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 KCNJ11-E23K Gene Variant Hastens Diabetes Progression by Impairing Glucose-Induced Insulin Secretion. Diabetes, 2021, 70, 1145-1156.	0.3	11
2	Measuring Nucleotide Binding to Intact, Functional Membrane Proteins in Real Time. Journal of Visualized Experiments, 2021, , .	0.2	0
3	Dissection-independent production ofPlasmodiumsporozoites from whole mosquitoes. Life Science Alliance, 2021, 4, e202101094.	1.3	2
4	Evaluating inositol phospholipid interactions with inward rectifier potassium channels and characterising their role in disease. Communications Chemistry, 2020, 3, .	2.0	23
5	New insights into KATP channel gene mutations and neonatal diabetes mellitus. Nature Reviews Endocrinology, 2020, 16, 378-393.	4.3	87
6	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. Wellcome Open Research, 2020, 5, 15.	0.9	1
7	Nucleotide inhibition of the pancreatic ATP-sensitive K+ channel explored with patch-clamp fluorometry. ELife, 2020, 9, .	2.8	20
8	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. Wellcome Open Research, 2020, 5, 15.	0.9	1
9	Diabetes causes marked inhibition of mitochondrial metabolism in pancreatic Î²-cells. Nature Communications, 2019, 10, 2474.	5.8	223
10	Low extracellular magnesium does not impair glucose-stimulated insulin secretion. PLoS ONE, 2019, 14, e0217925.	1.1	16
11	Increased NEFA levels reduce blood Mg2+ in hypertriacylglycerolaemic states via direct binding of NEFA to Mg2+. Diabetologia, 2019, 62, 311-321.	2.9	14
12	Activation mechanism of ATP-sensitive K+ channels explored with real-time nucleotide binding. ELife, 2019, 8, .	2.8	28
13	Cardiac Dysfunction and Metabolic Inflexibility in a Mouse Model of Diabetes Without Dyslipidemia. Diabetes, 2018, 67, 1057-1067.	0.3	28
14	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
15	Influences: Find a friend. Journal of General Physiology, 2018, 150, 895-896.	0.9	0
16	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
17	Magnesium deficiency prevents high-fat-diet-induced obesity in mice. Diabetologia, 2018, 61, 2030-2042.	2.9	16
18	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

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19	Binding of sulphonylureas to plasma proteins â€“ A KATP channel perspective. PLoS ONE, 2018, 13, e0197634.	1.1	14
20	Pancreatic Î²-Cell Electrical Activity and Insulin Secretion: Of Mice and Men. Physiological Reviews, 2018, 98, 117-214.	13.1	497
21	Neonatal Diabetes and the K ATP Channel: From Mutation to Therapy. Trends in Endocrinology and Metabolism, 2017, 28, 377-387.	3.1	79
22	Functional identification of islet cell types by electrophysiological fingerprinting. Journal of the Royal Society Interface, 2017, 14, 20160999.	1.5	45
23	Fumarate Hydratase Deletion in Pancreatic Î² Cells Leads to Progressive Diabetes. Cell Reports, 2017, 20, 3135-3148.	2.9	57
24	Is Type 2 Diabetes a Glycogen Storage Disease of Pancreatic Î² Cells?. Cell Metabolism, 2017, 26, 17-23.	7.2	70
25	Pancreatic Î²-Cells Express the Fetal Islet Hormone Gastrin in Rodent and Human Diabetes. Diabetes, 2017, 66, 426-436.	0.3	47
26	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
27	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
28	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
29	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
30	Hyperglycaemia induces metabolic dysfunction and glycogen accumulation in pancreatic Î²-cells. Nature Communications, 2016, 7, 13496.	5.8	90
31	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
32	Q&A: insulin secretion and type 2 diabetes: why do Î²-cells fail?. BMC Biology, 2015, 13, 33.	1.7	102
33	The Nucleotide-Binding Sites of SUR1: A Mechanistic Model. Biophysical Journal, 2015, 109, 2452-2460.	0.2	27
34	FTO influences adipogenesis by regulating mitotic clonal expansion. Nature Communications, 2015, 6, 6792.	5.8	186
35	Pharmacological Inhibition of FTO. PLoS ONE, 2015, 10, e0121829.	1.1	33
36	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

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37	Reversible changes in pancreatic islet structure and function produced by elevated blood glucose. Nature Communications, 2014, 5, 4639.	5.8	220
38	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
39	Changes in Gene Expression Associated with FTO Overexpression in Mice. PLoS ONE, 2014, 9, e97162.	1.1	31
40	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
41	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
42	A mutation causing increased KATP channel activity leads to reduced anxiety in mice. Physiology and Behavior, 2014, 129, 79-84.	1.0	14
43	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
44	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
45	Mouse models of Î²-cell KATP channel dysfunction. Drug Discovery Today: Disease Models, 2013, 10, e101-e109.	1.2	2
46	KATP channels and islet hormone secretion: new insights and controversies. Nature Reviews Endocrinology, 2013, 9, 660-669.	4.3	221
47	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
48	Adult Onset Global Loss of the Fto Gene Alters Body Composition and Metabolism in the Mouse. PLoS Genetics, 2013, 9, e1003166.	1.5	129
49	A Mouse Model of Human Hyperinsulinism Produced by the E1506K Mutation in the Sulphonylurea Receptor SUR1. Diabetes, 2013, 62, 3797-3806.	0.3	28
50	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
51	Tetrameric structure of SUR2B revealed by electron microscopy of oriented single particles. FEBS Journal, 2013, 280, 1051-1063.	2.2	9
52	Gain-of-Function Mutations in the KATP Channel (KCNJ11) Impair Coordinated Hand-Eye Tracking. PLoS ONE, 2013, 8, e62646.	1.1	7
53	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
54	Diabetes Mellitus and the Î² Cell: The Last Ten Years. Cell, 2012, 148, 1160-1171.	13.5	761

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55	Control of Pancreatic Î² Cell Regeneration by Glucose Metabolism. Cell Metabolism, 2011, 13, 440-449.	7.2	266
56	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
57	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
58	FTO Is Expressed in Neurones throughout the Brain and Its Expression Is Unaltered by Fasting. PLoS ONE, 2011, 6, e27968.	1.1	74
59	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
60	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
61	Overexpression of Fto leads to increased food intake and results in obesity. Nature Genetics, 2010, 42, 1086-1092.	9.4	612
62	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
63	Activation of the KATP channel by Mg-nucleotide interaction with SUR1. Journal of General Physiology, 2010, 136, 389-405.	0.9	51
64	Muscle Dysfunction Caused by a K _{ATP} Channel Mutation in Neonatal Diabetes Is Neuronal in Origin. Science, 2010, 329, 458-461.	6.0	87
65	New Uses for Old Drugs: Neonatal Diabetes and Sulphonylureas. Cell Metabolism, 2010, 11, 179-181.	7.2	40
66	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
67	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
68	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
69	Introduction. The blurred boundary between channels and transporters. Philosophical Transactions of the Royal Society B: Biological Sciences, 2009, 364, 145-147.	1.8	35
70	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
71	Modeling KATP channel gating and its regulation. Progress in Biophysics and Molecular Biology, 2009, 99, 7-19.	1.4	55
72	Adjacent mutations in the gating loop of Kir6.2 produce neonatal diabetes and hyperinsulinism. EMBO Molecular Medicine, 2009, 1, 166-177.	3.3	36

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73	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
74	PIP₂-Binding Site in Kir Channels: Definition by Multiscale Biomolecular Simulations. Biochemistry, 2009, 48, 10926-10933.	1.2	127
75	Chronic Palmitate Exposure Inhibits Insulin Secretion by Dissociation of Ca2+ Channels from Secretory Granules. Cell Metabolism, 2009, 10, 455-465.	7.2	131
76	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
77	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
78	How ATP Inhibits the Open KATP Channel. Journal of General Physiology, 2008, 132, 131-144.	0.9	53
79	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
80	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
81	ATP-sensitive K⁺ channels and disease: from molecule to malady. American Journal of Physiology - Endocrinology and Metabolism, 2007, 293, E880-E889.	1.8	98
82	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. Human Molecular Genetics, 2007, 16, 2011-2019.	1.4	51
83	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
84	The Obesity-Associated FTO Gene Encodes a 2-Oxoglutarate-Dependent Nucleic Acid Demethylase. Science, 2007, 318, 1469-1472.	6.0	1,305
85	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
86	Identification of the PIP2-binding site on Kir6.2 by molecular modelling and functional analysis. EMBO Journal, 2007, 26, 3749-3759.	3.5	75
87	Studies of the ATPase activity of the ABC protein SUR1. FEBS Journal, 2007, 274, 3532-3544.	2.2	62
88	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
89	Functional effects of naturally occurringKCNJ11mutations causing neonatal diabetes on cloned cardiac KATPchannels. Journal of Physiology, 2006, 571, 3-14.	1.3	32
90	Functional analysis of six Kir6.2 (KCNJ11) mutations causing neonatal diabetes. Pflugers Archiv European Journal of Physiology, 2006, 453, 323-332.	1.3	53

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91	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
92	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. Human Molecular Genetics, 2006, 15, 1793-1800.	1.4	196
93	ATP Sensitivity of the ATP-Sensitive K+Channel in Intact and Permeabilized Pancreatic Î²-Cells. Diabetes, 2006, 55, 2446-2454.	0.3	68
94	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
95	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
96	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
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	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
99	Kir6.2 mutations causing neonatal diabetes provide new insights into Kir6.2â€“SUR1 interactions. EMBO Journal, 2005, 24, 2318-2330.	3.5	63
100	3-D structural and functional characterization of the purified KATPchannel complex Kir6.2-SUR1. EMBO Journal, 2005, 24, 4166-4175.	3.5	156
101	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
102	ATP-sensitive potassium channelopathies: focus on insulin secretion. Journal of Clinical Investigation, 2005, 115, 2047-2058.	3.9	519
103	Relapsing diabetes can result from moderately activating mutations in KCNJ11. Human Molecular Genetics, 2005, 14, 925-934.	1.4	184
104	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
105	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. Human Molecular Genetics, 2005, 14, 2717-2726.	1.4	74
106	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
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	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

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109	Type 2 diabetes mellitus: not quite exciting enough?. Human Molecular Genetics, 2004, 13, 21R-31.	1.4	90
110	Mapping the architecture of the ATP-binding site of the KATPchannel subunit Kir6.2. Journal of Physiology, 2004, 557, 347-354.	1.3	37
111	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
112	Identification of residues contributing to the ATP binding site of Kir6.2. EMBO Journal, 2003, 22, 2903-2912.	3.5	74
113	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
114	Crystal Structure of the Potassium Channel KirBac1.1 in the Closed State. Science, 2003, 300, 1922-1926.	6.0	763
115	The ligandâ€sensitive gate of a potassium channel lies close to the selectivity filter. EMBO Reports, 2003, 4, 70-75.	2.0	49
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	Sulfonylurea Stimulation of Insulin Secretion. Diabetes, 2002, 51, S368-S376.	0.3	393
118	Differential Interactions of Nateglinide and Repaglinide on the Human Â-Cell Sulphonylurea Receptor 1. Diabetes, 2002, 51, 2789-2795.	0.3	88
119	ATP-sensitive K+ channels in the hypothalamus are essential for the maintenance of glucose homeostasis. Nature Neuroscience, 2001, 4, 507-512.	7.1	470
120	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
121	Differential Response of K_{ATP} Channels Containing SUR2A or SUR2B Subunits to Nucleotides and Pinacidil. Molecular Pharmacology, 2000, 58, 1318-1325.	1.0	54
122	A Novel Method for Measurement of Submembrane ATP Concentration. Journal of Biological Chemistry, 2000, 275, 30046-30049.	1.6	257
123	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
124	New windows on the mechanism of action of KATP channel openers. Trends in Pharmacological Sciences, 2000, 21, 439-445.	4.0	178
125	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
126	Altered functional properties of KATPchannel conferred by a novel splice variant of SUR1. Journal of Physiology, 1999, 521, 337-350.	1.3	38

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127	Involvement of the N-terminus of Kir6.2 in coupling to the sulphonylurea receptor. Journal of Physiology, 1999, 518, 325-336.	1.3	92
128	Expression of functionally active ATP-sensitive K-channels in insect cells using baculovirus. FEBS Letters, 1998, 429, 390-394.	1.3	38
129	Mechanism of Cloned ATP-sensitive Potassium Channel Activation by Oleoyl-CoA. Journal of Biological Chemistry, 1998, 273, 26383-26387.	1.6	119
130	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
131	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
132	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
133	Truncation of Kir6.2 produces ATP-sensitive K+ channels in the absence of the sulphonylurea receptor. Nature, 1997, 387, 179-183.	13.7	723
134	Promiscuous coupling between the sulphonylurea receptor and inwardly rectifying potassium channels. Nature, 1996, 379, 545-548.	13.7	156
135	Modification of K-ATP channels in pancreatic ?-cells by trypsin. Pflugers Archiv European Journal of Physiology, 1993, 424, 63-72.	1.3	37
136	The sulfonylurea receptor. Biochimica Et Biophysica Acta - Molecular Cell Research, 1992, 1175, 45-59.	1.9	235
137	Properties and functions of ATP-sensitive K-channels. Cellular Signalling, 1990, 2, 197-214.	1.7	688
138	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
139	Expression of voltage-gated K+ channels in insulin-producing cells. FEBS Letters, 1990, 263, 121-126.	1.3	31
140	Electrophysiology of the pancreatic Î²-cell. Progress in Biophysics and Molecular Biology, 1989, 54, 87-143.	1.4	984
141	The ATP-sensitivity of K+ channels in rat pancreatic B-cells is modulated by ADP. FEBS Letters, 1986, 208, 63-66.	1.3	235
142	Glucose induces closure of single potassium channels in isolated rat pancreatic Î²-cells. Nature, 1984, 312, 446-448.	13.7	1,075