Paola Imbrici

List of Publications by Year
in descending order

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71 papers	2,077 citations	236925 25 h-index	254184 43 g-index
72 all docs	72 docs citations	72 times ranked	2495 citing authors

#	Article	IF	Citations
1	Dysfunction of the brain calcium channel CaV2.1 in absence epilepsy and episodic ataxia. Brain, 2004, 127, 2682-2692.	7.6	191
2	Major channels involved in neuropsychiatric disorders and therapeutic perspectives. Frontiers in Genetics, 2013, 4, 76.	2.3	124
3	Therapeutic Approaches to Genetic Ion Channelopathies and Perspectives in Drug Discovery. Frontiers in Pharmacology, 2016, 7, 121.	3.5	121
4	pH Dependence of the Inwardly Rectifying Potassium Channel, Kir5.1, and Localization in Renal Tubular Epithelia. Journal of Biological Chemistry, 2000, 275, 16404-16407.	3.4	114
5	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.	2.9	112
6	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.	4.4	108
7	Andersenâ€“Tawil syndrome. Neurology, 2005, 65, 1083-1089.	1.1	77
8	Mutations in the KCNA1 gene associated with episodic ataxia typeâ€1 syndrome impair heteromeric voltageâ€gated K ⁺ channel function. FASEB Journal, 1999, 13, 1335-1345.	0.5	75
9	Kv1.1 Channelopathies: Pathophysiological Mechanisms and Therapeutic Approaches. International Journal of Molecular Sciences, 2020, 21, 2935.	4.1	55
10	ClC-1 chloride channels: state-of-the-art research and future challenges. Frontiers in Cellular Neuroscience, 2015, 09, 156.	3.7	53
11	Late-onset episodic ataxia type 2 due to an in-frame insertion in CACNA1A. Neurology, 2005, 65, 944-946.	1.1	52
12	Episodic ataxia type 1 mutations in theKCNA1gene impair the fast inactivation properties of the human potassium channels Kv1.4-1.1/Kvî²1.1 and Kv1.4-1.1/Kvî²1.2. European Journal of Neuroscience, 2006, 24, 3073-3083.	2.6	50
13	Genetic Inactivation of Kcnj16 Identifies Kir5.1 as an Important Determinant of Neuronal PCO2/pH Sensitivity. Journal of Biological Chemistry, 2011, 286, 192-198.	3.4	43
14	A large cohort of myotonia congenita probands: novel mutations and a high-frequency mutation region in exons 4 and 5 of the CLCN1 gene. Journal of Human Genetics, 2013, 58, 581-587.	2.3	42
15	Novel phenotype associated with a mutation in the KCNA1(Kv1.1) gene. Frontiers in Physiology, 2014, 5, 525.	2.8	42
16	Functional characterization of ClC-1 mutations from patients affected by recessive myotonia congenita presenting with different clinical phenotypes. Experimental Neurology, 2013, 248, 530-540.	4.1	40
17	A novel KCNA1 mutation identified in an Italian family affected by episodic ataxia type 1. Neuroscience, 2008, 157, 577-587.	2.3	39
18	Kv1.1 knock-in ataxic mice exhibit spontaneous myokymic activity exacerbated by fatigue, ischemia and low temperature. Neurobiology of Disease, 2012, 47, 310-321.	4.4	32

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19	Targeting kidney CLC-K channels: Pharmacological profile in a human cell line versus Xenopus oocytes. Biochimica Et Biophysica Acta - Biomembranes, 2014, 1838, 2484-2491.	2.6	32
20	Modification by ageing of the tetrodotoxin-sensitive sodium channels in rat skeletal muscle fibres. Biochimica Et Biophysica Acta - Biomembranes, 1998, 1373, 37-46.	2.6	30
21	Episodic ataxia type 1 mutation F184C alters Zn2+-induced modulation of the human K+ channel Kv1.4-Kv1.1/Kvî²1.1. American Journal of Physiology - Cell Physiology, 2007, 292, C778-C787.	4.6	29
22	Clinical, Molecular, and Functional Characterization of CLCN1 Mutations in Three Families with Recessive Myotonia Congenita. NeuroMolecular Medicine, 2015, 17, 285-296.	3.4	29
23	Skeletal muscle ClC-1 chloride channels in health and diseases. Pflugers Archiv European Journal of Physiology, 2020, 472, 961-975.	2.8	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.	4.6	28
25	Role of receptor protein tyrosine phosphatase Î± (RPTPÎ±) and tyrosine phosphorylation in the serotonergic inhibition of voltage-dependent potassium channels. Pflugers Archiv European Journal of Physiology, 2000, 441, 257-262.	2.8	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.	2.8	25
27	Pharmacogenetics of myotonic hNav1.4 sodium channel variants situated near the fast inactivation gate. Pharmacological Research, 2019, 141, 224-235.	7.1	25
28	Premature stop codons in a facilitating EF-hand splice variant of CaV2.1 cause episodic ataxia type 2. Neurobiology of Disease, 2008, 32, 10-15.	4.4	24
29	ClCâ€1 mutations in myotonia congenita patients: insights into molecular gating mechanisms and genotypeâ€“phenotype correlation. Journal of Physiology, 2015, 593, 4181-4199.	2.9	24
30	Multidisciplinary study of a new CICâ€1 mutation causing myotonia congenita: a paradigm to understand and treat ion channelopathies. FASEB Journal, 2016, 30, 3285-3295.	0.5	24
31	A novel KCNA1 mutation in a patient with paroxysmal ataxia, myokymia, painful contractures and metabolic dysfunctions. Molecular and Cellular Neurosciences, 2017, 83, 6-12.	2.2	23
32	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.	2.8	22
33	Trace amines depress D₂â€autoreceptorâ€mediated responses on midbrain dopaminergic cells. British Journal of Pharmacology, 2010, 160, 1509-1520.	5.4	22
34	Kidney CLC-K chloride channels inhibitors. Journal of Hypertension, 2016, 34, 981-992.	0.5	22
35	Translational approach to address therapy in myotonia permanens due to a new SCN4A mutation. Neurology, 2016, 86, 2100-2108.	1.1	22
36	Ion Channels Involvement in Neurodevelopmental Disorders. Neuroscience, 2020, 440, 337-359.	2.3	21

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37	ATP Sensitive Potassium Channels in the Skeletal Muscle Function: Involvement of the KCNJ11(Kir6.2) Gene in the Determination of Mechanical Warner Bratzer Shear Force. Frontiers in Physiology, 2016, 7, 167.	2.8	20
38	Coexistence of CLCN1 and SCN4A mutations in one family suffering from myotonia. Neurogenetics, 2017, 18, 219-225.	1.4	19
39	Pharmacovigilance database search discloses ClCâ€K channels as a novel target of the AT₁ receptor blockers valsartan and olmesartan. British Journal of Pharmacology, 2017, 174, 1972-1983.	5.4	16
40	Mapping ligand binding pockets in chloride ClCâ€1 channels through an integrated in silico and experimental approach using anthraceneâ€9â€carboxylic acid and niflumic acid. British Journal of Pharmacology, 2018, 175, 1770-1780.	5.4	16
41	Iâ€“J loop involvement in the pharmacological profile of CLC-K channels expressed in Xenopus oocytes. Biochimica Et Biophysica Acta - Biomembranes, 2014, 1838, 2745-2756.	2.6	15
42	The analysis of myotonia congenita mutations discloses functional clusters of amino acids within the CBS2 domain and the C-terminal peptide of the ClC-1 channel. Human Mutation, 2018, 39, 1273-1283.	2.5	15
43	Ion Channels in Drug Discovery and Safety Pharmacology. Methods in Molecular Biology, 2018, 1800, 313-326.	0.9	15
44	Safinamide's potential in treating nondystrophic myotonias: Inhibition of skeletal muscle voltage-gated sodium channels and skeletal muscle hyperexcitability in vitro and in vivo. Experimental Neurology, 2020, 328, 113287.	4.1	15
45	Next-generation sequencing application to investigate skeletal muscle channelopathies in a large cohort of Italian patients. Neuromuscular Disorders, 2021, 31, 336-347.	0.6	13
46	Partial recovery of skeletal muscle sodium channel properties in aged rats chronically treated with growth hormone or the GH-secretagogue hexarelin. Journal of Pharmacology and Experimental Therapeutics, 1998, 286, 903-12.	2.5	13
47	Gain-of-Function STIM1 L96V Mutation Causes Myogenesis Alteration in Muscle Cells From a Patient Affected by Tubular Aggregate Myopathy. Frontiers in Cell and Developmental Biology, 2021, 9, 635063.	3.7	10
48	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.	2.8	9
49	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.	4.1	9
50	Identification of a New de Novo Mutation Underlying Regressive Episodic Ataxia Type I. Frontiers in Neurology, 2018, 9, 587.	2.4	8
51	Alteration of STIM1/Orai1-Mediated SOCE in Skeletal Muscle: Impact in Genetic Muscle Diseases and Beyond. Cells, 2021, 10, 2722.	4.1	7
52	Paving the way for Bartter syndrome type 3 drug discovery: a hope from basic research. Journal of Physiology, 2017, 595, 5403-5404.	2.9	6
53	Functional Study of Novel Barttersâ€™s Syndrome Mutations in ClC-Kb and Rescue by the Accessory Subunit Barttin Toward Personalized Medicine. Frontiers in Pharmacology, 2020, 11, 327.	3.5	6
54	Pathomechanisms of a CLCN1 Mutation Found in a Russian Family Suffering From Becker's Myotonia. Frontiers in Neurology, 2020, 11, 1019.	2.4	5

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55	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.	3.2	5
56	Kcnj16 (Kir5.1) Gene Ablation Causes Subfertility and Increases the Prevalence of Morphologically Abnormal Spermatozoa. International Journal of Molecular Sciences, 2021, 22, 5972.	4.1	5
57	Mutations in MYBPC3 and MYH7 in Association with Brugada Type 1 ECG Pattern: Overlap between Brugada Syndrome and Hypertrophic Cardiomyopathy?. Neurology International, 2021, 11, 139-147.	0.5	5
58	Changes in Expression and Cellular Localization of Rat Skeletal Muscle ClC-1 Chloride Channel in Relation to Age, Myofiber Phenotype and PKC Modulation. Frontiers in Pharmacology, 2020, 11, 714.	3.5	4
59	Functional Characterization of Two Novel Mutations in SCN5A Associated with Brugada Syndrome Identified in Italian Patients. International Journal of Molecular Sciences, 2021, 22, 6513.	4.1	4
60	Increased sarcolemma chloride conductance as one of the mechanisms of action of carbonic anhydrase inhibitors in muscle excitability disorders. Experimental Neurology, 2021, 342, 113758.	4.1	4
61	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.	2.8	3
62	Dysfunction of the brain calcium channel CaV2.1 in absence epilepsy and episodic ataxiaâ€"authors' response. Brain, 2005, 128, E33-E33.	7.6	2
63	Prevalence study of muscle channelopathies in Italy. Neuromuscular Disorders, 2016, 26, S197.	0.6	1
64	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.	0.6	0
65	All-Atom Molecular Dynamics Simulations of the K+ Channel Chimera Kv1.2/Kv2.1. Biophysical Journal, 2010, 98, 519a.	0.5	0
66	The Kir5.1 Potassium Channel is an Important Determinant of Neuronal PCO2/pH Sensitivity. Biophysical Journal, 2011, 100, 430a.	0.5	0
67	Kidney CLC-K Chloride Channels Show Differential Pharmacological Profiles Depending on the Heterologous Expression System. Biophysical Journal, 2013, 104, 628a.	0.5	0
68	G.P.137. Neuromuscular Disorders, 2014, 24, 842.	0.6	0
69	Involvement of Barttin Subunit in Pharmacological Potentiation of CLC-K Channels Expressed in Xenopus Oocytes. Biophysical Journal, 2014, 106, 147a.	0.5	0
70	Kidney CLC-K Chloride Channels Inhibitors: Definition of Novel Structural Requirements and Efficacy in CLC-K Polymorphism Associated with Hypertension. Biophysical Journal, 2015, 108, 587a-588a.	0.5	0
71	Functional and pharmacological characterization of the new M1808I mutation in hNav1.4 found in a patient presenting with myotonia and myasthenia. Neuromuscular Disorders, 2015, 25, S210.	0.6	0