

Vikram G Shakkottai

List of Publications by Year in descending order

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Version: 2024-02-01

68
papers

3,890
citations

159585

30
h-index

138484

58
g-index

75
all docs

75
docs citations

75
times ranked

4651
citing authors

#	ARTICLE	IF	CITATIONS
1	Genetic evaluation of dementia with Lewy bodies implicates distinct disease subgroups. <i>Brain</i> , 2022, 145, 1757-1762.	7.6	17
2	Preliminary Study of Vibrotactile Feedback during Home-Based Balance and Coordination Training in Individuals with Cerebellar Ataxia. <i>Sensors</i> , 2022, 22, 3512.	3.8	2
3	Discovery of Novel Activators of Large-Conductance Calcium-Activated Potassium Channels for the Treatment of Cerebellar Ataxia. <i>Molecular Pharmacology</i> , 2022, 102, 17-28.	2.3	9
4	A Chlorzoxazoneâ€Baclofen Combination Improves Cerebellar Impairment in Spinocerebellar Ataxia Type 1. <i>Movement Disorders</i> , 2021, 36, 622-631.	3.9	25
5	Antisense Oligonucleotide Therapy Targeted Against ATXN3 Improves Potassium Channelâ€Mediated Purkinje Neuron Dysfunction in Spinocerebellar Ataxia Type 3. <i>Cerebellum</i> , 2021, 20, 41-53.	2.5	17
6	Genome sequencing analysis identifies new loci associated with Lewy body dementia and provides insights into its genetic architecture. <i>Nature Genetics</i> , 2021, 53, 294-303.	21.4	198
7	THAP1 modulates oligodendrocyte maturation by regulating ECM degradation in lysosomes. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2021, 118, .	7.1	7
8	Alanyl-tRNA Synthetase 2 (AARS2)-Related Ataxia Without Leukoencephalopathy. <i>Cerebellum</i> , 2020, 19, 154-160.	2.5	15
9	Nicotinamide Pathway-Dependent Sirt1 Activation Restores Calcium Homeostasis to Achieve Neuroprotection in Spinocerebellar Ataxia Type 7. <i>Neuron</i> , 2020, 105, 630-644.e9.	8.1	63
10	Toward allele-specific targeting therapy and pharmacodynamic marker for spinocerebellar ataxia type 3. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	32
11	Altered Capicua expression drives regional Purkinje neuron vulnerability through ion channel gene dysregulation in spinocerebellar ataxia type 1. <i>Human Molecular Genetics</i> , 2020, 29, 3249-3265.	2.9	20
12	Dysphagia in spinocerebellar ataxias type 1, 2, 3 and 6. <i>Journal of the Neurological Sciences</i> , 2020, 415, 116878.	0.6	3
13	In Vivo Molecular Signatures of Cerebellar Pathology in Spinocerebellar Ataxia Type 3. <i>Movement Disorders</i> , 2020, 35, 1774-1786.	3.9	23
14	The impact of ethnicity on the clinical presentations of spinocerebellar ataxia type 3. <i>Parkinsonism and Related Disorders</i> , 2020, 72, 37-43.	2.2	16
15	Multiple system atrophy pathology is associated with primary SjÃ¶grenâ€™s syndrome. <i>JCI Insight</i> , 2020, 5, .	5.0	3
16	The inherited ataxias. , 2020, , 75-97.		0
17	Polyglutamine Repeats in Neurodegenerative Diseases. <i>Annual Review of Pathology: Mechanisms of Disease</i> , 2019, 14, 1-27.	22.4	189
18	Antisense oligonucleotide therapy rescues aggresome formation in a novel spinocerebellar ataxia type 3 human embryonic stem cell line. <i>Stem Cell Research</i> , 2019, 39, 101504.	0.7	35

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19	Moving Towards Therapy in SCA1: Insights from Molecular Mechanisms, Identification of Novel Targets, and Planning for Human Trials. <i>Neurotherapeutics</i> , 2019, 16, 999-1008.	4.4	9
20	Synthetic high-density lipoprotein nanoparticles for the treatment of Niemann-Pick diseases. <i>BMC Medicine</i> , 2019, 17, 200.	5.5	19
21	Tremor in the Degenerative Cerebellum: Towards the Understanding of Brain Circuitry for Tremor. <i>Cerebellum</i> , 2019, 18, 519-526.	2.5	16
22	COQ4 Mutation Leads to Childhood-Onset Ataxia Improved by CoQ10 Administration. <i>Cerebellum</i> , 2019, 18, 665-669.	2.5	24
23	Expanding the genetic basis of ataxia. <i>Nature Genetics</i> , 2019, 51, 580-581.	21.4	5
24	Generation of Spinocerebellar Ataxia Type 2 induced pluripotent stem cell lines, CHOPi002-A and CHOPi003-A, from patients with abnormal CAG repeats in the coding region of the ATXN2 gene. <i>Stem Cell Research</i> , 2019, 34, 101361.	0.7	13
25	Ion channel dysfunction in cerebellar ataxia. <i>Neuroscience Letters</i> , 2019, 688, 41-48.	2.1	52
26	Targeted exome analysis identifies the genetic basis of disease in over 50% of patients with a wide range of ataxia-related phenotypes. <i>Genetics in Medicine</i> , 2019, 21, 195-206.	2.4	65
27	Targeting potassium channels to treat cerebellar ataxia. <i>Annals of Clinical and Translational Neurology</i> , 2018, 5, 297-314.	3.7	50
28	Mutations in <i>VPS13D</i> lead to a new recessive ataxia with spasticity and mitochondrial defects. <i>Annals of Neurology</i> , 2018, 83, 1075-1088.	5.3	122
29	Protein kinase C activity is a protective modifier of Purkinje neuron degeneration in cerebellar ataxia. <i>Human Molecular Genetics</i> , 2018, 27, 1396-1410.	2.9	30
30	Autosomal-dominant cerebellar ataxias. <i>Handbook of Clinical Neurology</i> / Edited By P J Vinken and G W Bruyn, 2018, 147, 173-185.	1.8	29
31	<i>C9orf72</i> repeat expansions as genetic modifiers for depression in spinocerebellar ataxias. <i>Movement Disorders</i> , 2018, 33, 497-498.	3.9	4
32	MTSS1/Src family kinase dysregulation underlies multiple inherited ataxias. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2018, 115, E12407-E12416.	7.1	20
33	Dendritic potassium channel dysfunction may contribute to dendrite degeneration in spinocerebellar ataxia type 1. <i>PLoS ONE</i> , 2018, 13, e0198040.	2.5	21
34	Coordinate regulation of mutant NPC1 degradation by selective ER autophagy and MARCH6-dependent ERAD. <i>Nature Communications</i> , 2018, 9, 3671.	12.8	82
35	Oligonucleotide therapy mitigates disease in spinocerebellar ataxia type 3 mice. <i>Annals of Neurology</i> , 2018, 84, 64-77.	5.3	127
36	Dystonia and ataxia progression in spinocerebellar ataxias. <i>Parkinsonism and Related Disorders</i> , 2017, 45, 75-80.	2.2	39

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37	Polyglutamine spinocerebellar ataxias " from genes to potential treatments. <i>Nature Reviews Neuroscience</i> , 2017, 18, 613-626.	10.2	270
38	Potassium channel dysfunction underlies Purkinje neuron spiking abnormalities in spinocerebellar ataxia type 2. <i>Human Molecular Genetics</i> , 2017, 26, 3935-3945.	2.9	54
39	The Initial Symptom and Motor Progression in Spinocerebellar Ataxias. <i>Cerebellum</i> , 2017, 16, 615-622.	2.5	42
40	Current Opinions and Areas of Consensus on the Role of the Cerebellum in Dystonia. <i>Cerebellum</i> , 2017, 16, 577-594.	2.5	184
41	Postural Tremor and Ataxia Progression in Spinocerebellar Ataxias. <i>Tremor and Other Hyperkinetic Movements</i> , 2017, 7, 492.	2.0	13
42	Heat Shock Protein Beta-1 Modifies Anterior to Posterior Purkinje Cell Vulnerability in a Mouse Model of Niemann-Pick Type C Disease. <i>PLoS Genetics</i> , 2016, 12, e1006042.	3.5	18
43	Single amino acid deletion in transmembrane segment D4S6 of sodium channel Scn8a (Nav1.6) in a mouse mutant with a chronic movement disorder. <i>Neurobiology of Disease</i> , 2016, 89, 36-45.	4.4	23
44	Depression and clinical progression in spinocerebellar ataxias. <i>Parkinsonism and Related Disorders</i> , 2016, 22, 87-92.	2.2	85
45	Precision medicine in spinocerebellar ataxias: treatment based on common mechanisms of disease. <i>Annals of Translational Medicine</i> , 2016, 4, 25.	1.7	22
46	Coenzyme Q10 and spinocerebellar ataxias. <i>Movement Disorders</i> , 2015, 30, 214-220.	3.9	36
47	Neuronal Atrophy Early in Degenerative Ataxia Is a Compensatory Mechanism to Regulate Membrane Excitability. <i>Journal of Neuroscience</i> , 2015, 35, 11292-11307.	3.6	93
48	Vascular risk factors and clinical progression in spinocerebellar ataxias. <i>Tremor and Other Hyperkinetic Movements</i> , 2015, 5, 287.	2.0	5
49	The Role for Alterations in Neuronal Activity in the Pathogenesis of Polyglutamine Repeat Disorders. <i>Neurotherapeutics</i> , 2014, 11, 751-763.	4.4	22
50	Translating cerebellar Purkinje neuron physiology to progress in dominantly inherited ataxia. <i>Future Neurology</i> , 2014, 9, 187-196.	0.5	27
51	Alterations in cerebellar physiology are associated with a stiff-legged gait in Atcyjji-hes mice. <i>Neurobiology of Disease</i> , 2014, 67, 140-148.	4.4	20
52	Physiologic Changes Associated with Cerebellar Dystonia. <i>Cerebellum</i> , 2014, 13, 637-644.	2.5	21
53	Clinical Neurogenetics. <i>Neurologic Clinics</i> , 2013, 31, 987-1007.	1.8	78
54	Clinical characteristics of patients with spinocerebellar ataxias 1, 2, 3 and 6 in the US; a prospective observational study. <i>Orphanet Journal of Rare Diseases</i> , 2013, 8, 177.	2.7	117

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55	Toward RNAi Therapy for the Polyglutamine Disease Machado-Joseph Disease. <i>Molecular Therapy</i> , 2013, 21, 1898-1908.	8.2	102
56	Mutations in <i>KCND3</i> cause spinocerebellar ataxia type 22. <i>Annals of Neurology</i> , 2012, 72, 859-869.	5.3	138
57	Temporal and cell-specific deletion establishes that neuronal <i>Npc1</i> deficiency is sufficient to mediate neurodegeneration. <i>Human Molecular Genetics</i> , 2011, 20, 4440-4451.	2.9	53
58	Early Changes in Cerebellar Physiology Accompany Motor Dysfunction in the Polyglutamine Disease Spinocerebellar Ataxia Type 3. <i>Journal of Neuroscience</i> , 2011, 31, 13002-13014.	3.6	190
59	Conditional Niemann-Pick C mice demonstrate cell autonomous Purkinje cell neurodegeneration. <i>Human Molecular Genetics</i> , 2010, 19, 837-847.	2.9	123
60	FGF14 regulates the intrinsic excitability of cerebellar Purkinje neurons. <i>Neurobiology of Disease</i> , 2009, 33, 81-88.	4.4	112
61	Physiologic Alterations in Ataxia. <i>Archives of Neurology</i> , 2009, 66, 1196-201.	4.5	25
62	Modulators of Small- and Intermediate-Conductance Calcium-Activated Potassium Channels and their Therapeutic Indications. <i>Current Medicinal Chemistry</i> , 2007, 14, 1437-1457.	2.4	189
63	SKCa Channels Mediate the Medium But Not the Slow Calcium-Activated Afterhyperpolarization in Cortical Neurons. <i>Journal of Neuroscience</i> , 2004, 24, 3537-3542.	3.6	113
64	SK3-1C, a Dominant-negative Suppressor of SKCa and IKCa Channels. <i>Journal of Biological Chemistry</i> , 2004, 279, 6893-6904.	3.4	34
65	Enhanced neuronal excitability in the absence of neurodegeneration induces cerebellar ataxia. <i>Journal of Clinical Investigation</i> , 2004, 113, 582-590.	8.2	94
66	Enhanced neuronal excitability in the absence of neurodegeneration induces cerebellar ataxia. <i>Journal of Clinical Investigation</i> , 2004, 113, 582-590.	8.2	60
67	Design and Characterization of a Highly Selective Peptide Inhibitor of the Small Conductance Calcium-activated K ⁺ Channel, <i>SkCa2</i> . <i>Journal of Biological Chemistry</i> , 2001, 276, 43145-43151.	3.4	106
68	Vulnerability of Human Cerebellar Neurons to Degeneration in Ataxia-Causing Channelopathies. <i>Frontiers in Systems Neuroscience</i> , 0, 16, .	2.5	1