

John Ravits

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/11693162/publications.pdf>

Version: 2024-02-01

35
papers

6,826
citations

257450

24
h-index

395702

33
g-index

36
all docs

36
docs citations

36
times ranked

8061
citing authors

#	ARTICLE	IF	CITATIONS
1	Nuclear RIPK1 promotes chromatin remodeling to mediate inflammatory response. Cell Research, 2022, 32, 621-637.	12.0	18
2	Cross-Comparison of Human iPSC Motor Neuron Models of Familial and Sporadic ALS Reveals Early and Convergent Transcriptomic Disease Signatures. Cell Systems, 2021, 12, 159-175.e9.	6.2	33
3	Unraveling molecular biology of C9ORF72 repeat expansions in amyotrophic lateral sclerosis-frontotemporal dementia: Implications for therapy. , 2021, , 19-47.		0
4	An integrated multi-omic analysis of iPSC-derived motor neurons from C9ORF72 ALS patients. IScience, 2021, 24, 103221.	4.1	27
5	Delivering Bad News in Amyotrophic Lateral Sclerosis. Neurology: Clinical Practice, 2021, 11, 521-526.	1.6	3
6	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. Nature Neuroscience, 2020, 23, 615-624.	14.8	157
7	Predicting disease specific spinal motor neurons and glia in sporadic ALS. Neurobiology of Disease, 2019, 130, 104523.	4.4	10
8	Pathogenic Mechanisms and Therapy Development for C9orf72 Amyotrophic Lateral Sclerosis/Frontotemporal Dementia. Neurotherapeutics, 2019, 16, 1115-1132.	4.4	30
9	Antisense RNA foci are associated with nucleoli and TDP-43 mislocalization in C9orf72-ALS/FTD: a quantitative study. Acta Neuropathologica, 2019, 137, 527-530.	7.7	21
10	Premature polyadenylation-mediated loss of stathmin-2 is a hallmark of TDP-43-dependent neurodegeneration. Nature Neuroscience, 2019, 22, 180-190.	14.8	345
11	Sense-encoded poly-GR dipeptide repeat proteins correlate to neurodegeneration and uniquely co-localize with TDP-43 in dendrites of repeat-expanded C9orf72 amyotrophic lateral sclerosis. Acta Neuropathologica, 2018, 135, 459-474.	7.7	152
12	Kinnier Wilson's puzzling features of amyotrophic lateral sclerosis. Journal of Neurology, Neurosurgery and Psychiatry, 2018, 89, 657-666.	1.9	4
13	Transcriptome-pathology correlation identifies interplay between TDP-43 and the expression of its kinase CK1E in sporadic ALS. Acta Neuropathologica, 2018, 136, 405-423.	7.7	69
14	TDP-43 protein variants as biomarkers in amyotrophic lateral sclerosis. BMC Neuroscience, 2017, 18, 20.	1.9	27
15	Pathological TDP-43 changes in Betz cells differ from those in bulbar and spinal α -motoneurons in sporadic amyotrophic lateral sclerosis. Acta Neuropathologica, 2017, 133, 79-90.	7.7	68
16	Distinct and shared functions of ALS-associated proteins TDP-43, FUS and TAF15 revealed by multisystem analyses. Nature Communications, 2016, 7, 12143.	12.8	137
17	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. Neuron, 2016, 90, 535-550.	8.1	437
18	RIPK1 mediates axonal degeneration by promoting inflammation and necroptosis in ALS. Science, 2016, 353, 603-608.	12.6	448

#	ARTICLE	IF	CITATIONS
19	Sleep Apnea in Familial Dysautonomia: A Reflection of Apnea Pathogenesis. Journal of Clinical Sleep Medicine, 2016, 12, 1583-1584.	2.6	0
20	Neuropathology of Amyotrophic Lateral Sclerosis and Its Variants. Neurologic Clinics, 2015, 33, 855-876.	1.8	199
21	Focality, stochasticity and neuroanatomic propagation in ALS pathogenesis. Experimental Neurology, 2014, 262, 121-126.	4.1	81
22	Targeting RNA Foci in iPSC-Derived Motor Neurons from ALS Patients with a <i>C9ORF72</i> Repeat Expansion. Science Translational Medicine, 2013, 5, 208ra149.	12.4	586
23	Controversies and priorities in amyotrophic lateral sclerosis. Lancet Neurology, The, 2013, 12, 310-322.	10.2	454
24	Lack of C9ORF72 coding mutations supports a gain of function for repeat expansions in amyotrophic lateral sclerosis. Neurobiology of Aging, 2013, 34, 2234.e13-2234.e19.	3.1	59
25	Targeted degradation of sense and antisense <i>C9orf72</i> RNA foci as therapy for ALS and frontotemporal degeneration. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E4530-9.	7.1	508
26	Deciphering amyotrophic lateral sclerosis: What phenotype, neuropathology and genetics are telling us about pathogenesis. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2013, 14, 5-18.	1.7	142
27	Aberrant Neuregulin 1 Signaling in Amyotrophic Lateral Sclerosis. Journal of Neuropathology and Experimental Neurology, 2012, 71, 104-115.	1.7	62
28	Divergent roles of ALS-linked proteins FUS/TLS and TDP-43 intersect in processing long pre-mRNAs. Nature Neuroscience, 2012, 15, 1488-1497.	14.8	628
29	Sporadic ALS has compartment-specific aberrant exon splicing and altered cellâ€matrix adhesion biology. Human Molecular Genetics, 2010, 19, 313-328.	2.9	114
30	TDP-43 and ubiquitinated cytoplasmic aggregates in sporadic ALS are low frequency and widely distributed in the lower motor neuron columns independent of disease spread. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders, 2010, 11, 321-327.	2.1	20
31	Implications of ALS focality: Rostral-caudal distribution of lower motor neuron loss postmortem. Neurology, 2007, 68, 1576-1582.	1.1	142
32	Pathological TDPâ€43 distinguishes sporadic amyotrophic lateral sclerosis from amyotrophic lateral sclerosis with <i>SOD1</i> mutations. Annals of Neurology, 2007, 61, 427-434.	5.3	840
33	Efficacy of minocycline in patients with amyotrophic lateral sclerosis: a phase III randomised trial. Lancet Neurology, The, 2007, 6, 1045-1053.	10.2	610
34	Clinical and electromyographic studies of postpoliomyelitis muscular atrophy. Muscle and Nerve, 1990, 13, 667-674.	2.2	44
35	A Long-Term Follow-up Study of Patients with Post-Poliomyelitis Neuromuscular Symptoms. New England Journal of Medicine, 1986, 314, 959-963.	27.0	350