0.3	11
2	Norfluoxetine inhibits TREK-2 K ₂ P channels by multiple mechanisms including state-independent effects on the selectivity filter gate. <i>Journal of General Physiology</i> , 2021, 153, .	0.9	17
3	Effects of ionic strength on gating and permeation of TREK-2 K ₂ P channels. <i>PLoS ONE</i> , 2021, 16, e0258275.	1.1	0
4	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. <i>Wellcome Open Research</i> , 2020, 5, 15.	0.9	1
5	Role of the C-terminus of SUR in the differential regulation of β -cell and cardiac K ^{ATP} channels by MgADP and metabolism. <i>Journal of Physiology</i> , 2018, 596, 6205-6217.	1.3	6
6	Binding of sulphonylureas to plasma proteins – A K ^{ATP} channel perspective. <i>PLoS ONE</i> , 2018, 13, e0197634.	1.1	14
7	Running out of time: the decline of channel activity and nucleotide activation in adenosine triphosphate-sensitive K-channels. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2016, 371, 20150426.	1.8	14
8	Neonatal diabetes caused by a homozygous <i>KCNJ11</i> mutation demonstrates that tiny changes in ATP sensitivity markedly affect diabetes risk. <i>Diabetologia</i> , 2016, 59, 1430-1436.	2.9	25
9	The value of <i>in vitro</i> studies in a case of neonatal diabetes with a novel Kir6.2 ^{W68G} mutation. <i>Clinical Case Reports (discontinued)</i> , 2015, 3, 884-887.	0.2	4
10	Molecular action of sulphonylureas on K ^{ATP} channels: a real partnership between drugs and nucleotides. <i>Biochemical Society Transactions</i> , 2015, 43, 901-907.	1.6	39
11	Reversible changes in pancreatic islet structure and function produced by elevated blood glucose. <i>Nature Communications</i> , 2014, 5, 4639.	5.8	220
12	Sulfonylureas suppress the stimulatory action of Mg-nucleotides on Kir6.2/SUR1 but not Kir6.2/SUR2A K ^{ATP} channels: A mechanistic study. <i>Journal of General Physiology</i> , 2014, 144, 469-486.	0.9	20
13	Molecular Mechanism of Sulphonylurea Block of K ^{ATP} Channels Carrying Mutations That Impair ATP Inhibition and Cause Neonatal Diabetes. <i>Diabetes</i> , 2013, 62, 3909-3919.	0.3	44
14	Neonatal Diabetes Caused by Activating Mutations in the Sulphonylurea Receptor. <i>Diabetes and Metabolism Journal</i> , 2013, 37, 157.	1.8	15
15	Development of postpartum Graves' disease and type 1 diabetes after delivery in a patient with gestational diabetes. <i>Journal of Diabetes Investigation</i> , 2011, 2, 328-330.	1.1	5
16	Activation of the K ^{ATP} channel by Mg-nucleotide interaction with SUR1. <i>Journal of General Physiology</i> , 2010, 136, 389-405.	0.9	51
17	Modeling K ^{ATP} channel gating and its regulation. <i>Progress in Biophysics and Molecular Biology</i> , 2009, 99, 7-19.	1.4	55
18	Expression of an activating mutation in the gene encoding the K ^{ATP} channel subunit Kir6.2 in mouse pancreatic β cells recapitulates neonatal diabetes. <i>Journal of Clinical Investigation</i> , 2009, 119, 80-90.	3.9	95

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19	How ATP Inhibits the Open KATP Channel. <i>Journal of General Physiology</i> , 2008, 132, 131-144.	0.9	53
20	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. <i>Human Molecular Genetics</i> , 2007, 16, 2011-2019.	1.4	51
21	Functional Effects of Mutations at F35 in the NH2-terminus of Kir6.2 (KCNJ11), Causing Neonatal Diabetes, and Response to Sulfonylurea Therapy. <i>Diabetes</i> , 2006, 55, 1731-1737.	0.3	41
22	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. <i>Human Molecular Genetics</i> , 2006, 15, 1793-1800.	1.4	196
23	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. <i>EMBO Reports</i> , 2005, 6, 470-475.	2.0	99
24	Relapsing diabetes can result from moderately activating mutations in KCNJ11. <i>Human Molecular Genetics</i> , 2005, 14, 925-934.	1.4	184
25	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. <i>Human Molecular Genetics</i> , 2005, 14, 2717-2726.	1.4	74
26	Molecular basis of Kir6.2 mutations associated with neonatal diabetes or neonatal diabetes plus neurological features. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2004, 101, 17539-17544.	3.3	223
27	Activating Mutations in the Gene Encoding the ATP-Sensitive Potassium-Channel Subunit Kir6.2 and Permanent Neonatal Diabetes. <i>New England Journal of Medicine</i> , 2004, 350, 1838-1849.	13.9	1,077
28	Sulfonylurea Stimulation of Insulin Secretion. <i>Diabetes</i> , 2002, 51, S368-S376.	0.3	393
29	Mutations within the P-Loop of Kir6.2 Modulate the Intraburst Kinetics of the Atp-Sensitive Potassium Channel. <i>Journal of General Physiology</i> , 2001, 118, 341-353.	0.9	88
30	Expression of functionally active ATP-sensitive K-channels in insect cells using baculovirus. <i>FEBS Letters</i> , 1998, 429, 390-394.	1.3	38
31	Molecular Analysis of ATP-sensitive K Channel Gating and Implications for Channel Inhibition by ATP. <i>Journal of General Physiology</i> , 1998, 112, 333-349.	0.9	168
32	Promiscuous coupling between the sulphonylurea receptor and inwardly rectifying potassium channels. <i>Nature</i> , 1996, 379, 545-548.	13.7	156