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List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	<i>De novo</i> point mutations in patients diagnosed with ataxic cerebral palsy. Brain, 2015, 138, 1817-1832.	3.7	129
2	pH Dependence of the Inwardly Rectifying Potassium Channel, Kir5.1, and Localization in Renal Tubular Epithelia. Journal of Biological Chemistry, 2000, 275, 16404-16407.	1.6	114
3	Differential pH sensitivity of Kir4.1 and Kir4.2 potassium channels and their modulation by heteropolymerisation with Kir5.1. Journal of Physiology, 2001, 532, 359-367.	1.3	112
4	Autism with Seizures and Intellectual Disability: Possible Causative Role of Gain-of-function of the Inwardly-Rectifying K+ Channel Kir4.1. Neurobiology of Disease, 2011, 43, 239-247.	2.1	108
5	Thymosin α1 represents a potential potent single-molecule-based therapy for cystic fibrosis. Nature Medicine, 2017, 23, 590-600.	15.2	91
6	K+ channelepsy: progress in the neurobiology of potassium channels and epilepsy. Frontiers in Cellular Neuroscience, 2013, 7, 134.	1.8	84
7	Episodic ataxia type-1 mutations in the hKv1.1 cytoplasmic pore region alter the gating properties of the channel. EMBO Journal, 1998, 17, 1200-1207.	3.5	76
8	Mutations in the <i>KCNA1</i> gene associated with episodic ataxia typeâ€1 syndrome impair heteromeric voltageâ€gated K ⁺ channel function. FASEB Journal, 1999, 13, 1335-1345.	0.2	75
9	Genetically induced dysfunctions of Kir2.1 channels: implications for short QT3 syndrome and autism–epilepsy phenotype. Human Molecular Genetics, 2014, 23, 4875-4886.	1.4	65
10	Dexamethasone in Glioblastoma Multiforme Therapy: Mechanisms and Controversies. Frontiers in Molecular Neuroscience, 2019, 12, 65.	1.4	64
11	Update on the implication of potassium channels in autism: K+ channelautism spectrum disorder. Frontiers in Cellular Neuroscience, 2015, 9, 34.	1.8	63
12	Gain-of-function defects of astrocytic Kir4.1 channels in children with autism spectrum disorders and epilepsy. Scientific Reports, 2016, 6, 34325.	1.6	56
13	Kv1.1 Channelopathies: Pathophysiological Mechanisms and Therapeutic Approaches. International Journal of Molecular Sciences, 2020, 21, 2935.	1.8	55
14	New insights into the pathogenesis and therapeutics of episodic ataxia type 1. Frontiers in Cellular Neuroscience, 2015, 9, 317.	1.8	54
15	Episodic ataxia type 1 mutations in theKCNA1gene impair the fast inactivation properties of the human potassium channels Kv1.4-1.1/Kvl²1.1 and Kv1.4-1.1/Kvl²1.2. European Journal of Neuroscience, 2006, 24, 3073-3083.	1.2	50
16	Genetic Inactivation of Kcnj16 Identifies Kir5.1 as an Important Determinant of Neuronal PCO2/pH Sensitivity. Journal of Biological Chemistry, 2011, 286, 192-198.	1.6	43
17	Identification of a Heteromeric Interaction That Influences the Rectification, Gating, and pH Sensitivity of Kir4.1/Kir5.1 Potassium Channels. Journal of Biological Chemistry, 2003, 278, 43533-43540.	1.6	42
18	Novel phenotype associated with a mutation in the KCNA1(Kv1.1) gene. Frontiers in Physiology, 2014, 5, 525.	1.3	42

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19	Localization and age-dependent expression of the inward rectifier K+ channel subunit Kir 5.1 in a mammalian reproductive system. FEBS Letters, 1999, 449, 146-152.	1.3	41
20	A novel KCNA1 mutation identified in an Italian family affected by episodic ataxia type 1. Neuroscience, 2008, 157, 577-587.	1.1	39
21	Kv1.1 knock-in ataxic mice exhibit spontaneous myokymic activity exacerbated by fatigue, ischemia and low temperature. Neurobiology of Disease, 2012, 47, 310-321.	2.1	32
22	Expression in E. coli and purification of recombinant fragments of wild type and mutant human prion protein. Neurochemistry International, 2002, 41, 55-63.	1.9	31
23	Episodic ataxia type 1 mutation F184C alters Zn2+-induced modulation of the human K+ channel Kv1.4-Kv1.1/Kvl²1.1. American Journal of Physiology - Cell Physiology, 2007, 292, C778-C787.	2.1	29
24	Episodic ataxia type 1 mutations affect fast inactivation of K ⁺ channels by a reduction in either subunit surface expression or affinity for inactivation domain. American Journal of Physiology - Cell Physiology, 2011, 300, C1314-C1322.	2.1	28
25	Role of receptor protein tyrosine phosphatase α (RPTPα) and tyrosine phosphorylation in the serotonergic inhibition of voltage-dependent potassium channels. Pflugers Archiv European Journal of Physiology, 2000, 441, 257-262.	1.3	26
26	Functional characterization of an episodic ataxia type-1 mutation occurring in the S1 segment of hKv1.1 channels. Pflugers Archiv European Journal of Physiology, 2003, 446, 373-379.	1.3	25
27	<i>KCNA4</i> deficiency leads to a syndrome of abnormal striatum, congenital cataract and intellectual disability. Journal of Medical Genetics, 2016, 53, 786-792.	1.5	24
28	A novel KCNA1 mutation in a patient with paroxysmal ataxia, myokymia, painful contractures and metabolic dysfunctions. Molecular and Cellular Neurosciences, 2017, 83, 6-12.	1.0	23
29	Neutrophil Derived Cathepsin G Induces Potentially Thrombogenic Changes in Human Endothelial Cells: a Scanning Electron Microscopy Study in Static and Dynamic Conditions. Thrombosis and Haemostasis, 1994, 72, 140-145.	1.8	23
30	Contribution of the central hydrophobic residue in the PXP motif of voltage-dependent K ⁺ channels to S6 flexibility and gating properties. Channels, 2009, 3, 39-45.	1.5	22
31	Reconciling the discrepancies on the involvement of large-conductance Ca2+-activated K channels in glioblastoma cell migration. Frontiers in Cellular Neuroscience, 2015, 9, 152.	1.8	21
32	Ion Channels Involvement in Neurodevelopmental Disorders. Neuroscience, 2020, 440, 337-359.	1.1	21
33	An episodic ataxia type-1 mutation in the S1 segment sensitises the hKv1.1 potassium channel to extracellular Zn2+. FEBS Letters, 2004, 576, 237-244.	1.3	17
34	Lethal digenic mutations in the K ⁺ channels Kir4.1 (<i>KCNJ10</i>) and SLACK (<i>KCNT1</i>) associated with severe-disabling seizures and neurodevelopmental delay. Journal of Neurophysiology, 2017, 118, 2402-2411.	0.9	17
35	The role of ion channels in the hypoxia-induced aggressiveness of glioblastoma. Frontiers in Cellular Neuroscience, 2014, 8, 467.	1.8	16
36	A Calsequestrin-1 Mutation Associated with a Skeletal Muscle Disease Alters Sarcoplasmic Ca2+ Release. PLoS ONE, 2016, 11, e0155516.	1.1	15

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37	A channelopathy mutation in the voltage-sensor discloses contributions of a conserved phenylalanine to gating properties of Kv1.1 channels and ataxia. Scientific Reports, 2017, 7, 4583.	1.6	15
38	Association of A Novel Splice Site Mutation in P/Q-Type Calcium Channels with Childhood Epilepsy and Late-Onset Slowly Progressive Non-Episodic Cerebellar Ataxia. International Journal of Molecular Sciences, 2020, 21, 3810.	1.8	14
39	Antithrombotic Activity of Dermatan Sulphates, Heparins and their Combination in an Animal Model of Arterial Thrombosis. Thrombosis and Haemostasis, 1996, 76, 1102-1107.	1.8	14
40	5-HT2 receptors-mediated modulation of voltage-gated K+ channels and neurophysiopathological correlates. Experimental Brain Research, 2013, 230, 453-462.	0.7	12
41	Altered functional properties of a missense variant in the TRESK K+ channel (KCNK18) associated with migraine and intellectual disability. Pflugers Archiv European Journal of Physiology, 2020, 472, 923-930.	1.3	9
42	A Novel KCNA2 Variant in a Patient with Non-Progressive Congenital Ataxia and Epilepsy: Functional Characterization and Sensitivity to 4-Aminopyridine. International Journal of Molecular Sciences, 2021, 22, 9913.	1.8	9
43	Identification of a New de Novo Mutation Underlying Regressive Episodic Ataxia Type I. Frontiers in Neurology, 2018, 9, 587.	1.1	8
44	Enhanced Vascular Plasminogen Activator (t-PA) Release by Epinephrine in Aged Rats. Thrombosis and Haemostasis, 1995, 73, 841-844.	1.8	8
45	Experimental Arterial Thrombosis in Genetically or Diet Induced Hyperlipidemia in Rats. Thrombosis and Haemostasis, 2001, 86, 1440-1448.	1.8	7
46	Morphological and hemostatic changes in rats with abdominal arterial prosthesis. Thrombosis Research, 1996, 82, 69-77.	0.8	5
47	Musculoskeletal Features without Ataxia Associated with a Novel de novo Mutation in KCNA1 Impairing the Voltage Sensitivity of Kv1.1 Channel. Biomedicines, 2021, 9, 75.	1.4	5
48	Kcnj16 (Kir5.1) Gene Ablation Causes Subfertility and Increases the Prevalence of Morphologically Abnormal Spermatozoa. International Journal of Molecular Sciences, 2021, 22, 5972.	1.8	5
49	Different response of vascular fibrinolysis to adrenergic stimulation in young and aged rats. Fibrinolysis, 1992, 6, 36-38.	0.5	4
50	A method to identify tissue cell subpopulations with distinct multi-molecular profiles from data on co-localization of two markers at a time: the case of sensory ganglia. Journal of Neuroscience Methods, 2014, 224, 88-95.	1.3	4
51	Commentary: A channelopathy mutation in the voltage-sensor discloses contributions of a conserved phenylalanine to gating properties of Kv1.1 channels and ataxia. Frontiers in Cellular Neuroscience, 2018, 12, 174.	1.8	4
52	Effect of aspirin on the fibrinolytic response in perfused rat hindquarters. European Journal of Pharmacology, 1992, 229, 39-44.	1.7	3
53	Expression and function of a CP339,818-sensitive K ⁺ current in a subpopulation of putative nociceptive neurons from adult mouse trigeminal ganglia. Journal of Neurophysiology, 2015, 113, 2653-2665.	0.9	3
54	Electromechanical coupling of the Kv1.1 voltage-gated K+ channel is fine-tuned by the simplest amino acid residue in the S4-S5 linker. Pflugers Archiv European Journal of Physiology, 2020, 472, 899-909.	1.3	3

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55	KCNK18 Biallelic Variants Associated with Intellectual Disability and Neurodevelopmental Disorders Alter TRESK Channel Activity. International Journal of Molecular Sciences, 2021, 22, 6064.	1.8	3
56	Amiloride Inhibits Tissue-type Plasminogen Activator (t-PA) Release from Vascular Endothelium. Thrombosis and Haemostasis, 1995, 74, 808-809.	1.8	1
57	Changes in primary hemostasis during thrombus formation in a model of arterial thrombosis in rats. Thrombosis Research, 1993, 70, S129.	0.8	0
58	Changes of the hemostatic balance in a model of aging in rats. Thrombosis Research, 1993, 70, S143.	0.8	0
59	The adrenergic mechanisms of acute t-PA release in normal and diseased animals. Fibrinolysis, 1993, 7, 33-34.	0.5	0
60	G.P.18.09 Functional characterisation of a novel mutation causing episodic ataxia type 1 occurring in the KCNA1 gene. Neuromuscular Disorders, 2007, 17, 892-893.	0.3	0
61	Animal Models of Episodic Ataxia Type 1 (EA1). , 2015, , 797-807.		0