Frances M Ashcroft

List of Publications by Citations

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 obesity-associated FTO gene encodes a 2-oxoglutarate-dependent nucleic acid demethylase. *Science*, **2007**, 318, 1469-72	33.3	1119
141	Glucose induces closure of single potassium channels in isolated rat pancreatic beta-cells. *Nature*, **1984**, 312, 446-8	50.4	962
140	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, 1838-49	59.2	930
139	Electrophysiology of the pancreatic beta-cell. *Progress in Biophysics and Molecular Biology*, **1989**, 54, 87-143	4.7	847
138	Switching from insulin to oral sulfonylureas in patients with diabetes due to Kir6.2 mutations. *New England Journal of Medicine*, **2006**, 355, 467-77	59.2	740
137	Crystal structure of the potassium channel KirBac1.1 in the closed state. *Science*, **2003**, 300, 1922-6	33.3	730
136	Truncation of Kir6.2 produces ATP-sensitive K+ channels in the absence of the sulphonylurea receptor. *Nature*, **1997**, 387, 179-83	50.4	668
135	Diabetes mellitus and the □cell: the last ten years. *Cell*, **2012**, 148, 1160-71	56.2	640
134	Properties and functions of ATP-sensitive K-channels. *Cellular Signalling*, **1990**, 2, 197-214	4.9	637
133	Overexpression of Fto leads to increased food intake and results in obesity. *Nature Genetics*, **2010**, 42, 1086-92	36.3	501
132	ATP-sensitive potassium channelopathies: focus on insulin secretion. *Journal of Clinical Investigation*, **2005**, 115, 2047-58	15.9	435
131	ATP-sensitive K+ channels in the hypothalamus are essential for the maintenance of glucose homeostasis. *Nature Neuroscience*, **2001**, 4, 507-12	25.5	430
130	Activating mutations in Kir6.2 and neonatal diabetes: new clinical syndromes, new scientific insights, and new therapy. *Diabetes*, **2005**, 54, 2503-13	0.9	356
129	Sulfonylurea stimulation of insulin secretion. *Diabetes*, **2002**, 51 Suppl 3, S368-76	0.9	312
128	Pancreatic □Cell Electrical Activity and Insulin Secretion: Of Mice and Men. *Physiological Reviews*, **2018**, 98, 117-214	47.9	290
127	Enhanced PIP3 signaling in POMC neurons causes KATP channel activation and leads to diet-sensitive obesity. *Journal of Clinical Investigation*, **2006**, 116, 1886-901	15.9	254
126	A mouse model for the metabolic effects of the human fat mass and obesity associated FTO gene. *PLoS Genetics*, **2009**, 5, e1000599	6	245

125	Control of pancreatic β-cell regeneration by glucose metabolism. *Cell Metabolism*, **2011**, 13, 440-449	24.6	229
124	A novel method for measurement of submembrane ATP concentration. *Journal of Biological Chemistry*, **2000**, 275, 30046-9	5.4	220
123	The sulfonylurea receptor. *Biochimica Et Biophysica Acta - Molecular Cell Research*, **1992**, 1175, 45-59	4.9	207
122	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, 17539-44	11.5	205
121	The ATP-sensitivity of K+ channels in rat pancreatic B-cells is modulated by ADP. *FEBS Letters*, **1986**, 208, 63-6	3.8	201
120	Overlapping distribution of K(ATP) channel-forming Kir6.2 subunit and the sulfonylurea receptor SUR1 in rodent brain. *FEBS Letters*, **1997**, 401, 59-64	3.8	198
119	A heterozygous activating mutation in the sulphonylurea receptor SUR1 (ABCC8) causes neonatal diabetes. *Human Molecular Genetics*, **2006**, 15, 1793-800	5.6	175
118	Functional analysis of a structural model of the ATP-binding site of the KATP channel Kir6.2 subunit. *EMBO Journal*, **2005**, 24, 229-39	13	169
117	K(ATP) channels and islet hormone secretion: new insights and controversies. *Nature Reviews Endocrinology*, **2013**, 9, 660-9	15.2	166
116	Relapsing diabetes can result from moderately activating mutations in KCNJ11. *Human Molecular Genetics*, **2005**, 14, 925-34	5.6	165
115	Permanent neonatal diabetes caused by dominant, recessive, or compound heterozygous SUR1 mutations with opposite functional effects. *American Journal of Human Genetics*, **2007**, 81, 375-82	11	161
114	New windows on the mechanism of action of K(ATP) channel openers. *Trends in Pharmacological Sciences*, **2000**, 21, 439-45	13.2	159
113	Molecular analysis of ATP-sensitive K channel gating and implications for channel inhibition by ATP. *Journal of General Physiology*, **1998**, 112, 333-49	3.4	156
112	Reversible changes in pancreatic islet structure and function produced by elevated blood glucose. *Nature Communications*, **2014**, 5, 4639	17.4	153
111	3-D structural and functional characterization of the purified KATP channel complex Kir6.2-SUR1. *EMBO Journal*, **2005**, 24, 4166-75	13	147
110	Role of KATP channels in glucose-regulated glucagon secretion and impaired counterregulation in type 2 diabetes. *Cell Metabolism*, **2013**, 18, 871-82	24.6	146
109	Promiscuous coupling between the sulphonylurea receptor and inwardly rectifying potassium channels. *Nature*, **1996**, 379, 545-8	50.4	144
108	A new subtype of autosomal dominant diabetes attributable to a mutation in the gene for sulfonylurea receptor 1. *Lancet, The*, **2003**, 361, 301-7	40	143

107	FTO influences adipogenesis by regulating mitotic clonal expansion. *Nature Communications*, **2015**, 6, 6792	17.4	142
106	Simultaneous recordings of glucose dependent electrical activity and ATP-regulated K(+)-currents in isolated mouse pancreatic beta-cells. *FEBS Letters*, **1990**, 261, 187-90	3.8	140
105	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 (Pt 1), 35-45	3.9	136
104	The role of the KATP channel in glucose homeostasis in health and disease: more than meets the islet. *Journal of Physiology*, **2010**, 588, 3201-9	3.9	123
103	Review. 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, 257-67	5.8	118
102	Chronic palmitate exposure inhibits insulin secretion by dissociation of Ca(2+) channels from secretory granules. *Cell Metabolism*, **2009**, 10, 455-65	24.6	116
101	PIP(2)-binding site in Kir channels: definition by multiscale biomolecular simulations. *Biochemistry*, **2009**, 48, 10926-33	3.2	108
100	Adult onset global loss of the fto gene alters body composition and metabolism in the mouse. *PLoS Genetics*, **2013**, 9, e1003166	6	103
99	Mechanism of cloned ATP-sensitive potassium channel activation by oleoyl-CoA. *Journal of Biological Chemistry*, **1998**, 273, 26383-7	5.4	103
98	Diabetes causes marked inhibition of mitochondrial metabolism in pancreatic □cells. *Nature Communications*, **2019**, 10, 2474	17.4	102
97	Type 2 diabetes and congenital hyperinsulinism cause DNA double-strand breaks and p53 activity in □cells. *Cell Metabolism*, **2014**, 19, 109-21	24.6	101
96	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. *EMBO Reports*, **2005**, 6, 470-5	6.5	95
95	The Walter B. Cannon Physiology in Perspective Lecture, 2007. ATP-sensitive K+ channels and disease: from molecule to malady. *American Journal of Physiology - Endocrinology and Metabolism*, **2007**, 293, E880-9	6	91
94	Sulfonylurea improves CNS function in a case of intermediate DEND syndrome caused by a mutation in KCNJ11. *Nature Clinical Practice Neurology*, **2007**, 3, 640-5		88
93	Expression of an activating mutation in the gene encoding the KATP channel subunit Kir6.2 in mouse pancreatic beta cells recapitulates neonatal diabetes. *Journal of Clinical Investigation*, **2009**, 119, 80-90	15.9	86
92	Mutations within the P-loop of Kir6.2 modulate the intraburst kinetics of the ATP-sensitive potassium channel. *Journal of General Physiology*, **2001**, 118, 341-53	3.4	85
91	Differential interactions of nateglinide and repaglinide on the human beta-cell sulphonylurea receptor 1. *Diabetes*, **2002**, 51, 2789-95	0.9	85
90	Direct photoaffinity labeling of the Kir6.2 subunit of the ATP-sensitive K+ channel by 8-azido-ATP. *Journal of Biological Chemistry*, **1999**, 274, 3931-3	5.4	85

89	Involvement of the n-terminus of Kir6.2 in coupling to the sulphonylurea receptor. *Journal of Physiology*, **1999**, 518 (Pt 2), 325-36	3.9	81
88	Type 2 diabetes mellitus: not quite exciting enough?. *Human Molecular Genetics*, **2004**, 13 Spec No 1, R21-31	5.6	78
87	Muscle dysfunction caused by a KATP channel mutation in neonatal diabetes is neuronal in origin. *Science*, **2010**, 329, 458-61	33.3	77
86	Functional effects of KCNJ11 mutations causing neonatal diabetes: enhanced activation by MgATP. *Human Molecular Genetics*, **2005**, 14, 2717-26	5.6	72
85	Identification of the PIP2-binding site on Kir6.2 by molecular modelling and functional analysis. *EMBO Journal*, **2007**, 26, 3749-59	13	69
84	Hyperglycaemia induces metabolic dysfunction and glycogen accumulation in pancreatic Ecells. *Nature Communications*, **2016**, 7, 13496	17.4	67
83	Identification of residues contributing to the ATP binding site of Kir6.2. *EMBO Journal*, **2003**, 22, 2903-12	13	63
82	Q&A: insulin secretion and type 2 diabetes: why do Ecells fail?. *BMC Biology*, **2015**, 13, 33	7.3	62
81	ATP sensitivity of the ATP-sensitive K+ channel in intact and permeabilized pancreatic beta-cells. *Diabetes*, **2006**, 55, 2446-54	0.9	62
80	Na+ current properties in islet Eand Ecells reflect cell-specific Scn3a and Scn9a expression. *Journal of Physiology*, **2014**, 592, 4677-96	3.9	60
79	FTO is expressed in neurones throughout the brain and its expression is unaltered by fasting. *PLoS ONE*, **2011**, 6, e27968	3.7	60
78	Studies of the ATPase activity of the ABC protein SUR1. *FEBS Journal*, **2007**, 274, 3532-3544	5.7	59
77	Mutations at the same residue (R50) of Kir6.2 (KCNJ11) that cause neonatal diabetes produce different functional effects. *Diabetes*, **2006**, 55, 1705-12	0.9	56
76	Kir6.2 mutations causing neonatal diabetes provide new insights into Kir6.2-SUR1 interactions. *EMBO Journal*, **2005**, 24, 2318-30	13	56
75	Successful transfer to sulfonylureas in KCNJ11 neonatal diabetes is determined by the mutation and duration of diabetes. *Diabetologia*, **2016**, 59, 1162-6	10.3	54
74	Neonatal Diabetes and the K Channel: From Mutation to Therapy. *Trends in Endocrinology and Metabolism*, **2017**, 28, 377-387	8.8	53
73	Focus on Kir6.2: a key component of the ATP-sensitive potassium channel. *Journal of Molecular and Cellular Cardiology*, **2005**, 38, 927-36	5.8	52
72	Systemic Administration of Glibenclamide Fails to Achieve Therapeutic Levels in the Brain and Cerebrospinal Fluid of Rodents. *PLoS ONE*, **2015**, 10, e0134476	3.7	51

71	Modeling K(ATP) channel gating and its regulation. *Progress in Biophysics and Molecular Biology*, **2009**, 99, 7-19	4.7	49
70	Mechanism of action of a sulphonylurea receptor SUR1 mutation (F132L) that causes DEND syndrome. *Human Molecular Genetics*, **2007**, 16, 2011-9	5.6	49
69	The ligand-sensitive gate of a potassium channel lies close to the selectivity filter. *EMBO Reports*, **2003**, 4, 70-5	6.5	49
68	Functional analysis of six Kir6.2 (KCNJ11) mutations causing neonatal diabetes. *Pflugers Archiv European Journal of Physiology*, **2006**, 453, 323-32	4.6	48
67	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, 18988-92	11.5	47
66	Differential response of K(ATP) channels containing SUR2A or SUR2B subunits to nucleotides and pinacidil. *Molecular Pharmacology*, **2000**, 58, 1318-25	4.3	47
65	How ATP inhibits the open K(ATP) channel. *Journal of General Physiology*, **2008**, 132, 131-44	3.4	43
64	New insights into K channel gene mutations and neonatal diabetes mellitus. *Nature Reviews Endocrinology*, **2020**, 16, 378-393	15.2	41
63	Is Type 2 Diabetes a Glycogen Storage Disease of Pancreatic □Cells?. *Cell Metabolism*, **2017**, 26, 17-23	24.6	39
62	New uses for old drugs: neonatal diabetes and sulphonylureas. *Cell Metabolism*, **2010**, 11, 179-81	24.6	39
61	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, 1731-7	0.9	39
60	Activation of the K(ATP) channel by Mg-nucleotide interaction with SUR1. *Journal of General Physiology*, **2010**, 136, 389-405	3.4	37
59	A Kir6.2 mutation causing neonatal diabetes impairs electrical activity and insulin secretion from INS-1 beta-cells. *Diabetes*, **2006**, 55, 3075-82	0.9	37
58	Molecular mechanism of sulphonylurea block of K(ATP) channels carrying mutations that impair ATP inhibition and cause neonatal diabetes. *Diabetes*, **2013**, 62, 3909-19	0.9	36
57	Pancreatic □Cells Express the Fetal Islet Hormone Gastrin in Rodent and Human Diabetes. *Diabetes* , **2017**, 66, 426-436	0.9	36
56	Expression of functionally active ATP-sensitive K-channels in insect cells using baculovirus. *FEBS Letters*, **1998**, 429, 390-4	3.8	35
55	A mutation (R826W) in nucleotide-binding domain 1 of ABCC8 reduces ATPase activity and causes transient neonatal diabetes. *EMBO Reports*, **2008**, 9, 648-54	6.5	35
54	Direct photoaffinity labeling of Kir6.2 by [gamma-(32)P]ATP-[gamma]4-azidoanilide. *Biochemical and Biophysical Research Communications*, **2000**, 272, 316-9	3.4	35

53	Fumarate Hydratase Deletion in Pancreatic □Cells Leads to Progressive Diabetes. *Cell Reports*, **2017**, 20, 3135-3148	10.6	34
52	Adjacent mutations in the gating loop of Kir6.2 produce neonatal diabetes and hyperinsulinism. *EMBO Molecular Medicine*, **2009**, 1, 166-77	12	34
51	Functional identification of islet cell types by electrophysiological fingerprinting. *Journal of the Royal Society Interface*, **2017**, 14,	4.1	33
50	Modification of K-ATP channels in pancreatic beta-cells by trypsin. *Pflugers Archiv European Journal of Physiology*, **1993**, 424, 63-72	4.6	33
49	Altered functional properties of KATP channel conferred by a novel splice variant of SUR1. *Journal of Physiology*, **1999**, 521 Pt 2, 337-50	3.9	31
48	Kir6.2-dependent high-affinity repaglinide binding to beta-cell K(ATP) channels. *British Journal of Pharmacology*, **2005**, 144, 551-7	8.6	30
47	Introduction. The blurred boundary between channels and transporters. *Philosophical Transactions of the Royal Society B: Biological Sciences*, **2009**, 364, 145-7	5.8	29
46	Mapping the architecture of the ATP-binding site of the KATP channel subunit Kir6.2. *Journal of Physiology*, **2004**, 557, 347-54	3.9	29
45	Expression of voltage-gated K+ channels in insulin-producing cells. Analysis by polymerase chain reaction. *FEBS Letters*, **1990**, 263, 121-6	3.8	28
44	Changes in gene expression associated with FTO overexpression in mice. *PLoS ONE*, **2014**, 9, e97162	3.7	27
43	A mouse model of human hyperinsulinism produced by the E1506K mutation in the sulphonylurea receptor SUR1. *Diabetes*, **2013**, 62, 3797-806	0.9	25
42	Functional effects of naturally occurring KCNJ11 mutations causing neonatal diabetes on cloned cardiac KATP channels. *Journal of Physiology*, **2006**, 571, 3-14	3.9	25
41	Pharmacological inhibition of FTO. *PLoS ONE*, **2015**, 10, e0121829	3.7	25
40	Identification of a functionally important negatively charged residue within the second catalytic site of the SUR1 nucleotide-binding domains. *Diabetes*, **2004**, 53 Suppl 3, S123-7	0.9	24
39	Switching to sulphonylureas in children with iDEND syndrome caused by KCNJ11 mutations results in improved cerebellar perfusion. *Diabetes Care*, **2013**, 36, 2311-6	14.6	23
38	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, 1813-22	0.9	20
37	Analysis of the differential modulation of sulphonylurea block of beta-cell and cardiac ATP-sensitive K+ (K(ATP)) channels by Mg-nucleotides. *Journal of Physiology*, **2003**, 547, 159-68	3.9	19
36	Activation mechanism of ATP-sensitive K channels explored with real-time nucleotide binding. *ELife* , **2019**, 8,	8.9	19

35	Neonatal diabetes caused by a homozygous KCNJ11 mutation demonstrates that tiny changes in ATP sensitivity markedly affect diabetes risk. *Diabetologia*, **2016**, 59, 1430-1436	10.3	18
34	Cardiac Dysfunction and Metabolic Inflexibility in a Mouse Model of Diabetes Without Dyslipidemia. *Diabetes*, **2018**, 67, 1057-1067	0.9	17
33	The Nucleotide-Binding Sites of SUR1: A Mechanistic Model. *Biophysical Journal*, **2015**, 109, 2452-2460	2.9	17
32	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, 469-86	3.4	17
31	Fetal macrosomia and neonatal hyperinsulinemic hypoglycemia associated with transplacental transfer of sulfonylurea in a mother with KCNJ11-related neonatal diabetes. *Diabetes Care*, **2014**, 37, 3333-5	14.6	16
30	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	6.9	16
29	Interaction between mutations in the slide helix of Kir6.2 associated with neonatal diabetes and neurological symptoms. *Human Molecular Genetics*, **2010**, 19, 963-72	5.6	15
28	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, 2551-7	5.6	15
27	A mutation causing increased KATP channel activity leads to reduced anxiety in mice. *Physiology and Behavior*, **2014**, 129, 79-84	3.5	14
26	A conserved tryptophan at the membrane-water interface acts as a gatekeeper for Kir6.2/SUR1 channels and causes neonatal diabetes when mutated. *Journal of Physiology*, **2011**, 589, 3071-83	3.9	13
25	The ATPase activities of sulfonylurea receptor 2A and sulfonylurea receptor 2B are influenced by the C-terminal 42 amino acids. *FEBS Journal*, **2010**, 277, 2654-2662	5.7	12
24	Mosaic paternal uniparental isodisomy and an ABCC8 gene mutation in a patient with permanent neonatal diabetes and hemihypertrophy. *Diabetes*, **2008**, 57, 255-8	0.9	12
23	The ATPase activities of sulfonylurea receptor 2A and sulfonylurea receptor 2B are influenced by the C-terminal 42 amino acids. *FEBS Journal*, **2010**, 277, 2654-62	5.7	12
22	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,	5.8	11
21	Monitoring real-time hormone release kinetics via high-content 3-D imaging of compensatory endocytosis. *Lab on A Chip*, **2018**, 18, 2838-2848	7.2	10
20	Binding of sulphonylureas to plasma proteins - A KATP channel perspective. *PLoS ONE*, **2018**, 13, e0197634	3.7	10
19	A universally conserved residue in the SUR1 subunit of the KATP channel is essential for translating nucleotide binding at SUR1 into channel opening. *Journal of Physiology*, **2012**, 590, 5025-36	3.9	10
18	An Nonsense Mutation Causing Neonatal Diabetes Through Altered Transcript Expression. *JCRPE Journal of Clinical Research in Pediatric Endocrinology*, **2017**, 9, 260-264	1.9	10

17	Magnesium deficiency prevents high-fat-diet-induced obesity in mice. *Diabetologia*, **2018**, 61, 2030-2042	10.3	9
16	Nucleotide inhibition of the pancreatic ATP-sensitive K+ channel explored with patch-clamp fluorometry. *ELife*, **2020**, 9,	8.9	9
15	Increased NEFA levels reduce blood Mg in hypertriacylglycerolaemic states via direct binding of NEFA to Mg. *Diabetologia*, **2019**, 62, 311-321	10.3	9
14	Tetrameric structure of SUR2B revealed by electron microscopy of oriented single particles. *FEBS Journal*, **2013**, 280, 1051-63	5.7	8
13	Evaluating inositol phospholipid interactions with inward rectifier potassium channels and characterising their role in disease. *Communications Chemistry*, **2020**, 3,	6.3	8
12	Low extracellular magnesium does not impair glucose-stimulated insulin secretion. *PLoS ONE*, **2019**, 14, e0217925	3.7	6
11	Gain-of-function mutations in the K(ATP) channel (KCNJ11) impair coordinated hand-eye tracking. *PLoS ONE*, **2013**, 8, e62646	3.7	6
10	The Gene Variant Hastens Diabetes Progression by Impairing Glucose-Induced Insulin Secretion. *Diabetes*, **2021**, 70, 1145-1156	0.9	6
9	The value of in□vitro studies in a case of neonatal diabetes with a novel Kir6.2-W68G mutation. *Clinical Case Reports (discontinued)*, **2015**, 3, 884-7	0.7	4
8	Role of the C-terminus of SUR in the differential regulation of □cell and cardiac K channels by MgADP and metabolism. *Journal of Physiology*, **2018**, 596, 6205-6217	3.9	3
7	Mouse Models of □cell K Channel Dysfunction. *Drug Discovery Today: Disease Models*, **2013**, 10, e101-e109	1.3	2
6	A cytosolic factor that inhibits KATP channels expressed in Xenopus oocytes by impairing Mg-nucleotide activation by SUR1. *Journal of Physiology*, **2009**, 587, 1649-56	3.9	2
5	Evaluating Inositol phospholipid interactions with Inward Rectifier Potassium Channels and characterising their role in Disease		1
4	The dynamic interplay of PIP2 and ATP in the regulation of the KATP channel		1
3	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. *Wellcome Open Research*, **2020**, 5, 15	4.8	0
2	Phenotype of a transient neonatal diabetes point mutation (SUR1-R1183W) in mice. *Wellcome Open Research*, **2020**, 5, 15	4.8	
1	Influences: Find a friend. *Journal of General Physiology*, **2018**, 150, 895-896	3.4	