

Bryan P Traynor

List of Publications by Year in descending order

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Version: 2024-02-01

43
papers

11,927
citations

304743

22
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276875

41
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docs citations

44
times ranked

13315
citing authors

#	ARTICLE	IF	CITATIONS
1	A Hexanucleotide Repeat Expansion in C9ORF72 Is the Cause of Chromosome 9p21-Linked ALS-FTD. <i>Neuron</i> , 2011, 72, 257-268.	8.1	3,833
2	State of play in amyotrophic lateral sclerosis genetics. <i>Nature Neuroscience</i> , 2014, 17, 17-23.	14.8	1,300
3	Mutations in prion-like domains in hnRNPA2B1 and hnRNPA1 cause multisystem proteinopathy and ALS. <i>Nature</i> , 2013, 495, 467-473.	27.8	1,249
4	Exome Sequencing Reveals VCP Mutations as a Cause of Familial ALS. <i>Neuron</i> , 2010, 68, 857-864.	8.1	1,100
5	Frequency of the C9orf72 hexanucleotide repeat expansion in patients with amyotrophic lateral sclerosis and frontotemporal dementia: a cross-sectional study. <i>Lancet Neurology</i> , The, 2012, 11, 323-330.	10.2	1,039
6	Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. <i>Neuron</i> , 2018, 97, 1268-1283.e6.	8.1	517
7	Novel genes associated with amyotrophic lateral sclerosis: diagnostic and clinical implications. <i>Lancet Neurology</i> , The, 2018, 17, 94-102.	10.2	432
8	Mutations in the Matrin 3 gene cause familial amyotrophic lateral sclerosis. <i>Nature Neuroscience</i> , 2014, 17, 664-666.	14.8	398
9	Clinico-pathological features in amyotrophic lateral sclerosis with expansions in C9ORF72. <i>Brain</i> , 2012, 135, 751-764.	7.6	293
10	Ethnic variation in the incidence of ALS. <i>Neurology</i> , 2007, 68, 1002-1007.	1.1	236
11	Chromosome 9p21 in amyotrophic lateral sclerosis in Finland: a genome-wide association study. <i>Lancet Neurology</i> , The, 2010, 9, 978-985.	10.2	236
12	Common and rare variant association analyses in amyotrophic lateral sclerosis identify 15 risk loci with distinct genetic architectures and neuron-specific biology. <i>Nature Genetics</i> , 2021, 53, 1636-1648.	21.4	223
13	Clinical characteristics of patients with familial amyotrophic lateral sclerosis carrying the pathogenic GGGGCC hexanucleotide repeat expansion of C9ORF72. <i>Brain</i> , 2012, 135, 784-793.	7.6	182
14	A Genome-Wide Association Study of Myasthenia Gravis. <i>JAMA Neurology</i> , 2015, 72, 396.	9.0	139
15	Genetic causes of amyotrophic lateral sclerosis: New genetic analysis methodologies entailing new opportunities and challenges. <i>Brain Research</i> , 2015, 1607, 75-93.	2.2	132
16	Age-related penetrance of the C9orf72 repeat expansion. <i>Scientific Reports</i> , 2017, 7, 2116.	3.3	102
17	Genetic architecture of ALS in Sardinia. <i>Neurobiology of Aging</i> , 2014, 35, 2882.e7-2882.e12.	3.1	60
18	Pathogenic Huntingtin Repeat Expansions in Patients with Frontotemporal Dementia and Amyotrophic Lateral Sclerosis. <i>Neuron</i> , 2021, 109, 448-460.e4.	8.1	56

#	ARTICLE	IF	CITATIONS
19	ALS-associated KIF5A mutations abolish autoinhibition resulting in a toxic gain of function. <i>Cell Reports</i> , 2022, 39, 110598.	6.4	47
20	TBK1 is associated with ALS and ALS-FTD in Sardinian patients. <i>Neurobiology of Aging</i> , 2016, 43, 180.e1-180.e5.	3.1	40
21	<i>OPTN</i> 691_692insAG is a founder mutation causing recessive ALS and increased risk in heterozygotes. <i>Neurology</i> , 2016, 86, 446-453.	1.1	37
22	Association of a Novel <i>ACTA1</i> Mutation With a Dominant Progressive Scapulo-peroneal Myopathy in an Extended Family. <i>JAMA Neurology</i> , 2015, 72, 689.	9.0	35
23	Unraveling the complex interplay between genes, environment, and climate in ALS. <i>EBioMedicine</i> , 2022, 75, 103795.	6.1	32
24	Mutational Analysis of Known ALS Genes in an Italian Population-Based Cohort. <i>Neurology</i> , 2021, 96, e600-e609.	1.1	23
25	<i>ATXN2</i> is a modifier of phenotype in ALS patients of Sardinian ancestry. <i>Neurobiology of Aging</i> , 2015, 36, 2906.e1-2906.e5.	3.1	19
26	Identification of a pathogenic intronic KIF5A mutation in an ALS-FTD kindred. <i>Neurology</i> , 2020, 95, 1015-1018.	1.1	19
27	Identifying and predicting amyotrophic lateral sclerosis clinical subgroups: a population-based machine-learning study. <i>The Lancet Digital Health</i> , 2022, 4, e359-e369.	12.3	19
28	Alzheimer risk loci and associated neuropathology in a population-based study (Vantaa 85+). <i>Neurology: Genetics</i> , 2018, 4, e211.	1.9	16
29	<i>C9orf72</i> hexanucleotide repeat length in older population: normal variation and effects on cognition. <i>Neurobiology of Aging</i> , 2019, 84, 242.e7-242.e12.	3.1	16
30	The NGS technology for the identification of genes associated with the ALS. A systematic review. <i>European Journal of Clinical Investigation</i> , 2020, 50, e13228.	3.4	16
31	Use of Genetic Testing in Amyotrophic Lateral Sclerosis by Neurologists. <i>JAMA Neurology</i> , 2017, 74, 125.	9.0	15
32	Oligogenic basis of sporadic ALS. <i>Neurology: Genetics</i> , 2019, 5, e335.	1.9	15
33	High frequency of <i>C9orf72</i> hexanucleotide repeat expansion in amyotrophic lateral sclerosis patients from two founder populations sharing the same risk haplotype. <i>Neurobiology of Aging</i> , 2018, 64, 160.e1-160.e7.	3.1	11
34	ALS in Finland. <i>Neurology: Genetics</i> , 2022, 8, e665.	1.9	11
35	Exploring the Epigenetics of Alzheimer Disease. <i>JAMA Neurology</i> , 2015, 72, 8.	9.0	8
36	Investigating <i>RFC1</i> expansions in sporadic amyotrophic lateral sclerosis. <i>Journal of the Neurological Sciences</i> , 2021, 430, 118061.	0.6	8

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37	Dementia Researchâ€™A Roadmap for the Next Decade. JAMA Neurology, 2017, 74, 141.	9.0	3
38	Highlighting the clinical potential of HTT repeat expansions in Frontotemporal Dementia and Amyotrophic Lateral Sclerosis. Neuron, 2021, 109, 1947-1948.	8.1	3
39	C9orf72 hexanucleotide repeat expansions are not a common cause of obsessive-compulsive disorder. Journal of the Neurological Sciences, 2017, 375, 71-72.	0.6	2
40	High BMI is associated with low ALS risk. Neurology, 2019, 93, 189-191.	1.1	2
41	Special Issue on amyotrophic lateral sclerosis. Experimental Neurology, 2014, 262, 73-74.	4.1	1
42	Genetic counselling: Psychological impact and concerns. , 2018, , .		1
43	To Dement or Not to Dement, That Is the Question. JAMA Neurology, 2016, 73, 383.	9.0	0