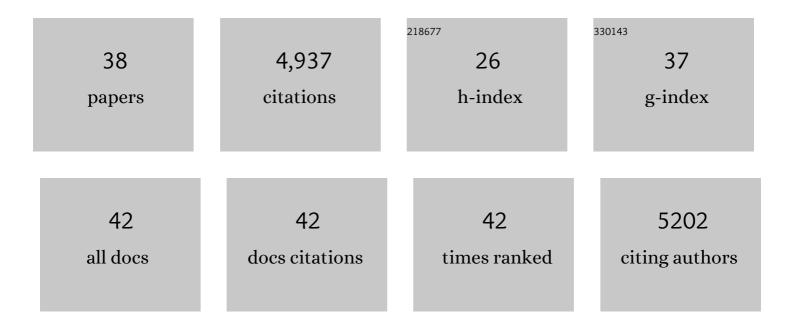
Karen R Jansen-West

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/2986206/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Unconventional Translation of C9ORF72 GGGGCC Expansion Generates Insoluble Polypeptides Specific to c9FTD/ALS. Neuron, 2013, 77, 639-646.	8.1	962
2	Antisense transcripts of the expanded C9ORF72 hexanucleotide repeat form nuclear RNA foci and undergo repeat-associated non-ATG translation in c9FTD/ALS. Acta Neuropathologica, 2013, 126, 829-844.	7.7	506
3	<i>C9ORF72</i> repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. Science, 2015, 348, 1151-1154.	12.6	332
4	Discovery of a Biomarker and Lead Small Molecules to Target r(GGGGCC)-Associated Defects in c9FTD/ALS. Neuron, 2014, 83, 1043-1050.	8.1	289
5	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	14.8	268
6	Interaction of tau with the RNA-Binding Protein TIA1 Regulates tau Pathophysiology and Toxicity. Cell Reports, 2016, 15, 1455-1466.	6.4	260
7	Poly(GR) impairs protein translation and stress granule dynamics in C9orf72-associated frontotemporal dementia and amyotrophic lateral sclerosis. Nature Medicine, 2018, 24, 1136-1142.	30.7	241
8	TDP-43 represses cryptic exon inclusion in the FTD–ALS gene UNC13A. Nature, 2022, 603, 124-130.	27.8	193
9	Heterochromatin anomalies and double-stranded RNA accumulation underlie <i>C9orf72</i> poly(PR) toxicity. Science, 2019, 363, .	12.6	181
10	Poly(GP) proteins are a useful pharmacodynamic marker for <i>C9ORF72</i> -associated amyotrophic lateral sclerosis. Science Translational Medicine, 2017, 9, .	12.4	179
11	CUG initiation and frameshifting enable production of dipeptide repeat proteins from ALS/FTD C9ORF72 transcripts. Nature Communications, 2018, 9, 152.	12.8	123
12	Systematic analysis of dark and camouflaged genes reveals disease-relevant genes hiding in plain sight. Genome Biology, 2019, 20, 97.	8.8	122
13	Truncated stathmin-2 is a marker of TDP-43 pathology in frontotemporal dementia. Journal of Clinical Investigation, 2020, 130, 6080-6092.	8.2	117
14	Spt4 selectively regulates the expression of <i>C9orf72</i> sense and antisense mutant transcripts. Science, 2016, 353, 708-712.	12.6	116
15	<i>C9orf72</i> poly(GR) aggregation induces TDP-43 proteinopathy. Science Translational Medicine, 2020, 12, .	12.4	115
16	Long-read sequencing across the C9orf72 â€~GGGGCC' repeat expansion: implications for clinical use and genetic discovery efforts in human disease. Molecular Neurodegeneration, 2018, 13, 46.	10.8	111
17	Aberrant deposition of stress granule-resident proteins linked to C9orf72-associated TDP-43 proteinopathy. Molecular Neurodegeneration, 2019, 14, 9.	10.8	111
18	Repetitive element transcripts are elevated in the brain of C9orf72 ALS/FTLD patients. Human Molecular Genetics, 2017, 26, 3421-3431.	2.9	101

#	Article	IF	CITATIONS
19	Misregulation of human sortilin splicing leads to the generation of a nonfunctional progranulin receptor. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 21510-21515.	7.1	82
20	TDP-43 functions within a network of hnRNP proteins to inhibit the production of a truncated human SORT1 receptor. Human Molecular Genetics, 2016, 25, 534-545.	2.9	70
21	Serum neurofilament light protein correlates with unfavorable clinical outcomes in hospitalized patients with COVID-19. Science Translational Medicine, 2021, 13, .	12.4	67
22	Plasma neurofilament light predicts mortality in patients with stroke. Science Translational Medicine, 2020, 12, .	12.4	51
23	Chimeric Peptide Species Contribute to Divergent Dipeptide Repeat Pathology in c9ALS/FTD and SCA36. Neuron, 2020, 107, 292-305.e6.	8.1	51
24	Conserved DNA methylation combined with differential frontal cortex and cerebellar expression distinguishes C9orf72-associated and sporadic ALS, and implicates SERPINA1 in disease. Acta Neuropathologica, 2017, 134, 715-728.	7.7	40
25	Hexanucleotide Repeat Expansions in c9FTD/ALS and SCA36 Confer Selective Patterns of Neurodegeneration InÂVivo. Cell Reports, 2020, 31, 107616.	6.4	37
26	Toward allele-specific targeting therapy and pharmacodynamic marker for spinocerebellar ataxia type 3. Science Translational Medicine, 2020, 12, .	12.4	32
27	Tau exhibits unique seeding properties in globular glial tauopathy. Acta Neuropathologica Communications, 2019, 7, 36.	5.2	28
28	Astrocyte-derived clusterin suppresses amyloid formation in vivo. Molecular Neurodegeneration, 2020, 15, 71.	10.8	26
29	Tau and neurofilament lightâ€chain as fluid biomarkers in spinocerebellar ataxia type 3. European Journal of Neurology, 2022, 29, 2439-2452.	3.3	25
30	Clusterin ameliorates tau pathology in vivo by inhibiting fibril formation. Acta Neuropathologica Communications, 2020, 8, 210.	5.2	24
31	Premature termination codon readthrough upregulates progranulin expression and improves lysosomal function in preclinical models of GRN deficiency. Molecular Neurodegeneration, 2020, 15, 21.	10.8	19
32	Abnormal expression of homeobox genes and transthyretin in <i>C9ORF72</i> expansion carriers. Neurology: Genetics, 2017, 3, e161.	1.9	12
33	Urine levels of the polyglutamine ataxin-3 protein are elevated in patients with spinocerebellar ataxia type 3. Parkinsonism and Related Disorders, 2021, 89, 151-154.	2.2	9
34	TRIO gene segregation in a family with cerebellar ataxia. Neurologia I Neurochirurgia Polska, 2018, 52, 743-749.	1.2	5
35	Plasma PolyQ-ATXN3 Levels Associate With Cerebellar Degeneration and Behavioral Abnormalities in a New AAV-Based SCA3 Mouse Model. Frontiers in Cell and Developmental Biology, 2022, 10, 863089.	3.7	5
36	HDAC6 Interacts With Poly (GA) and Modulates its Accumulation in c9FTD/ALS. Frontiers in Cell and Developmental Biology, 2021, 9, 809942.	3.7	4

#	Article	IF	CITATIONS
37	Aß40 displays amyloidogenic properties in the non-transgenic mouse brain but does not exacerbate Aß42 toxicity in Drosophila. Alzheimer's Research and Therapy, 2020, 12, 132.	6.2	3
38	Comment on: <scp>Polyglutamineâ€Expanded</scp> Ataxinâ€3: A Target Engagement Marker for Spinocerebellar Ataxia Type 3 in Peripheral Blood. Movement Disorders, 2022, 37, 1120-1121.	3.9	0