

John M Ravits

List of Publications by Year in descending order

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Version: 2024-02-01

42
papers

7,695
citations

218381

26
h-index

315357

38
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46
all docs

46
docs citations

46
times ranked

8876
citing authors

#	ARTICLE	IF	CITATIONS
1	Aberrant NOVA1 function disrupts alternative splicing in early stages of amyotrophic lateral sclerosis. <i>Acta Neuropathologica</i> , 2022, 144, 413-435.	3.9	11
2	Cross-Comparison of Human iPSC Motor Neuron Models of Familial and Sporadic ALS Reveals Early and Convergent Transcriptomic Disease Signatures. <i>Cell Systems</i> , 2021, 12, 159-175.e9.	2.9	33
3	Nucleolar stress in C9orf72 and sporadic ALS spinal motor neurons precedes TDP-43 mislocalization. <i>Acta Neuropathologica Communications</i> , 2021, 9, 26.	2.4	14
4	Novel STMN2 Variant Linked to Amyotrophic Lateral Sclerosis Risk and Clinical Phenotype. <i>Frontiers in Aging Neuroscience</i> , 2021, 13, 658226.	1.7	38
5	Unraveling molecular biology of C9ORF72 repeat expansions in amyotrophic lateral sclerosis-frontotemporal dementia: Implications for therapy. , 2021, , 19-47.		0
6	Nuclear depletion of RNA-binding protein ELAVL3 (HuC) in sporadic and familial amyotrophic lateral sclerosis. <i>Acta Neuropathologica</i> , 2021, 142, 985-1001.	3.9	12
7	Phase 1â€² Trial of Antisense Oligonucleotide Tofersen for <i>SOD1</i> ALS. <i>New England Journal of Medicine</i> , 2020, 383, 109-119.	13.9	354
8	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. <i>Nature Neuroscience</i> , 2020, 23, 615-624.	7.1	157
9	Predicting disease specific spinal motor neurons and glia in sporadic ALS. <i>Neurobiology of Disease</i> , 2019, 130, 104523.	2.1	10
10	Pathogenic Mechanisms and Therapy Development for C9orf72 Amyotrophic Lateral Sclerosis/ Frontotemporal Dementia. <i>Neurotherapeutics</i> , 2019, 16, 1115-1132.	2.1	30
11	Antisense RNA foci are associated with nucleoli and TDP-43 mislocalization in C9orf72-ALS/FTD: a quantitative study. <i>Acta Neuropathologica</i> , 2019, 137, 527-530.	3.9	21
12	Human genetics and neuropathology suggest a link between miR-218 and amyotrophic lateral sclerosis pathophysiology. <i>Science Translational Medicine</i> , 2019, 11, .	5.8	37
13	TDP-43 extracted from frontotemporal lobar degeneration subject brains displays distinct aggregate assemblies and neurotoxic effects reflecting disease progression rates. <i>Nature Neuroscience</i> , 2019, 22, 65-77.	7.1	143
14	Premature polyadenylation-mediated loss of stathmin-2 is a hallmark of TDP-43-dependent neurodegeneration. <i>Nature Neuroscience</i> , 2019, 22, 180-190.	7.1	345
15	Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. <i>Neuron</i> , 2018, 97, 1268-1283.e6.	3.8	517
16	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. <i>Acta Neuropathologica</i> , 2018, 135, 459-474.	3.9	152
17	Kinnier Wilsonâ€™s puzzling features of amyotrophic lateral sclerosis. <i>Journal of Neurology, Neurosurgery and Psychiatry</i> , 2018, 89, 657-666.	0.9	4
18	Transcriptomeâ€™ pathology correlation identifies interplay between TDP-43 and the expression of its kinase CK1E in sporadic ALS. <i>Acta Neuropathologica</i> , 2018, 136, 405-423.	3.9	69

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19	Misfolded SOD1 is not a primary component of sporadic ALS. <i>Acta Neuropathologica</i> , 2017, 134, 97-111.	3.9	74
20	Pathological TDP-43 changes in Betz cells differ from those in bulbar and spinal \pm -motoneurons in sporadic amyotrophic lateral sclerosis. <i>Acta Neuropathologica</i> , 2017, 133, 79-90.	3.9	68
21	Modeling the neuroanatomic propagation of ALS in the spinal cord. AIP Conference Proceedings, 2017, , .	0.3	0
22	Modeling the neuroanatomic propagation of ALS in the spinal cord. AIP Conference Proceedings, 2017, , .	0.3	0
23	Distinct and shared functions of ALS-associated proteins TDP-43, FUS and TAF15 revealed by multisystem analyses. <i>Nature Communications</i> , 2016, 7, 12143.	5.8	137
24	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. <i>Neuron</i> , 2016, 90, 535-550.	3.8	437
25	RIPK1 mediates axonal degeneration by promoting inflammation and necroptosis in ALS. <i>Science</i> , 2016, 353, 603-608.	6.0	448
26	Dysregulated mi <i>RNA</i> biogenesis downstream of cellular stress and <i>ALS</i> causing mutations: a new mechanism for <i>ALS</i> . <i>EMBO Journal</i> , 2015, 34, 2633-2651.	3.5	176
27	Amyotrophic lateral sclerosis: Is the spinal fluid pathway involved in seeding and spread?. <i>Medical Hypotheses</i> , 2015, 85, 576-583.	0.8	22
28	Neuropathology of Amyotrophic Lateral Sclerosis and Its Variants. <i>Neurologic Clinics</i> , 2015, 33, 855-876.	0.8	199
29	Amyotrophic lateral sclerosis onset is influenced by the burden of rare variants in known amyotrophic lateral sclerosis genes. <i>Annals of Neurology</i> , 2015, 77, 100-113.	2.8	171
30	Focality, stochasticity and neuroanatomic propagation in ALS pathogenesis. <i>Experimental Neurology</i> , 2014, 262, 121-126.	2.0	81
31	Targeting RNA Foci in iPSC-Derived Motor Neurons from ALS Patients with a <i>C9ORF72</i> Repeat Expansion. <i>Science Translational Medicine</i> , 2013, 5, 208ra149.	5.8	586
32	Targeted degradation of sense and antisense <i>C9orf72</i> RNA foci as therapy for ALS and frontotemporal degeneration. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2013, 110, E4530-9.	3.3	508
33	Deciphering amyotrophic lateral sclerosis: What phenotype, neuropathology and genetics are telling us about pathogenesis. <i>Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration</i> , 2013, 14, 5-18.	1.1	142
34	Divergent roles of ALS-linked proteins FUS/TLS and TDP-43 intersect in processing long pre-mRNAs. <i>Nature Neuroscience</i> , 2012, 15, 1488-1497.	7.1	628
35	Sporadic ALS has compartment-specific aberrant exon splicing and altered cell matrix adhesion biology. <i>Human Molecular Genetics</i> , 2010, 19, 313-328.	1.4	114
36	ALS motor phenotype heterogeneity, focality, and spread. <i>Neurology</i> , 2009, 73, 805-811.	1.5	507

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37	Current and Future Directions in Genomics of Amyotrophic Lateral Sclerosis. Physical Medicine and Rehabilitation Clinics of North America, 2008, 19, 461-477.	0.7	4
38	Implications of ALS focality: Rostral-caudal distribution of lower motor neuron loss postmortem. Neurology, 2007, 68, 1576-1582.	1.5	142
39	Focality of upper and lower motor neuron degeneration at the clinical onset of ALS. Neurology, 2007, 68, 1571-1575.	1.5	270
40	Pathological TDP-43 distinguishes sporadic amyotrophic lateral sclerosis from amyotrophic lateral sclerosis with SOD1 mutations. Annals of Neurology, 2007, 61, 427-434.	2.8	840
41	Sporadic amyotrophic lateral sclerosis: A hypothesis of persistent (non-cytolytic) enteroviral infection. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders, 2005, 6, 77-87.	2.3	26
42	AAEM minimonograph #48: Autonomic nervous system testing. , 1997, 20, 919-937.		157