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	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
2	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
3	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
4	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
5	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
6	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
7	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
8	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
9	Alteration of STIM1/Orai1-Mediated SOCE in Skeletal Muscle: Impact in Genetic Muscle Diseases and Beyond. Cells, 2021, 10, 2722.	4.1	7
10	Pathomechanisms of a CLCN1 Mutation Found in a Russian Family Suffering From Becker's Myotonia. Frontiers in Neurology, 2020, 11, 1019.	2.4	5
11	Skeletal muscle ClC-1 chloride channels in health and diseases. Pflugers Archiv European Journal of Physiology, 2020, 472, 961-975.	2.8	29
12	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
13	Ion Channels Involvement in Neurodevelopmental Disorders. Neuroscience, 2020, 440, 337-359.	2.3	21
14	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
15	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
16	Kv1.1 Channelopathies: Pathophysiological Mechanisms and Therapeutic Approaches. International Journal of Molecular Sciences, 2020, 21, 2935.	4.1	55
17	Functional Study of Novel Bartterâ€™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
18	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

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19	Pharmacogenetics of myotonic hNav1.4 sodium channel variants situated near the fast inactivation gate. Pharmacological Research, 2019, 141, 224-235.	7.1	25
20	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
21	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
22	Ion Channels in Drug Discovery and Safety Pharmacology. Methods in Molecular Biology, 2018, 1800, 313-326.	0.9	15
23	Identification of a New de Novo Mutation Underlying Regressive Episodic Ataxia Type I. Frontiers in Neurology, 2018, 9, 587.	2.4	8
24	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
25	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
26	Coexistence of CLCN1 and SCN4A mutations in one family suffering from myotonia. Neurogenetics, 2017, 18, 219-225.	1.4	19
27	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
28	Therapeutic Approaches to Genetic Ion Channelopathies and Perspectives in Drug Discovery. Frontiers in Pharmacology, 2016, 7, 121.	3.5	121
29	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
30	Kidney CLC-K chloride channels inhibitors. Journal of Hypertension, 2016, 34, 981-992.	0.5	22
31	Translational approach to address therapy in myotonia permanens due to a new SCN4A mutation. Neurology, 2016, 86, 2100-2108.	1.1	22
32	Prevalence study of muscle channelopathies in Italy. Neuromuscular Disorders, 2016, 26, S197.	0.6	1
33	Multidisciplinary study of a new ClCâ€1 mutation causing myotonia congenita: a paradigm to understand and treat ion channelopathies. FASEB Journal, 2016, 30, 3285-3295.	0.5	24
34	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
35	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
36	ClC-1 chloride channels: state-of-the-art research and future challenges. Frontiers in Cellular Neuroscience, 2015, 09, 156.	3.7	53

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37	Clinical, Molecular, and Functional Characterization of CLCN1 Mutations in Three Families with Recessive Myotonia Congenita. NeuroMolecular Medicine, 2015, 17, 285-296.	3.4	29
38	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
39	G.P.137. Neuromuscular Disorders, 2014, 24, 842.	0.6	0
40	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
41	Involvement of Barttin Subunit in Pharmacological Potentiation of CLC-K Channels Expressed in Xenopus Oocytes. Biophysical Journal, 2014, 106, 147a.	0.5	0
42	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
43	Novel phenotype associated with a mutation in the KCNA1(Kv1.1) gene. Frontiers in Physiology, 2014, 5, 525.	2.8	42
44	Kidney CLC-K Chloride Channels Show Differential Pharmacological Profiles Depending on the Heterologous Expression System. Biophysical Journal, 2013, 104, 628a.	0.5	0
45	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
46	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
47	Major channels involved in neuropsychiatric disorders and therapeutic perspectives. Frontiers in Genetics, 2013, 4, 76.	2.3	124
48	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
49	The Kir5.1 Potassium Channel is an Important Determinant of Neuronal PCO2/pH Sensitivity. Biophysical Journal, 2011, 100, 430a.	0.5	0
50	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
51	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
52	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
53	Trace amines depress D₂â€autoreceptorâ€mediated responses on midbrain dopaminergic cells. British Journal of Pharmacology, 2010, 160, 1509-1520.	5.4	22
54	All-Atom Molecular Dynamics Simulations of the K+ Channel Chimera Kv1.2/Kv2.1. Biophysical Journal, 2010, 98, 519a.	0.5	0

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55	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
56	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
57	A novel KCNA1 mutation identified in an Italian family affected by episodic ataxia type 1. Neuroscience, 2008, 157, 577-587.	2.3	39
58	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
59	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
60	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
61	Dysfunction of the brain calcium channel CaV2.1 in absence epilepsy and episodic ataxiaâ€"authors' response. Brain, 2005, 128, E33-E33.	7.6	2
62	Andersenâ€"Tawil syndrome. Neurology, 2005, 65, 1083-1089.	1.1	77
63	Late-onset episodic ataxia type 2 due to an in-frame insertion in CACNA1A. Neurology, 2005, 65, 944-946.	1.1	52
64	Dysfunction of the brain calcium channel CaV2.1 in absence epilepsy and episodic ataxia. Brain, 2004, 127, 2682-2692.	7.6	191
65	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
66	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
67	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
68	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
69	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
70	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
71	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