Frances M Ashcroft

List of Publications by Year
in descending order

142 papers	20,964 citations	15466 65 h-index	10127 140 g-index
151 all docs	151 docs citations	151 times ranked	15253 citing authors

#	Article	IF	Citations
1	The Obesity-Associated FTO Gene Encodes a 2-Oxoglutarate-Dependent Nucleic Acid Demethylase. Science, 2007, 318, 1469-1472.	6.0	1,305
2	Activating Mutations in the Gene Encoding the ATP-Sensitive Potassium-Channel Subunit Kir6.2 and Permanent Neonatal Diabetes. New England Journal of Medicine, 2004, 350, 1838-1849.	13.9	1,077
3	Glucose induces closure of single potassium channels in isolated rat pancreatic Î²-cells. Nature, 1984, 312, 446-448.	13.7	1,075
4	Electrophysiology of the pancreatic Î²-cell. Progress in Biophysics and Molecular Biology, 1989, 54, 87-143.	1.4	984
5	Switching from Insulin to Oral Sulfonylureas in Patients with Diabetes Due to Kir6.2 Mutations. New England Journal of Medicine, 2006, 355, 467-477.	13.9	878
6	Crystal Structure of the Potassium Channel KirBac1.1 in the Closed State. Science, 2003, 300, 1922-1926.	6.0	763
7	Diabetes Mellitus and the Î² Cell: The Last Ten Years. Cell, 2012, 148, 1160-1171.	13.5	761
8	Truncation of Kir6.2 produces ATP-sensitive K+ channels in the absence of the sulphonylurea receptor. Nature, 1997, 387, 179-183.	13.7	723
9	Properties and functions of ATP-sensitive K-channels. Cellular Signalling, 1990, 2, 197-214.	1.7	688
10	Overexpression of Fto leads to increased food intake and results in obesity. Nature Genetics, 2010, 42, 1086-1092.	9.4	612
11	ATP-sensitive potassium channelopathies: focus on insulin secretion. Journal of Clinical Investigation, 2005, 115, 2047-2058.	3.9	519
12	Pancreatic Î²-Cell Electrical Activity and Insulin Secretion: Of Mice and Men. Physiological Reviews, 2018, 98, 117-214.	13.1	497
13	ATP-sensitive K+ channels in the hypothalamus are essential for the maintenance of glucose homeostasis. Nature Neuroscience, 2001, 4, 507-512.	7.1	470
14	Activating Mutations in Kir6.2 and Neonatal Diabetes: New Clinical Syndromes, New Scientific Insights, and New Therapy. Diabetes, 2005, 54, 2503-2513.	0.3	399
15	Sulfonylurea Stimulation of Insulin Secretion. Diabetes, 2002, 51, S368-S376.	0.3	393
16	A Mouse Model for the Metabolic Effects of the Human Fat Mass and Obesity Associated FTO Gene. PLoS Genetics, 2009, 5, e1000599.	1.5	282
17	Enhanced PIP3 signaling in POMC neurons causes KATP channel activation and leads to diet-sensitive obesity. Journal of Clinical Investigation, 2006, 116, 1886-1901.	3.9	281
18	Control of Pancreatic Î² Cell Regeneration by Glucose Metabolism. Cell Metabolism, 2011, 13, 440-449.	7.2	266

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19	A Novel Method for Measurement of Submembrane ATP Concentration. Journal of Biological Chemistry, 2000, 275, 30046-30049.	1.6	257
20	The ATP-sensitivity of K+ channels in rat pancreatic B-cells is modulated by ADP. FEBS Letters, 1986, 208, 63-66.	1.3	235
21	The sulfonylurea receptor. Biochimica Et Biophysica Acta - Molecular Cell Research, 1992, 1175, 45-59.	1.9	235
22	Molecular basis of Kir6.2 mutations associated with neonatal diabetes or neonatal diabetes plus neurological features. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 17539-17544.	3.3	223
23	Diabetes causes marked inhibition of mitochondrial metabolism in pancreatic Î²-cells. Nature Communications, 2019, 10, 2474.	5.8	223
24	KATP channels and islet hormone secretion: new insights and controversies. Nature Reviews Endocrinology, 2013, 9, 660-669.	4.3	221
25	Reversible changes in pancreatic islet structure and function produced by elevated blood glucose. Nature Communications, 2014, 5, 4639.	5.8	220
26	Overlapping distribution of KATP channel-forming Kir6.2 subunit and the sulfonylurea receptor SUR1 in rodent brain. FEBS Letters, 1997, 401, 59-64.	1.3	216
27	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. Human Molecular Genetics, 2006, 15, 1793-1800.	1.4	196
28	Permanent Neonatal Diabetes Caused by Dominant, Recessive, or Compound Heterozygous SUR1 Mutations with Opposite Functional Effects. American Journal of Human Genetics, 2007, 81, 375-382.	2.6	194
29	FTO influences adipogenesis by regulating mitotic clonal expansion. Nature Communications, 2015, 6, 6792.	5.8	186
30	Relapsing diabetes can result from moderately activating mutations in KCNJ11. Human Molecular Genetics, 2005, 14, 925-934.	1.4	184
31	Role of KATP Channels in Glucose-Regulated Glucagon Secretion and Impaired Counterregulation in Type 2 Diabetes. Cell Metabolism, 2013, 18, 871-882.	7.2	179
32	New windows on the mechanism of action of KATP channel openers. Trends in Pharmacological Sciences, 2000, 21, 439-445.	4.0	178
33	Functional analysis of a structural model of the ATP-binding site of the KATP channel Kir6.2 subunit. EMBO Journal, 2005, 24, 229-239.	3.5	177
34	Molecular Analysis of ATP-sensitive K Channel Gating and Implications for Channel Inhibition by ATP. Journal of General Physiology, 1998, 112, 333-349.	0.9	168
35	A new subtype of autosomal dominant diabetes attributable to a mutation in the gene for sulfonylurea receptor 1. Lancet, The, 2003, 361, 301-307.	6.3	163
36	Simultaneous recordings of glucose dependent electrical activity and ATP-regulated K+ -currents in isolated mouse pancreatic Î²-cells. FEBS Letters, 1990, 261, 187-190.	1.3	159

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37	Promiscuous coupling between the sulphonylurea receptor and inwardly rectifying potassium channels. Nature, 1996, 379, 545-548.	13.7	156
38	3-D structural and functional characterization of the purified KATPchannel complex Kir6.2-SUR1. EMBO Journal, 2005, 24, 4166-4175.	3.5	156
39	The Interaction of nucleotides with the tolbutamide block of cloned atp-sensitive k+channel currents expressed in xenopus oocytes: a reinterpretation. Journal of Physiology, 1997, 504, 35-45.	1.3	149
40	SYMPOSIUM REVIEW: The role of the K_{ATP}channel in glucose homeostasis in health and disease: more than meets the islet. Journal of Physiology, 2010, 588, 3201-3209.	1.3	147
41	SUR1: a unique ATP-binding cassette protein that functions as an ion channel regulator. Philosophical Transactions of the Royal Society B: Biological Sciences, 2009, 364, 257-267.	1.8	138
42	Chronic Palmitate Exposure Inhibits Insulin Secretion by Dissociation of Ca2+ Channels from Secretory Granules. Cell Metabolism, 2009, 10, 455-465.	7.2	131
43	Adult Onset Global Loss of the Fto Gene Alters Body Composition and Metabolism in the Mouse. PLoS Genetics, 2013, 9, e1003166.	1.5	129
44	PIP₂-Binding Site in Kir Channels: Definition by Multiscale Biomolecular Simulations. Biochemistry, 2009, 48, 10926-10933.	1.2	127
45	Type 2 Diabetes and Congenital Hyperinsulinism Cause DNA Double-Strand Breaks and p53 Activity in Î² Cells. Cell Metabolism, 2014, 19, 109-121.	7.2	123
46	Mechanism of Cloned ATP-sensitive Potassium Channel Activation by Oleoyl-CoA. Journal of Biological Chemistry, 1998, 273, 26383-26387.	1.6	119
47	Sulfonylurea improves CNS function in a case of intermediate DEND syndrome caused by a mutation in KCNJ11. Nature Clinical Practice Neurology, 2007, 3, 640-645.	2.7	102
48	Q&A: insulin secretion and type 2 diabetes: why do Î²-cells fail?. BMC Biology, 2015, 13, 33.	1.7	102
49	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. EMBO Reports, 2005, 6, 470-475.	2.0	99
50	ATP-sensitive K⁺ channels and disease: from molecule to malady. American Journal of Physiology - Endocrinology and Metabolism, 2007, 293, E880-E889.	1.8	98
51	Expression of an activating mutation in the gene encoding the KATP channel subunit Kir6.2 in mouse pancreatic Î² cells recapitulates neonatal diabetes. Journal of Clinical Investigation, 2009, 119, 80-90.	3.9	95
52	Direct Photoaffinity Labeling of the Kir6.2 Subunit of the ATP-sensitive K+ Channel by 8-Azido-ATP. Journal of Biological Chemistry, 1999, 274, 3931-3933.	1.6	93
53	Involvement of the N-terminus of Kir6.2 in coupling to the sulphonylurea receptor. Journal of Physiology, 1999, 518, 325-336.	1.3	92
54	Type 2 diabetes mellitus: not quite exciting enough?. Human Molecular Genetics, 2004, 13, 21R-31.	1.4	90

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55	Hyperglycaemia induces metabolic dysfunction and glycogen accumulation in pancreatic Î²-cells. Nature Communications, 2016, 7, 13496.	5.8	90
56	Mutations within the P-Loop of Kir6.2 Modulate the Intraburst Kinetics of the Atp-Sensitive Potassium Channel. Journal of General Physiology, 2001, 118, 341-353.	0.9	88
57	Differential Interactions of Nateglinide and Repaglinide on the Human Â-Cell Sulphonylurea Receptor 1. Diabetes, 2002, 51, 2789-2795.	0.3	88
58	Muscle Dysfunction Caused by a K _{ATP} Channel Mutation in Neonatal Diabetes Is Neuronal in Origin. Science, 2010, 329, 458-461.	6.0	87
59	New insights into KATP channel gene mutations and neonatal diabetes mellitus. Nature Reviews Endocrinology, 2020, 16, 378-393.	4.3	87
60	Neonatal Diabetes and the K ATP Channel: From Mutation to Therapy. Trends in Endocrinology and Metabolism, 2017, 28, 377-387.	3.1	79
61	Na⁺ current properties in islet Î±â€•and Î²â€cells reflect cellâ€specific Scn3a and Scn9a expression. Journal of Physiology, 2014, 592, 4677-4696.	1.3	78
62	Identification of the PIP2-binding site on Kir6.2 by molecular modelling and functional analysis. EMBO Journal, 2007, 26, 3749-3759.	3.5	75
63	Identification of residues contributing to the ATP binding site of Kir6.2. EMBO Journal, 2003, 22, 2903-2912.	3.5	74
64	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. Human Molecular Genetics, 2005, 14, 2717-2726.	1.4	74
65	FTO Is Expressed in Neurones throughout the Brain and Its Expression Is Unaltered by Fasting. PLoS ONE, 2011, 6, e27968.	1.1	74
66	Is Type 2 Diabetes a Glycogen Storage Disease of Pancreatic Î² Cells?. Cell Metabolism, 2017, 26, 17-23.	7.2	70
67	ATP Sensitivity of the ATP-Sensitive K+Channel in Intact and Permeabilized Pancreatic Î²-Cells. Diabetes, 2006, 55, 2446-2454.	0.3	68
68	Successful transfer to sulfonylureas in KCNJ11 neonatal diabetes is determined by the mutation and duration of diabetes. Diabetologia, 2016, 59, 1162-1166.	2.9	68
69	Systemic Administration of Glibenclamide Fails to Achieve Therapeutic Levels in the Brain and Cerebrospinal Fluid of Rodents. PLoS ONE, 2015, 10, e0134476.	1.1	67
70	Mutations at the Same Residue (R50) of Kir6.2 (KCNJ11) That Cause Neonatal Diabetes Produce Different Functional Effects. Diabetes, 2006, 55, 1705-1712.	0.3	64
71	Kir6.2 mutations causing neonatal diabetes provide new insights into Kir6.2â€“SUR1 interactions. EMBO Journal, 2005, 24, 2318-2330.	3.5	63
72	Studies of the ATPase activity of the ABC protein SUR1. FEBS Journal, 2007, 274, 3532-3544.	2.2	62

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73	Focus on Kir6.2: a key component of the ATP-sensitive potassium channel. Journal of Molecular and Cellular Cardiology, 2005, 38, 927-936.	0.9	61
74	Fumarate Hydratase Deletion in Pancreatic Î² Cells Leads to Progressive Diabetes. Cell Reports, 2017, 20, 3135-3148.	2.9	57
75	Modeling KATP channel gating and its regulation. Progress in Biophysics and Molecular Biology, 2009, 99, 7-19.	1.4	55
76	Differential Response of K_{ATP} Channels Containing SUR2A or SUR2B Subunits to Nucleotides and Pinacidil. Molecular Pharmacology, 2000, 58, 1318-1325.	1.0	54
77	Functional analysis of six Kir6.2 (KCNJ11) mutations causing neonatal diabetes. Pflugers Archiv European Journal of Physiology, 2006, 453, 323-332.	1.3	53
78	How ATP Inhibits the Open KATP Channel. Journal of General Physiology, 2008, 132, 131-144.	0.9	53
79	Increased ATPase activity produced by mutations at arginine-1380 in nucleotide-binding domain 2 of ABCC8 causes neonatal diabetes. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 18988-18992.	3.3	51
80	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. Human Molecular Genetics, 2007, 16, 2011-2019.	1.4	51
81	Activation of the KATP channel by Mg-nucleotide interaction with SUR1. Journal of General Physiology, 2010, 136, 389-405.	0.9	51
82	The ligandâ€sensitive gate of a potassium channel lies close to the selectivity filter. EMBO Reports, 2003, 4, 70-75.	2.0	49
83	Pancreatic Î²-Cells Express the Fetal Islet Hormone Gastrin in Rodent and Human Diabetes. Diabetes, 2017, 66, 426-436.	0.3	47
84	Functional identification of islet cell types by electrophysiological fingerprinting. Journal of the Royal Society Interface, 2017, 14, 20160999.	1.5	45
85	Molecular Mechanism of Sulphonylurea Block of KATP Channels Carrying Mutations That Impair ATP Inhibition and Cause Neonatal Diabetes. Diabetes, 2013, 62, 3909-3919.	0.3	44
86	A Kir6.2 Mutation Causing Neonatal Diabetes Impairs Electrical Activity and Insulin Secretion From INS-1 Â-Cells. Diabetes, 2006, 55, 3075-3082.	0.3	43
87	Functional Effects of Mutations at F35 in the NH2-terminus of Kir6.2 (KCNJ11), Causing Neonatal Diabetes, and Response to Sulfonylurea Therapy. Diabetes, 2006, 55, 1731-1737.	0.3	41
88	A mutation (R826W) in nucleotideâ€binding domain 1 of ABCC8 reduces ATPase activity and causes transient neonatal diabetes. EMBO Reports, 2008, 9, 648-654.	2.0	40
89	New Uses for Old Drugs: Neonatal Diabetes and Sulphonylureas. Cell Metabolism, 2010, 11, 179-181.	7.2	40
90	Expression of functionally active ATP-sensitive K-channels in insect cells using baculovirus. FEBS Letters, 1998, 429, 390-394.	1.3	38

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91	Altered functional properties of KATPchannel conferred by a novel splice variant of SUR1. Journal of Physiology, 1999, 521, 337-350.	1.3	38
92	Modification of K-ATP channels in pancreatic ?-cells by trypsin. Pflugers Archiv European Journal of Physiology, 1993, 424, 63-72.	1.3	37
93	Mapping the architecture of the ATP-binding site of the KATPchannel subunit Kir6.2. Journal of Physiology, 2004, 557, 347-354.	1.3	37
94	Direct Photoaffinity Labeling of Kir6.2 by [Î³-32P]ATP-[Î³]4-Azidoanilide. Biochemical and Biophysical Research Communications, 2000, 272, 316-319.	1.0	36
95	Adjacent mutations in the gating loop of Kir6.2 produce neonatal diabetes and hyperinsulinism. EMBO Molecular Medicine, 2009, 1, 166-177.	3.3	36
96	Introduction. The blurred boundary between channels and transporters. Philosophical Transactions of the Royal Society B: Biological Sciences, 2009, 364, 145-147.	1.8	35
97	Kir6.2-dependent high-affinity repaglinide binding to Î² -cell KATP channels. British Journal of Pharmacology, 2005, 144, 551-557.	2.7	34
98	Pharmacological Inhibition of FTO. PLoS ONE, 2015, 10, e0121829.	1.1	33
99	Functional effects of naturally occurringKCNJ11mutations causing neonatal diabetes on cloned cardiac KATPchannels. Journal of Physiology, 2006, 571, 3-14.	1.3	32
100	Switching to Sulphonylureas in Children With iDEND Syndrome Caused by KCNJ11 Mutations Results in Improved Cerebellar Perfusion. Diabetes Care, 2013, 36, 2311-2316.	4.3	32
101	Expression of voltage-gated K+ channels in insulin-producing cells. FEBS Letters, 1990, 263, 121-126.	1.3	31
102	Changes in Gene Expression Associated with FTO Overexpression in Mice. PLoS ONE, 2014, 9, e97162.	1.1	31
103	A Mouse Model of Human Hyperinsulinism Produced by the E1506K Mutation in the Sulphonylurea Receptor SUR1. Diabetes, 2013, 62, 3797-3806.	0.3	28
104	Cardiac Dysfunction and Metabolic Inflexibility in a Mouse Model of Diabetes Without Dyslipidemia. Diabetes, 2018, 67, 1057-1067.	0.3	28
105	Activation mechanism of ATP-sensitive K+ channels explored with real-time nucleotide binding. ELife, 2019, 8, .	2.8	28
106	The Nucleotide-Binding Sites of SUR1: A Mechanistic Model. Biophysical Journal, 2015, 109, 2452-2460.	0.2	27
107	Identification of a Functionally Important Negatively Charged Residue Within the Second Catalytic Site of the SUR1 Nucleotide-Binding Domains. Diabetes, 2004, 53, S123-S127.	0.3	26
108	Mutations of the Same Conserved Glutamate Residue in NBD2 of the Sulfonylurea Receptor 1 Subunit of the KATP Channel Can Result in Either Hyperinsulinism or Neonatal Diabetes. Diabetes, 2011, 60, 1813-1822.	0.3	25

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109	Neonatal diabetes caused by a homozygous KCNJ11 mutation demonstrates that tiny changes in ATP sensitivity markedly affect diabetes risk. Diabetologia, 2016, 59, 1430-1436.	2.9	25
110	FTO demethylase activity is essential for normal bone growth and bone mineralization in mice. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2018, 1864, 843-850.	1.8	23
111	Evaluating inositol phospholipid interactions with inward rectifier potassium channels and characterising their role in disease. Communications Chemistry, 2020, 3, .	2.0	23
112	Sulfonylureas suppress the stimulatory action of Mg-nucleotides on Kir6.2/SUR1 but not Kir6.2/SUR2A KATP channels: A mechanistic study. Journal of General Physiology, 2014, 144, 469-486.	0.9	20
113	Nucleotide inhibition of the pancreatic ATP-sensitive K+ channel explored with patch-clamp fluorometry. ELife, 2020, 9, .	2.8	20
114	A conserved tryptophan at the membraneâ€“water interface acts as a gatekeeper for Kir6.2/SUR1 channels and causes neonatal diabetes when mutated. Journal of Physiology, 2011, 589, 3071-3083.	1.3	19
115	Fetal Macrosomia and Neonatal Hyperinsulinemic Hypoglycemia Associated With Transplacental Transfer of Sulfonylurea in a Mother With KCNJ11-Related Neonatal Diabetes. Diabetes Care, 2014, 37, 3333-3335.	4.3	19
116	Analysis of the differential modulation of sulphonylurea block of Â-cell and cardiac ATP-sensitive K+ (KATP) channels by Mg-nucleotides. Journal of Physiology, 2003, 547, 159-168.	1.3	19
117	Monitoring real-time hormone release kinetics via high-content 3-D imaging of compensatory endocytosis. Lab on A Chip, 2018, 18, 2838-2848.	3.1	17
118	An In-Frame Deletion in Kir6.2 (KCNJ11) Causing Neonatal Diabetes Reveals a Site of Interaction between Kir6.2 and SUR1. Journal of Clinical Endocrinology and Metabolism, 2009, 94, 2551-2557.	1.8	16
119	Magnesium deficiency prevents high-fat-diet-induced obesity in mice. Diabetologia, 2018, 61, 2030-2042.	2.9	16
120	Low extracellular magnesium does not impair glucose-stimulated insulin secretion. PLoS ONE, 2019, 14, e0217925.	1.1	16
121	Mosaic Paternal Uniparental Isodisomy and an ABCC8 Gene Mutation in a Patient With Permanent Neonatal Diabetes and Hemihypertrophy. Diabetes, 2008, 57, 255-258.	0.3	15
122	Interaction between mutations in the slide helix of Kir6.2 associated with neonatal diabetes and neurological symptoms. Human Molecular Genetics, 2010, 19, 963-972.	1.4	15
123	The ATPase activities of sulfonylurea receptorâ€ƒ2A and sulfonylurea receptorâ€ƒ2B are influenced by the Câ€terminal 42 amino acids. FEBS Journal, 2010, 277, 2654-2662.	2.2	14
124	A mutation causing increased KATP channel activity leads to reduced anxiety in mice. Physiology and Behavior, 2014, 129, 79-84.	1.0	14
125	Running out of time: the decline of channel activity and nucleotide activation in adenosine triphosphate-sensitive K-channels. Philosophical Transactions of the Royal Society B: Biological Sciences, 2016, 371, 20150426.	1.8	14
126	Binding of sulphonylureas to plasma proteins â€“ A KATP channel perspective. PLoS ONE, 2018, 13, e0197634.	1.1	14

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127	Increased NEFA levels reduce blood Mg2+ in hypertriacylglycerolaemic states via direct binding of NEFA to Mg2+. Diabetologia, 2019, 62, 311-321.	2.9	14
128	A universally conserved residue in the SUR1 subunit of the K ATP channel is essential for translating nucleotide binding at SUR1 into channel opening. Journal of Physiology, 2012, 590, 5025-5036.	1.3	13
129	An ABCC8 Nonsense Mutation Causing Neonatal Diabetes Through Altered Transcript Expression. JCRPE Journal of Clinical Research in Pediatric Endocrinology, 2017, 9, 260-264.	0.4	13
130	The ATPase activities of sulfonylurea receptorâ€ƒ2A and sulfonylurea receptorâ€ƒ2B are influenced by the C-terminal 42 amino acids. FEBS Journal, 2010, 277, 2654-2662.	2.2	12
131	The KCNJ11-E23K Gene Variant Hastens Diabetes Progression by Impairing Glucose-Induced Insulin Secretion. Diabetes, 2021, 70, 1145-1156.	0.3	11
132	Tetrameric structure of SUR2B revealed by electron microscopy of oriented single particles. FEBS Journal, 2013, 280, 1051-1063.	2.2	9
133	Gain-of-Function Mutations in the KATP Channel (KCNJ11) Impair Coordinated Hand-Eye Tracking. PLoS ONE, 2013, 8, e62646.	1.1	7
134	Role of the Câ€terminus of SUR in the differential regulation of Î²â€cell and cardiac K ATP channels by MgADP and metabolism. Journal of Physiology, 2018, 596, 6205-6217.	1.3	6
135	The value of inÂvitro studies in a case of neonatal diabetes with a novel Kir6.2â€W68G mutation. Clinical Case Reports (discontinued), 2015, 3, 884-887.	0.2	4
136	A cytosolic factor that inhibits K_{ATP} channels expressed in Xenopus oocytes by impairing Mgâ€nucleotide activation by SUR1. Journal of Physiology, 2009, 587, 1649-1656.	1.3	2
137	Mouse models of Î²-cell KATP channel dysfunction. Drug Discovery Today: Disease Models, 2013, 10, e101-e109.	1.2	2
138	Dissection-independent production ofPlasmodiumsporozoites from whole mosquitoes. Life Science Alliance, 2021, 4, e202101094.	1.3	2
139	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. Wellcome Open Research, 2020, 5, 15.	0.9	1
140	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. Wellcome Open Research, 2020, 5, 15.	0.9	1
141	Influences: Find a friend. Journal of General Physiology, 2018, 150, 895-896.	0.9	0
142	Measuring Nucleotide Binding to Intact, Functional Membrane Proteins in Real Time. Journal of Visualized Experiments, 2021, , .	0.2	0