

Jacqueline S De Bellerocche

List of Publications by Citations

Source: <https://exaly.com/author-pdf/2725178/jacqueline-s-de-bellerocche-publications-by-citations.pdf>

Version: 2024-04-24

This document has been generated based on the publications and citations recorded by exaly.com. For the latest version of this publication list, visit the link given above.

The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

33
papers

6,041
citations

22
h-index

33
g-index

33
ext. papers

7,081
ext. citations

9.5
avg, IF

4.74
L-index

#	Paper	IF	Citations
33	Mutations in FUS, an RNA processing protein, cause familial amyotrophic lateral sclerosis type 6. <i>Science</i> , 2009 , 323, 1208-1211	33.3	1890
32	TDP-43 mutations in familial and sporadic amyotrophic lateral sclerosis. <i>Science</i> , 2008 , 319, 1668-72	33.3	1877
31	Endoplasmic reticulum stress signalling - from basic mechanisms to clinical applications. <i>FEBS Journal</i> , 2019 , 286, 241-278	5.7	309
30	Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. <i>Neuron</i> , 2018 , 97, 1268-1283.e6	13.9	296
29	Exome-wide rare variant analysis identifies TUBA4A mutations associated with familial ALS. <i>Neuron</i> , 2014 , 84, 324-31	13.9	229
28	Familial amyotrophic lateral sclerosis is associated with a mutation in D-amino acid oxidase. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2010 , 107, 7556-61	11.5	191
27	The C9ORF72 expansion mutation is a common cause of ALS+/-FTD in Europe and has a single founder. <i>European Journal of Human Genetics</i> , 2013 , 21, 102-8	5.3	159
26	NEK1 variants confer susceptibility to amyotrophic lateral sclerosis. <i>Nature Genetics</i> , 2016 , 48, 1037-42	36.3	149
25	CCNF mutations in amyotrophic lateral sclerosis and frontotemporal dementia. <i>Nature Communications</i> , 2016 , 7, 11253	17.4	126
24	Vesicle associated membrane protein B (VAPB) is decreased in ALS spinal cord. <i>Neurobiology of Aging</i> , 2010 , 31, 969-85	5.6	99
23	Protective effects of heat shock protein 27 in a model of ALS occur in the early stages of disease progression. <i>Neurobiology of Disease</i> , 2008 , 30, 42-55	7.5	89
22	Reply to Millecamps et al.: Elucidating the role of D amino acid oxidase in familial amyotrophic lateral sclerosis. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2010 , 107, E108-E108	11.5	78
21	Mutations in the vesicular trafficking protein annexin A11 are associated with amyotrophic lateral sclerosis. <i>Science Translational Medicine</i> , 2017 , 9,	17.5	74
20	Transcription and pathway analysis of the superior temporal cortex and anterior prefrontal cortex in schizophrenia. <i>Journal of Neuroscience Research</i> , 2011 , 89, 1218-27	4.4	55
19	ALS-associated missense and nonsense TBK1 mutations can both cause loss of kinase function. <i>Neurobiology of Aging</i> , 2018 , 71, 266.e1-266.e10	5.6	44
18	Sequestosome-1 (SQSTM1) sequence variants in ALS cases in the UK: prevalence and coexistence of SQSTM1 mutations in ALS kindred with PDB. <i>European Journal of Human Genetics</i> , 2014 , 22, 492-6	5.3	41
17	Elevated levels of amino acids in the CSF of motor neuron disease patients. <i>Neurochemical Pathology</i> , 1984 , 2, 1-6		39

16	Amyotrophic lateral sclerosis (ALS) and Alzheimer's disease (AD) are characterised by differential activation of ER stress pathways: focus on UPR target genes. <i>Cell Stress and Chaperones</i> , 2018 , 23, 897-912	4.2	38
15	The role of D-serine and glycine as co-agonists of NMDA receptors in motor neuron degeneration and amyotrophic lateral sclerosis (ALS). <i>Frontiers in Synaptic Neuroscience</i> , 2014 , 6, 10	3.5	38
14	The role of D-amino acids in amyotrophic lateral sclerosis pathogenesis: a review. <i>Amino Acids</i> , 2012 , 43, 1823-31	3.5	38
13	Identification of genetic heterogeneity in Refsum's disease. <i>European Journal of Human Genetics</i> , 2000 , 8, 649-51	5.3	33
12	Pathogenic effects of amyotrophic lateral sclerosis-linked mutation in D-amino acid oxidase are mediated by D-serine. <i>Neurobiology of Aging</i> , 2014 , 35, 876-85	5.6	26
11	The CHCHD10 P34S variant is not associated with ALS in a UK cohort of familial and sporadic patients. <i>Neurobiology of Aging</i> , 2015 , 36, 2908.e17-8	5.6	19
10	Focus on the Role of D-serine and D-amino Acid Oxidase in Amyotrophic Lateral Sclerosis/Motor Neuron Disease (ALS). <i>Frontiers in Molecular Biosciences</i> , 2018 , 5, 8	5.6	17
9	Thioredoxin reductase 1 haplotypes modify familial amyotrophic lateral sclerosis onset. <i>Free Radical Biology and Medicine</i> , 2009 , 46, 202-11	7.8	17
8	Allelic variants in the zinc transporter-3 gene, SLC30A3, a candidate gene identified from gene expression studies, show gender-specific association with schizophrenia. <i>European Psychiatry</i> , 2014 , 29, 172-8	6	15
7	Common variants in the chromosome 2p23 region containing the SLC30A3 (ZnT3) gene are associated with schizophrenia in female but not male individuals in a large collection of European samples. <i>Psychiatry Research</i> , 2016 , 246, 335-340	9.9	14
6	Experimental approaches for elucidating co-agonist regulation of NMDA receptor in motor neurons: Therapeutic implications for amyotrophic lateral sclerosis (ALS). <i>Journal of Pharmaceutical and Biomedical Analysis</i> , 2015 , 116, 2-6	3.5	10
5	Hypothalamic Hypertensive Factor. <i>Hypertension</i> , 1997 , 30, 1493-1498	8.5	10
4	VCP mutations are not a major cause of familial amyotrophic lateral sclerosis in the UK. <i>Journal of the Neurological Sciences</i> , 2015 , 349, 209-13	3.2	9
3	Tissue-selective regulation of protein homeostasis and unfolded protein response signalling in sporadic ALS. <i>Journal of Cellular and Molecular Medicine</i> , 2020 , 24, 6055-6069	5.6	6
2	Characterisation of the pathogenic effects of the in vivo expression of an ALS-linked mutation in D-amino acid oxidase: Phenotype and loss of spinal cord motor neurons. <i>PLoS ONE</i> , 2017 , 12, e0188912	3.7	6
1	FC09.06 Cholecystokinin CCKb Receptor mRNA Isoforms: Expression in Postmortem Monkey and Human Brain [Alterations Following Schizophrenia. <i>European Psychiatry</i> , 2000 , 15, 284s-285s	6	