

Frances M Ashcroft

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

142
papers

17,834
citations

62
h-index

133
g-index

151
ext. papers

19,414
ext. citations

10
avg, IF

6.65
L-index

#	Paper	IF	Citations
142	The Gene Variant Hastens Diabetes Progression by Impairing Glucose-Induced Insulin Secretion. <i>Diabetes</i> , 2021 , 70, 1145-1156	0.9	6
141	New insights into K channel gene mutations and neonatal diabetes mellitus. <i>Nature Reviews Endocrinology</i> , 2020 , 16, 378-393	15.2	41
140	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. <i>Wellcome Open Research</i> , 2020 , 5, 15	4.8	
139	Nucleotide inhibition of the pancreatic ATP-sensitive K ⁺ channel explored with patch-clamp fluorometry. <i>ELife</i> , 2020 , 9,	8.9	9
138	Evaluating inositol phospholipid interactions with inward rectifier potassium channels and characterising their role in disease. <i>Communications Chemistry</i> , 2020 , 3,	6.3	8
137	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. <i>Wellcome Open Research</i> , 2020 , 5, 15	4.8	0
136	Diabetes causes marked inhibition of mitochondrial metabolism in pancreatic β cells. <i>Nature Communications</i> , 2019 , 10, 2474	17.4	102
135	Low extracellular magnesium does not impair glucose-stimulated insulin secretion. <i>PLoS ONE</i> , 2019 , 14, e0217925	3.7	6
134	Activation mechanism of ATP-sensitive K channels explored with real-time nucleotide binding. <i>ELife</i> , 2019 , 8,	8.9	19
133	Increased NEFA levels reduce blood Mg in hypertriglycerolaemic states via direct binding of NEFA to Mg. <i>Diabetologia</i> , 2019 , 62, 311-321	10.3	9
132	Cardiac Dysfunction and Metabolic Inflexibility in a Mouse Model of Diabetes Without Dyslipidemia. <i>Diabetes</i> , 2018 , 67, 1057-1067	0.9	17
131	Magnesium deficiency prevents high-fat-diet-induced obesity in mice. <i>Diabetologia</i> , 2018 , 61, 2030-2042	10.3	9
130	Monitoring real-time hormone release kinetics via high-content 3-D imaging of compensatory endocytosis. <i>Lab on A Chip</i> , 2018 , 18, 2838-2848	7.2	10
129	Binding of sulphonylureas to plasma proteins - A KATP channel perspective. <i>PLoS ONE</i> , 2018 , 13, e0197634	3.7	10
128	Pancreatic β Cell Electrical Activity and Insulin Secretion: Of Mice and Men. <i>Physiological Reviews</i> , 2018 , 98, 117-214	47.9	290
127	FTO demethylase activity is essential for normal bone growth and bone mineralization in mice. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2018 , 1864, 843-850	6.9	16
126	Influences: Find a friend. <i>Journal of General Physiology</i> , 2018 , 150, 895-896	3.4	

125	Role of the C-terminus of SUR in the differential regulation of β cell and cardiac K channels by MgADP and metabolism. <i>Journal of Physiology</i> , 2018 , 596, 6205-6217	3.9	3
124	Neonatal Diabetes and the K Channel: From Mutation to Therapy. <i>Trends in Endocrinology and Metabolism</i> , 2017 , 28, 377-387	8.8	53
123	Functional identification of islet cell types by electrophysiological fingerprinting. <i>Journal of the Royal Society Interface</i> , 2017 , 14,	4.1	33
122	Fumarate Hydratase Deletion in Pancreatic β Cells Leads to Progressive Diabetes. <i>Cell Reports</i> , 2017 , 20, 3135-3148	10.6	34
121	Is Type 2 Diabetes a Glycogen Storage Disease of Pancreatic β Cells?. <i>Cell Metabolism</i> , 2017 , 26, 17-23	24.6	39
120	Pancreatic β Cells Express the Fetal Islet Hormone Gastrin in Rodent and Human Diabetes. <i>Diabetes</i> , 2017 , 66, 426-436	0.9	36
119	An Nonsense Mutation Causing Neonatal Diabetes Through Altered Transcript Expression. <i>JCRPE Journal of Clinical Research in Pediatric Endocrinology</i> , 2017 , 9, 260-264	1.9	10
118	Hyperglycaemia induces metabolic dysfunction and glycogen accumulation in pancreatic β cells. <i>Nature Communications</i> , 2016 , 7, 13496	17.4	67
117	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,	5.8	11
116	Successful transfer to sulfonylureas in KCNJ11 neonatal diabetes is determined by the mutation and duration of diabetes. <i>Diabetologia</i> , 2016 , 59, 1162-6	10.3	54
115	Neonatal diabetes caused by a homozygous KCNJ11 mutation demonstrates that tiny changes in ATP sensitivity markedly affect diabetes risk. <i>Diabetologia</i> , 2016 , 59, 1430-1436	10.3	18
114	FTO influences adipogenesis by regulating mitotic clonal expansion. <i>Nature Communications</i> , 2015 , 6, 6792	17.4	142
113	The value of in vitro studies in a case of neonatal diabetes with a novel Kir6.2-W68G mutation. <i>Clinical Case Reports (discontinued)</i> , 2015 , 3, 884-7	0.7	4
112	Q&A: insulin secretion and type 2 diabetes: why do β cells fail?. <i>BMC Biology</i> , 2015 , 13, 33	7.3	62
111	The Nucleotide-Binding Sites of SUR1: A Mechanistic Model. <i>Biophysical Journal</i> , 2015 , 109, 2452-2460	2.9	17
110	Pharmacological inhibition of FTO. <i>PLoS ONE</i> , 2015 , 10, e0121829	3.7	25
109	Systemic Administration of Glibenclamide Fails to Achieve Therapeutic Levels in the Brain and Cerebrospinal Fluid of Rodents. <i>PLoS ONE</i> , 2015 , 10, e0134476	3.7	51
108	Fetal macrosomia and neonatal hyperinsulinemic hypoglycemia associated with transplacental transfer of sulfonylurea in a mother with KCNJ11-related neonatal diabetes. <i>Diabetes Care</i> , 2014 , 37, 3333-5	14.6	16

107	Type 2 diabetes and congenital hyperinsulinism cause DNA double-strand breaks and p53 activity in β cells. <i>Cell Metabolism</i> , 2014 , 19, 109-21	24.6	101
106	A mutation causing increased KATP channel activity leads to reduced anxiety in mice. <i>Physiology and Behavior</i> , 2014 , 129, 79-84	3.5	14
105	Na ⁺ current properties in islet β and δ cells reflect cell-specific Scn3a and Scn9a expression. <i>Journal of Physiology</i> , 2014 , 592, 4677-96	3.9	60
104	Reversible changes in pancreatic islet structure and function produced by elevated blood glucose. <i>Nature Communications</i> , 2014 , 5, 4639	17.4	153
103	Sulfonylureas suppress the stimulatory action of Mg-nucleotides on Kir6.2/SUR1 but not Kir6.2/SUR2A KATP channels: a mechanistic study. <i>Journal of General Physiology</i> , 2014 , 144, 469-86	3.4	17
102	Changes in gene expression associated with FTO overexpression in mice. <i>PLoS ONE</i> , 2014 , 9, e97162	3.7	27
101	Molecular mechanism of sulphonylurea block of K(ATP) channels carrying mutations that impair ATP inhibition and cause neonatal diabetes. <i>Diabetes</i> , 2013 , 62, 3909-19	0.9	36
100	Mouse Models of β cell K Channel Dysfunction. <i>Drug Discovery Today: Disease Models</i> , 2013 , 10, e101-e109.3		2
99	K(ATP) channels and islet hormone secretion: new insights and controversies. <i>Nature Reviews Endocrinology</i> , 2013 , 9, 660-9	15.2	166
98	Role of KATP channels in glucose-regulated glucagon secretion and impaired counterregulation in type 2 diabetes. <i>Cell Metabolism</i> , 2013 , 18, 871-82	24.6	146
97	Adult onset global loss of the fto gene alters body composition and metabolism in the mouse. <i>PLoS Genetics</i> , 2013 , 9, e1003166	6	103
96	A mouse model of human hyperinsulinism produced by the E1506K mutation in the sulphonylurea receptor SUR1. <i>Diabetes</i> , 2013 , 62, 3797-806	0.9	25
95	Switching to sulphonylureas in children with iDEND syndrome caused by KCNJ11 mutations results in improved cerebellar perfusion. <i>Diabetes Care</i> , 2013 , 36, 2311-6	14.6	23
94	Tetrameric structure of SUR2B revealed by electron microscopy of oriented single particles. <i>FEBS Journal</i> , 2013 , 280, 1051-63	5.7	8
93	Gain-of-function mutations in the K(ATP) channel (KCNJ11) impair coordinated hand-eye tracking. <i>PLoS ONE</i> , 2013 , 8, e62646	3.7	6
92	A universally conserved residue in the SUR1 subunit of the KATP channel is essential for translating nucleotide binding at SUR1 into channel opening. <i>Journal of Physiology</i> , 2012 , 590, 5025-36	3.9	10
91	Diabetes mellitus and the β cell: the last ten years. <i>Cell</i> , 2012 , 148, 1160-71	56.2	640
90	Control of pancreatic β cell regeneration by glucose metabolism. <i>Cell Metabolism</i> , 2011 , 13, 440-449	24.6	229

89	A conserved tryptophan at the membrane-water interface acts as a gatekeeper for Kir6.2/SUR1 channels and causes neonatal diabetes when mutated. <i>Journal of Physiology</i> , 2011 , 589, 3071-83	3.9	13
88	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. <i>Diabetes</i> , 2011 , 60, 1813-22	0.9	20
87	FTO is expressed in neurones throughout the brain and its expression is unaltered by fasting. <i>PLoS ONE</i> , 2011 , 6, e27968	3.7	60
86	The ATPase activities of sulfonylurea receptor 2A and sulfonylurea receptor 2B are influenced by the C-terminal 42 amino acids. <i>FEBS Journal</i> , 2010 , 277, 2654-2662	5.7	12
85	The role of the KATP channel in glucose homeostasis in health and disease: more than meets the islet. <i>Journal of Physiology</i> , 2010 , 588, 3201-9	3.9	123
84	Overexpression of Fto leads to increased food intake and results in obesity. <i>Nature Genetics</i> , 2010 , 42, 1086-92	36.3	501
83	Interaction between mutations in the slide helix of Kir6.2 associated with neonatal diabetes and neurological symptoms. <i>Human Molecular Genetics</i> , 2010 , 19, 963-72	5.6	15
82	Activation of the K(ATP) channel by Mg-nucleotide interaction with SUR1. <i>Journal of General Physiology</i> , 2010 , 136, 389-405	3.4	37
81	Muscle dysfunction caused by a KATP channel mutation in neonatal diabetes is neuronal in origin. <i>Science</i> , 2010 , 329, 458-61	33.3	77
80	New uses for old drugs: neonatal diabetes and sulphonylureas. <i>Cell Metabolism</i> , 2010 , 11, 179-81	24.6	39
79	The ATPase activities of sulfonylurea receptor 2A and sulfonylurea receptor 2B are influenced by the C-terminal 42 amino acids. <i>FEBS Journal</i> , 2010 , 277, 2654-62	5.7	12
78	An in-frame deletion in Kir6.2 (KCNJ11) causing neonatal diabetes reveals a site of interaction between Kir6.2 and SUR1. <i>Journal of Clinical Endocrinology and Metabolism</i> , 2009 , 94, 2551-7	5.6	15
77	A mouse model for the metabolic effects of the human fat mass and obesity associated FTO gene. <i>PLoS Genetics</i> , 2009 , 5, e1000599	6	245
76	Introduction. The blurred boundary between channels and transporters. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2009 , 364, 145-7	5.8	29
75	Review. SUR1: a unique ATP-binding cassette protein that functions as an ion channel regulator. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2009 , 364, 257-67	5.8	118
74	Modeling K(ATP) channel gating and its regulation. <i>Progress in Biophysics and Molecular Biology</i> , 2009 , 99, 7-19	4.7	49
73	Adjacent mutations in the gating loop of Kir6.2 produce neonatal diabetes and hyperinsulinism. <i>EMBO Molecular Medicine</i> , 2009 , 1, 166-77	12	34
72	A cytosolic factor that inhibits KATP channels expressed in <i>Xenopus</i> oocytes by impairing Mg-nucleotide activation by SUR1. <i>Journal of Physiology</i> , 2009 , 587, 1649-56	3.9	2

71	PIP(2)-binding site in Kir channels: definition by multiscale biomolecular simulations. <i>Biochemistry</i> , 2009 , 48, 10926-33	3.2	108
70	Chronic palmitate exposure inhibits insulin secretion by dissociation of Ca(2+) channels from secretory granules. <i>Cell Metabolism</i> , 2009 , 10, 455-65	24.6	116
69	Expression of an activating mutation in the gene encoding the KATP channel subunit Kir6.2 in mouse pancreatic beta cells recapitulates neonatal diabetes. <i>Journal of Clinical Investigation</i> , 2009 , 119, 80-90	15.9	86
68	A mutation (R826W) in nucleotide-binding domain 1 of ABCC8 reduces ATPase activity and causes transient neonatal diabetes. <i>EMBO Reports</i> , 2008 , 9, 648-54	6.5	35
67	How ATP inhibits the open K(ATP) channel. <i>Journal of General Physiology</i> , 2008 , 132, 131-44	3.4	43
66	Mosaic paternal uniparental isodisomy and an ABCC8 gene mutation in a patient with permanent neonatal diabetes and hemihypertrophy. <i>Diabetes</i> , 2008 , 57, 255-8	0.9	12
65	Identification of the PIP2-binding site on Kir6.2 by molecular modelling and functional analysis. <i>EMBO Journal</i> , 2007 , 26, 3749-59	13	69
64	Studies of the ATPase activity of the ABC protein SUR1. <i>FEBS Journal</i> , 2007 , 274, 3532-3544	5.7	59
63	Increased ATPase activity produced by mutations at arginine-1380 in nucleotide-binding domain 2 of ABCC8 causes neonatal diabetes. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007 , 104, 18988-92	11.5	47
62	The Walter B. Cannon Physiology in Perspective Lecture, 2007. ATP-sensitive K+ channels and disease: from molecule to malady. <i>American Journal of Physiology - Endocrinology and Metabolism</i> , 2007 , 293, E880-9	6	91
61	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. <i>Human Molecular Genetics</i> , 2007 , 16, 2011-9	5.6	49
60	Permanent neonatal diabetes caused by dominant, recessive, or compound heterozygous SUR1 mutations with opposite functional effects. <i>American Journal of Human Genetics</i> , 2007 , 81, 375-82	11	161
59	The obesity-associated FTO gene encodes a 2-oxoglutarate-dependent nucleic acid demethylase. <i>Science</i> , 2007 , 318, 1469-72	33.3	1119
58	Sulphonylurea improves CNS function in a case of intermediate DEND syndrome caused by a mutation in KCNJ11. <i>Nature Clinical Practice Neurology</i> , 2007 , 3, 640-5		88
57	Functional effects of mutations at F35 in the NH2-terminus of Kir6.2 (KCNJ11), causing neonatal diabetes, and response to sulphonylurea therapy. <i>Diabetes</i> , 2006 , 55, 1731-7	0.9	39
56	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. <i>Human Molecular Genetics</i> , 2006 , 15, 1793-800	5.6	175
55	ATP sensitivity of the ATP-sensitive K+ channel in intact and permeabilized pancreatic beta-cells. <i>Diabetes</i> , 2006 , 55, 2446-54	0.9	62
54	Mutations at the same residue (R50) of Kir6.2 (KCNJ11) that cause neonatal diabetes produce different functional effects. <i>Diabetes</i> , 2006 , 55, 1705-12	0.9	56

53	A Kir6.2 mutation causing neonatal diabetes impairs electrical activity and insulin secretion from INS-1 beta-cells. <i>Diabetes</i> , 2006 , 55, 3075-82	0.9	37
52	Switching from insulin to oral sulfonylureas in patients with diabetes due to Kir6.2 mutations. <i>New England Journal of Medicine</i> , 2006 , 355, 467-77	59.2	74 ^o
51	Functional effects of naturally occurring KCNJ11 mutations causing neonatal diabetes on cloned cardiac KATP channels. <i>Journal of Physiology</i> , 2006 , 571, 3-14	3.9	25
50	Functional analysis of six Kir6.2 (KCNJ11) mutations causing neonatal diabetes. <i>Pflugers Archiv European Journal of Physiology</i> , 2006 , 453, 323-32	4.6	48
49	Enhanced PIP3 signaling in POMC neurons causes KATP channel activation and leads to diet-sensitive obesity. <i>Journal of Clinical Investigation</i> , 2006 , 116, 1886-901	15.9	254
48	Focus on Kir6.2: a key component of the ATP-sensitive potassium channel. <i>Journal of Molecular and Cellular Cardiology</i> , 2005 , 38, 927-36	5.8	52
47	Kir6.2-dependent high-affinity repaglinide binding to beta-cell K(ATP) channels. <i>British Journal of Pharmacology</i> , 2005 , 144, 551-7	8.6	30
46	Functional analysis of a structural model of the ATP-binding site of the KATP channel Kir6.2 subunit. <i>EMBO Journal</i> , 2005 , 24, 229-39	13	169
45	Kir6.2 mutations causing neonatal diabetes provide new insights into Kir6.2-SUR1 interactions. <i>EMBO Journal</i> , 2005 , 24, 2318-30	13	56
44	3-D structural and functional characterization of the purified KATP channel complex Kir6.2-SUR1. <i>EMBO Journal</i> , 2005 , 24, 4166-75	13	147
43	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. <i>EMBO Reports</i> , 2005 , 6, 470-5	6.5	95
42	ATP-sensitive potassium channelopathies: focus on insulin secretion. <i>Journal of Clinical Investigation</i> , 2005 , 115, 2047-58	15.9	435
41	Relapsing diabetes can result from moderately activating mutations in KCNJ11. <i>Human Molecular Genetics</i> , 2005 , 14, 925-34	5.6	165
40	Activating mutations in Kir6.2 and neonatal diabetes: new clinical syndromes, new scientific insights, and new therapy. <i>Diabetes</i> , 2005 , 54, 2503-13	0.9	356
39	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. <i>Human Molecular Genetics</i> , 2005 , 14, 2717-26	5.6	72
38	Identification of a functionally important negatively charged residue within the second catalytic site of the SUR1 nucleotide-binding domains. <i>Diabetes</i> , 2004 , 53 Suppl 3, S123-7	0.9	24
37	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, 17539-44	11.5	205
36	Type 2 diabetes mellitus: not quite exciting enough?. <i>Human Molecular Genetics</i> , 2004 , 13 Spec No 1, R21-31	5.6	78

35	Mapping the architecture of the ATP-binding site of the KATP channel subunit Kir6.2. <i>Journal of Physiology</i> , 2004 , 557, 347-54	3.9	29
34	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, 1838-49	59.2	93 ^o
33	The ligand-sensitive gate of a potassium channel lies close to the selectivity filter. <i>EMBO Reports</i> , 2003 , 4, 70-5	6.5	49
32	Identification of residues contributing to the ATP binding site of Kir6.2. <i>EMBO Journal</i> , 2003 , 22, 2903-12	3	63
31	A new subtype of autosomal dominant diabetes attributable to a mutation in the gene for sulfonylurea receptor 1. <i>Lancet, The</i> , 2003 , 361, 301-7	4 ^o	143
30	Crystal structure of the potassium channel KirBac1.1 in the closed state. <i>Science</i> , 2003 , 300, 1922-6	33.3	73 ^o
29	Analysis of the differential modulation of sulphonylurea block of beta-cell and cardiac ATP-sensitive K ⁺ (K(ATP)) channels by Mg-nucleotides. <i>Journal of Physiology</i> , 2003 , 547, 159-68	3.9	19
28	Sulfonylurea stimulation of insulin secretion. <i>Diabetes</i> , 2002 , 51 Suppl 3, S368-76	0.9	312
27	Differential interactions of nateglinide and repaglinide on the human beta-cell sulphonylurea receptor 1. <i>Diabetes</i> , 2002 , 51, 2789-95	0.9	85
26	ATP-sensitive K ⁺ channels in the hypothalamus are essential for the maintenance of glucose homeostasis. <i>Nature Neuroscience</i> , 2001 , 4, 507-12	25.5	43 ^o
25	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, 341-53	3.4	85
24	Differential response of K(ATP) channels containing SUR2A or SUR2B subunits to nucleotides and pinacidil. <i>Molecular Pharmacology</i> , 2000 , 58, 1318-25	4.3	47
23	A novel method for measurement of submembrane ATP concentration. <i>Journal of Biological Chemistry</i> , 2000 , 275, 30046-9	5.4	22 ^o
22	Direct photoaffinity labeling of Kir6.2 by [gamma-(32)P]ATP-[gamma]4-azidoanilide. <i>Biochemical and Biophysical Research Communications</i> , 2000 , 272, 316-9	3.4	35
21	New windows on the mechanism of action of K(ATP) channel openers. <i>Trends in Pharmacological Sciences</i> , 2000 , 21, 439-45	13.2	159
20	Direct photoaffinity labeling of the Kir6.2 subunit of the ATP-sensitive K ⁺ channel by 8-azido-ATP. <i>Journal of Biological Chemistry</i> , 1999 , 274, 3931-3	5.4	85
19	Altered functional properties of KATP channel conferred by a novel splice variant of SUR1. <i>Journal of Physiology</i> , 1999 , 521 Pt 2, 337-50	3.9	31
18	Involvement of the n-terminus of Kir6.2 in coupling to the sulphonylurea receptor. <i>Journal of Physiology</i> , 1999 , 518 (Pt 2), 325-36	3.9	81

17	Expression of functionally active ATP-sensitive K-channels in insect cells using baculovirus. <i>FEBS Letters</i> , 1998 , 429, 390-4	3.8	35
16	Mechanism of cloned ATP-sensitive potassium channel activation by oleoyl-CoA. <i>Journal of Biological Chemistry</i> , 1998 , 273, 26383-7	5.4	103
15	Molecular analysis of ATP-sensitive K channel gating and implications for channel inhibition by ATP. <i>Journal of General Physiology</i> , 1998 , 112, 333-49	3.4	156
14	Overlapping distribution of K(ATP) channel-forming Kir6.2 subunit and the sulfonylurea receptor SUR1 in rodent brain. <i>FEBS Letters</i> , 1997 , 401, 59-64	3.8	198
13	The interaction of nucleotides with the tolbutamide block of cloned ATP-sensitive K ⁺ channel currents expressed in <i>Xenopus</i> oocytes: a reinterpretation. <i>Journal of Physiology</i> , 1997 , 504 (Pt 1), 35-45	3.9	136
12	Truncation of Kir6.2 produces ATP-sensitive K ⁺ channels in the absence of the sulphonylurea receptor. <i>Nature</i> , 1997 , 387, 179-83	5.4	668
11	Promiscuous coupling between the sulphonylurea receptor and inwardly rectifying potassium channels. <i>Nature</i> , 1996 , 379, 545-8	5.4	144
10	Modification of K-ATP channels in pancreatic beta-cells by trypsin. <i>Pflugers Archiv European Journal of Physiology</i> , 1993 , 424, 63-72	4.6	33
9	The sulfonylurea receptor. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 1992 , 1175, 45-59	4.9	207
8	Properties and functions of ATP-sensitive K-channels. <i>Cellular Signalling</i> , 1990 , 2, 197-214	4.9	637
7	Simultaneous recordings of glucose dependent electrical activity and ATP-regulated K(+) currents in isolated mouse pancreatic beta-cells. <i>FEBS Letters</i> , 1990 , 261, 187-90	3.8	140
6	Expression of voltage-gated K ⁺ channels in insulin-producing cells. Analysis by polymerase chain reaction. <i>FEBS Letters</i> , 1990 , 263, 121-6	3.8	28
5	Electrophysiology of the pancreatic beta-cell. <i>Progress in Biophysics and Molecular Biology</i> , 1989 , 54, 87-143	4.3	847
4	The ATP-sensitivity of K ⁺ channels in rat pancreatic B-cells is modulated by ADP. <i>FEBS Letters</i> , 1986 , 208, 63-6	3.8	201
3	Glucose induces closure of single potassium channels in isolated rat pancreatic beta-cells. <i>Nature</i> , 1984 , 312, 446-8	5.4	962
2	Evaluating Inositol phospholipid interactions with Inward Rectifier Potassium Channels and characterising their role in Disease		1
1	The dynamic interplay of PIP2 and ATP in the regulation of the KATP channel		1