

Yong-jie Zhang

List of Publications by Year in descending order

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Version: 2024-02-01

32
papers

5,317
citations

201674

27
h-index

414414

32
g-index

35
all docs

35
docs citations

35
times ranked

4834
citing authors

#	ARTICLE	IF	CITATIONS
1	p53 is a central regulator driving neurodegeneration caused by C9orf72 poly(PR). <i>Cell</i> , 2021, 184, 689-708.e20.	28.9	104
2	The AD tau core spontaneously self-assembles and recruits full-length tau to filaments. <i>Cell Reports</i> , 2021, 34, 108843.	6.4	30
3	Microglial lysosome dysfunction contributes to white matter pathology and TDP-43 proteinopathy in GRN-associated FTD. <i>Cell Reports</i> , 2021, 36, 109581.	6.4	33
4	Ribonuclease recruitment using a small molecule reduced c9ALS/FTD r(G ₄ C ₂) Tj ETQq0,00 rgBT /Overlock 1	12.4	39
5	A Small Molecule Exploits Hidden Structural Features within the RNA Repeat Expansion That Causes c9ALS/FTD and Rescues Pathological Hallmarks. <i>ACS Chemical Neuroscience</i> , 2021, 12, 4076-4089.	3.5	8
6	Structural Features of Small Molecules Targeting the RNA Repeat Expansion That Causes Genetically Defined ALS/FTD. <i>ACS Chemical Biology</i> , 2020, 15, 3112-3123.	3.4	12
7	<i>C9orf72</i> poly(GR) aggregation induces TDP-43 proteinopathy. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	115
8	Hexanucleotide Repeat Expansions in c9FTD/ALS and SCA36 Confer Selective Patterns of Neurodegeneration In Vivo. <i>Cell Reports</i> , 2020, 31, 107616.	6.4	37
9	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. <i>Nature Neuroscience</i> , 2020, 23, 615-624.	14.8	157
10	C-terminal and full length TDP-43 specie differ according to FTLT-DTP lesion type but not genetic mutation. <i>Acta Neuropathologica Communications</i> , 2019, 7, 100.	5.2	11
11	Aberrant deposition of stress granule-resident proteins linked to C9orf72-associated TDP-43 proteinopathy. <i>Molecular Neurodegeneration</i> , 2019, 14, 9.	10.8	111
12	Heterochromatin anomalies and double-stranded RNA accumulation underlie <i>C9orf72</i> poly(PR) toxicity. <i>Science</i> , 2019, 363, .	12.6	181
13	The Hairpin Form of r(G4C2) _{exp} in c9ALS/FTD Is Repeat-Associated Non-ATG Translated and a Target for Bioactive Small Molecules. <i>Cell Chemical Biology</i> , 2019, 26, 179-190.e12.	5.2	80
14	TDP-43 pathology disrupts nuclear pore complexes and nucleocytoplasmic transport in ALS/FTD. <i>Nature Neuroscience</i> , 2018, 21, 228-239.	14.8	404
15	Poly(GR) impairs protein translation and stress granule dynamics in C9orf72-associated frontotemporal dementia and amyotrophic lateral sclerosis. <i>Nature Medicine</i> , 2018, 24, 1136-1142.	30.7	241
16	Poly-GR dipeptide repeat polymers correlate with neurodegeneration and Clinicopathological subtypes in C9ORF72-related brain disease. <i>Acta Neuropathologica Communications</i> , 2018, 6, 63.	5.2	79
17	Poly(GP) proteins are a useful pharmacodynamic marker for <i>C9ORF72</i> -associated amyotrophic lateral sclerosis. <i>Science Translational Medicine</i> , 2017, 9, .	12.4	179
18	Repetitive element transcripts are elevated in the brain of C9orf72 ALS/FTLD patients. <i>Human Molecular Genetics</i> , 2017, 26, 3421-3431.	2.9	101

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19	Spt4 selectively regulates the expression of <i>C9orf72</i> sense and antisense mutant transcripts. <i>Science</i> , 2016, 353, 708-712.	12.6	116
20	The extreme N-terminus of TDP-43 mediates the cytoplasmic aggregation of TDP-43 and associated toxicity in vivo. <i>Brain Research</i> , 2016, 1647, 57-64.	2.2	44
21	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. <i>Nature Neuroscience</i> , 2016, 19, 668-677.	14.8	268
22	<i>C9ORF72</i> repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. <i>Science</i> , 2015, 348, 1151-1154.	12.6	332
23	Human C9ORF72 Hexanucleotide Expansion Reproduces RNA Foci and Dipeptide Repeat Proteins but Not Neurodegeneration in BAC Transgenic Mice. <i>Neuron</i> , 2015, 88, 902-909.	8.1	219
24	Aggregation-prone c9FTD/ALS poly(GA) RAN-translated proteins cause neurotoxicity by inducing ER stress. <i>Acta Neuropathologica</i> , 2014, 128, 505-524.	7.7	284
25	Mechanisms of toxicity in C9FTLD/ALS. <i>Acta Neuropathologica</i> , 2014, 127, 359-376.	7.7	134
26	Discovery of a Biomarker and Lead Small Molecules to Target r(GGGGCC)-Associated Defects in c9FTD/ALS. <i>Neuron</i> , 2014, 83, 1043-1050.	8.1	289
27	Antisense transcripts of the expanded C9ORF72 hexanucleotide repeat form nuclear RNA foci and undergo repeat-associated non-ATG translation in c9FTD/ALS. <i>Acta Neuropathologica</i> , 2013, 126, 829-844.	7.7	506
28	The dual functions of the extreme N-terminus of TDP-43 in regulating its biological activity and inclusion formation. <i>Human Molecular Genetics</i> , 2013, 22, 3112-3122.	2.9	156
29	Misregulation of human sortilin splicing leads to the generation of a nonfunctional progranulin receptor. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2012, 109, 21510-21515.	7.1	82
30	Phosphorylation regulates proteasomal-mediated degradation and solubility of TAR DNA binding protein-43 C-terminal fragments. <i>Molecular Neurodegeneration</i> , 2010, 5, 33.	10.8	103
31	Aberrant cleavage of TDP-43 enhances aggregation and cellular toxicity. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2009, 106, 7607-7612.	7.1	523
32	Progranulin Mediates Caspase-Dependent Cleavage of TAR DNA Binding Protein-43. <i>Journal of Neuroscience</i> , 2007, 27, 10530-10534.	3.6	339