C9ORF72 repeat expansions in mice cause TDP-4 behavioral deficits

Science

348, 1151-1154

DOI: 10.1126/science.aaa9344

Citation Report

#	Article	IF	Citations
1	<scp>C</scp>9orf72 ablation in mice does not cause motor neuron degeneration or motor deficits. Annals of Neurology, 2015, 78, 426-438.	2.8	225
2	ALS Patient Stem Cells for Unveiling Disease Signatures of Motoneuron Susceptibility: Perspectives on the Deadly Mitochondria, ER Stress and Calcium Triad. Frontiers in Cellular Neuroscience, 2015, 9, 448.	1.8	33
3	Expanding neurodegeneration modelling. Nature Reviews Neuroscience, 2015, 16, 376-376.	4.9	3
4	Tau deposition drives neuropathological, inflammatory and behavioral abnormalities independently of neuronal loss in a novel mouse model. Human Molecular Genetics, 2015, 24, 6198-6212.	1.4	52
5	Distribution of dipeptide repeat proteins in cellular models and C9orf72 mutation cases suggests link to transcriptional silencing. Acta Neuropathologica, 2015, 130, 537-555.	3.9	157
6	Dysfunction of autophagy as the pathological mechanism of motor neuron disease based on a patient-specific disease model. Neuroscience Bulletin, 2015, 31, 445-451.	1.5	9
7	Amyotrophic lateral sclerosis: Current perspectives from basic research to the clinic. Progress in Neurobiology, 2015, 133, 1-26.	2.8	99
9	Differential Toxicity of Nuclear RNA Foci versus Dipeptide Repeat Proteins in a Drosophila Model of C9ORF72 FTD/ALS. Neuron, 2015, 87, 1207-1214.	3.8	176
10	Novel clinical associations with specific C9ORF72 transcripts in patients with repeat expansions in C9ORF72. Acta Neuropathologica, 2015, 130, 863-876.	3.9	104
11	A genetic association study of two genes linked to neurodegeneration in a Sardinian multiple sclerosis population: The TARDBP Ala382Thr mutation and C9orf72 expansion. Journal of the Neurological Sciences, 2015, 357, 229-234.	0.3	6
12	Problems at the nuclear pore. Nature, 2015, 525, 36-37.	13.7	13
13	Modifiers of C9orf72 dipeptide repeat toxicity connect nucleocytoplasmic transport defects to FTD/ALS. Nature Neuroscience, 2015, 18, 1226-1229.	7.1	528
14	A retrospective review of the progress in amyotrophic lateral sclerosis drug discovery over the last decade and a look at the latest strategies. Expert Opinion on Drug Discovery, 2015, 10, 1099-1118.	2.5	26
15	Dipeptide repeat protein inclusions are rare in the spinal cord and almost absent from motor neurons in C9ORF72 mutant amyotrophic lateral sclerosis and are unlikely to cause their degeneration. Acta Neuropathologica Communications, 2015, 3, 38.	2.4	80
16	C9orf72 BAC Transgenic Mice Display Typical Pathologic Features of ALS/FTD. Neuron, 2015, 88, 892-901.	3.8	249
17	Human C9ORF72 Hexanucleotide Expansion Reproduces RNA Foci and Dipeptide Repeat Proteins but Not Neurodegeneration in BAC Transgenic Mice. Neuron, 2015, 88, 902-909.	3.8	219
18	Epidemiology and molecular mechanism of frontotemporal lobar degeneration/amyotrophic lateral sclerosis with repeat expansion mutation inC9orf72. Journal of Neurogenetics, 2015, 29, 85-94.	0.6	17
19	The ALS/FTLD associated protein C9orf72 associates with SMCR8 and WDR41 to regulate the autophagy-lysosome pathway. Acta Neuropathologica Communications, 2016, 4, 51.	2.4	243

#	Article	IF	Citations
20	The genetics of amyotrophic lateral sclerosis: current insights. Degenerative Neurological and Neuromuscular Disease, 2016, 6, 49.	0.7	65
21	Selective Vulnerability of Neuronal Subtypes in ALS: A Fertile Ground for the Identification of Therapeutic Targets. , 0, , .		1
22	Molecular neuropathology of frontotemporal dementia: insights into disease mechanisms from postmortem studies. Journal of Neurochemistry, 2016, 138, 54-70.	2.1	252
23	Identification of plexin A4 as a novel clusterin receptor links two Alzheimerâ€™s disease risk genes. Human Molecular Genetics, 2016, 25, 3467-3475.	1.4	21
24	Old versus New Mechanisms in the Pathogenesis of ALS. Brain Pathology, 2016, 26, 276-286.	2.1	45
25	Mass spectrometric analysis of accumulated TDP-43 in amyotrophic lateral sclerosis brains. Scientific Reports, 2016, 6, 23281.	1.6	118
26	Bidirectional Transcriptional Inhibition as Therapy for ALS/FTD Caused by Repeat Expansion in C9orf72. Neuron, 2016, 92, 1160-1163.	3.8	18
27	Reduced hn <scp>RNPA</scp> 3 increases C9orf72 repeat <scp>RNA</scp> levels and dipeptideâ€repeat protein deposition. EMBO Reports, 2016, 17, 1314-1325.	2.0	39
28	Cytoplasmic poly-GA aggregates impair nuclear import of TDP-43 in C9orf72 ALS/FTLD. Human Molecular Genetics, 2017, 26, ddw432.	1.4	82
29	Amyotrophic lateral sclerosis: recent genetic highlights. Current Opinion in Neurology, 2016, 29, 557-564.	1.8	37
30	ALS: A bucket of genes, environment, metabolism and unknown ingredients. Progress in Neurobiology, 2016, 142, 104-129.	2.8	158
31	A system to study mechanisms of neuromuscular junction development and maintenance. Development (Cambridge), 2016, 143, 2464-77.	1.2	35
32	RAN translationâ€"What makes it run?. Brain Research, 2016, 1647, 30-42.	1.1	89
33	Evaluating Behavior in Mouse Models of the Behavioral Variant of Frontotemporal Dementia: Which Test for Which Symptom?. Neurodegenerative Diseases, 2016, 16, 127-139.	0.8	11
34	There has been an awakening: Emerging mechanisms of C9orf72 mutations in FTD/ALS. Brain Research, 2016, 1647, 19-29.	1.1	133
35	The expanding biology of the C9orf72 nucleotide repeat expansion in neurodegenerative disease. Nature Reviews Neuroscience, 2016, 17, 383-395.	4.9	173
36	C9ORF72 -ALS/FTD: Transgenic Mice Make a Come-BAC. Neuron, 2016, 90, 427-431.	3.8	16
37	Distinct neurological disorders with C9orf72 mutations: genetics, pathogenesis, and therapy. Neuroscience and Biobehavioral Reviews, 2016, 66, 127-142.	2.9	11

#	Article	IF	Citations
38	C9orf72 BAC Mouse Model with Motor Deficits and Neurodegenerative Features of ALS/FTD. Neuron, 2016, 90, 521-534.	3.8	294
39	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. Neuron, 2016, 90, 535-550.	3.8	437
40	Identification of a novel loss-of-function C9orf72 splice site mutation in a patient with amyotrophic lateral sclerosis. Neurobiology of Aging, 2016, 47, 219.e1-219.e5.	1.5	17
41	Poly(GR) in C9ORF72 -Related ALS/FTD Compromises Mitochondrial Function and Increases Oxidative Stress and DNA Damage in iPSC-Derived Motor Neurons. Neuron, 2016, 92, 383-391.	3.8	323
42	Spt4 selectively regulates the expression of C9orf72 sense and antisense mutant transcripts. Science, 2016, 353, 708-712.	6.0	116
43	Genetics of <scp>FTLD</scp>: overview and what else we can expect from genetic studies. Journal of Neurochemistry, 2016, 138, 32-53.	2.1	118
44	Insights into the pathogenic mechanisms of Chromosome 9 open reading frame 72 (C9orf72) repeat expansions. Journal of Neurochemistry, 2016, 138, 145-162.	2.1	59
45	Nuclear transport dysfunction: a common theme in amyotrophic lateral sclerosis and frontotemporal dementia. Journal of Neurochemistry, 2016, 138, 134-144.	2.1	45
46	Decoding ALS: from genes to mechanism. Nature, 2016, 539, 197-206.	13.7	1,533
47	From animal models to human disease: a genetic approach for personalized medicine in ALS. Acta Neuropathologica Communications, 2016, 4, 70.	2.4	115
48	Î±<scp>CAR IGF</scp>â€1 vector targeting of motor neurons ameliorates disease progression in <scp>ALS</scp> mice. Annals of Clinical and Translational Neurology, 2016, 3, 752-768.	1.7	8
49	Modelling C9orf72 dipeptide repeat proteins of a physiologically relevant size. Human Molecular Genetics, 2016, 25, ddw327.	1.4	25
50	Monitoring peripheral nerve degeneration in ALS by label-free stimulated Raman scattering imaging. Nature Communications, 2016, 7, 13283.	5.8	82
51	Hypo- and Hyper-Assembly Diseases of RNAâ€“Protein Complexes. Trends in Molecular Medicine, 2016, 22, 615-628.	3.5	59
52	Structural insights into the multi-determinant aggregation of TDP-43 in motor neuron-like cells. Neurobiology of Disease, 2016, 94, 63-72.	2.1	29
53	Activation of HIPK2 Promotes ER Stress-Mediated Neurodegeneration in Amyotrophic Lateral Sclerosis. Neuron, 2016, 91, 41-55.	3.8	75
54	Loss of C9 <scp>ORF</scp> 72 impairs autophagy and synergizes with polyQ Ataxinâ€2 to induce motor neuron dysfunction and cell death. EMBO Journal, 2016, 35, 1276-1297.	3.5	343
55	Amyotrophic lateral sclerosis and frontotemporal dementia: distinct and overlapping changes in eating behaviour and metabolism. Lancet Neurology, The, 2016, 15, 332-342.	4.9	120

#	Article	IF	Citations
56	Unstable repeat expansions in neurodegenerative diseases: nucleocytoplasmic transport emerges on the scene. Neurobiology of Aging, 2016, 39, 174-183.	1.5	32
57	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. Nature Neuroscience, 2016, 19, 668-677.	7.1	268
58	Advances in the Development of Disease-Modifying Treatments for Amyotrophic Lateral Sclerosis. CNS Drugs, 2016, 30, 227-243.	2.7	36
59	Atypical parkinsonism in C9orf72 expansions: a case report and systematic review of 45 cases from the literature. Journal of Neurology, 2016, 263, 558-574.	1.8	40
60	An amyloid-like cascade hypothesis for C9orf72 ALS/FTD. Current Opinion in Neurobiology, 2016, 36, 99-106.	2.0	59
61	The C9orf72 repeat size correlates with onset age of disease, DNA methylation and transcriptional downregulation of the promoter. Molecular Psychiatry, 2016, 21, 1112-1124.	4.1	201
62	Oxidative stress and mitochondrial damage in the pathogenesis of ALS: New perspectives. Neuroscience Letters, 2017, 636, 3-8.	1.0	92
63	Insulin-like growth factor 1 in diabetic neuropathy and amyotrophic lateral sclerosis. Neurobiology of Disease, 2017, 97, 103-113.	2.1	39
64	Frontotemporal lobar degeneration: Pathogenesis, pathology and pathways to phenotype. Brain Pathology, 2017, 27, 723-736.	2.1	112
65	ALS/FTLD: experimental models and reality. Acta Neuropathologica, 2017, 133, 177-196.	3.9	78
66	Mouse models of frontotemporal dementia: A comparison of phenotypes with clinical symptomatology. Neuroscience and Biobehavioral Reviews, 2017, 74, 126-138.	2.9	23
67	Glycine-alanine dipeptide repeat protein contributes to toxicity in a zebrafish model of C9orf72 associated neurodegeneration. Molecular Neurodegeneration, 2017, 12, 6.	4.4	57
68	Loss of Ranbp2 in motor neurons causes the disruption of nucleocytoplasmic and chemokine signaling and proteostasis of hnRNPH3 and Mmp28, and the development of amyotrophic lateral sclerosis (ALS)-like syndromes. DMM Disease Models and Mechanisms, 2017, 10, 559-579.	1.2	34
69	Frontotemporal dementia. , 2017, , 199-249.		1
70	Spinal poly-GA inclusions in a C9orf72 mouse model trigger motor deficits and inflammation without neuron loss. Acta Neuropathologica, 2017, 134, 241-254.	3.9	99
71	Polyâ€<scp>GP</scp> in cerebrospinal fluid links C9orf72â€associated dipeptide repeat expression to the asymptomatic phase of <scp>ALS</scp>/<scp>FTD</scp>. EMBO Molecular Medicine, 2017, 9, 859-868.	3.3	90
72	Abnormal Function of Metalloproteins Underlies Most Neurodegenerative Diseases. , 2017, , 415-438.		2
73	In-depth clinico-pathological examination of RNA foci in a large cohort of C9ORF72 expansion carriers. Acta Neuropathologica, 2017, 134, 255-269.	3.9	76

#	Article	IF	Citations
74	New routes in frontotemporal dementia drug discovery. Expert Opinion on Drug Discovery, 2017, 12, 659-671.	2.5	8
75	Modeling the C9ORF72 repeat expansion mutation using human induced pluripotent stem cells. Brain Pathology, 2017, 27, 518-524.	2.1	9
76	Viral delivery of C9ORF72 hexanucleotide repeat expansions in mice lead to repeat length dependent neuropathology and behavioral deficits.. DMM Disease Models and Mechanisms, 2017, 10, 859-868.	1.2	25
77	RNA phase transitions in repeat expansion disorders. Nature, 2017, 546, 243-247.	13.7	651
78	Genetic models of C9orf72: what is toxic?. Current Opinion in Genetics and Development, 2017, 44, 92-101.	1.5	50
79	New developments in RAN translation: insights from multiple diseases. Current Opinion in Genetics and Development, 2017, 44, 125-134.	1.5	81
80	Poly(GP) proteins are a useful pharmacodynamic marker for C9ORF72 -associated amyotrophic lateral sclerosis. Science Translational Medicine, 2017, 9, .	5.8	179
81	Antibodies inhibit transmission and aggregation of C9orf72 polyâ€•<scp>GA</scp> dipeptide repeat proteins. EMBO Molecular Medicine, 2017, 9, 687-702.	3.3	70
82	Dysregulated molecular pathways in amyotrophic lateral sclerosisâ€“frontotemporal dementia spectrum disorder. EMBO Journal, 2017, 36, 2931-2950.	3.5	150
83	Genetic mutations in RNA-binding proteins and their roles in ALS. Human Genetics, 2017, 136, 1193-1214.	1.8	168
84	Homeostatic plasticity can be induced and expressed to restore synaptic strength at neuromuscular junctions undergoing ALS-related degeneration. Human Molecular Genetics, 2017, 26, 4153-4167.	1.4	56
85	RNA binding proteins and the pathological cascade in ALS/FTD neurodegeneration. Science Translational Medicine, 2017, 9, .	5.8	72
86	Motoneuron Disease: Basic Science. Advances in Neurobiology, 2017, 15, 163-190.	1.3	5
87	TDP-43 in the spectrum of MND-FTLD pathologies. Molecular and Cellular Neurosciences, 2017, 83, 46-54.	1.0	20
88	Neonatal AAV delivery of alpha-synuclein induces pathology in the adult mouse brain. Acta Neuropathologica Communications, 2017, 5, 51.	2.4	24
89	C9ORF72 is a GDP/GTP exchange factor for Rab8 and Rab39 and regulates autophagy. Small GTPases, 2017, 8, 181-186.	0.7	69
90	Pathogenic determinants and mechanisms of ALS/FTD linked to hexanucleotide repeat expansions in the C9orf72 gene. Neuroscience Letters, 2017, 636, 16-26.	1.0	36
91	Body Mass Index and Amyotrophic Lateral Sclerosis: A Study of US Military Veterans. American Journal of Epidemiology, 2017, 185, 362-371.	1.6	50

#	Article	IF	Citations
92	Stem cells in neurodegeneration: mind the gap. , 2017, , 81-100.		0
93	Mouse Models of C9orf72 Hexanucleotide Repeat Expansion in Amyotrophic Lateral Sclerosis/ Frontotemporal Dementia. Frontiers in Cellular Neuroscience, 2017, 11, 196.	1.8	44
94	Unraveling the Role of RNA Mediated Toxicity of C9orf72 Repeats in C9-FTD/ALS. Frontiers in Neuroscience, 2017, 11, 711.	1.4	46
95	The Role of Dipeptide Repeats in C9ORF72-Related ALS-FTD. Frontiers in Molecular Neuroscience, 2017, 10, 35.	1.4	207
96	Amyotrophic Lateral Sclerosis Pathogenesis Converges on Defects in Protein Homeostasis Associated with TDP-43 Mislocalization and Proteasome-Mediated Degradation Overload. Current Topics in Developmental Biology, 2017, 121, 111-171.	1.0	26
97	C9ORF72 hexanucleotide repeat exerts toxicity in a stable, inducible motor neuronal cell model, which is rescued by partial depletion of Pten. Human Molecular Genetics, 2017, 26, 1133-1145.	1.4	23
98	Animal Models for the Study of Human Neurodegenerative Diseases. , 2017, , 1109-1129.		4
99	Modelling amyotrophic lateral sclerosis in mice. Drug Discovery Today: Disease Models, 2017, 25-26, 35-44.	1.2	11
100	Emerging understanding of the genotypeâ€“phenotype relationship in amyotrophic lateral sclerosis. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2018, 148, 603-623.	1.0	30
101	Myelin abnormality in Charcotâ€“Marieâ€“Tooth type 4J recapitulates features of acquired demyelination. Annals of Neurology, 2018, 83, 756-770.	2.8	28
102	Disruption of ERâ^'mitochondria signalling in fronto-temporal dementia and related amyotrophic lateral sclerosis. Cell Death and Disease, 2018, 9, 327.	2.7	54
103	Interrelationship between the Levels of C9orf72 and Amyloid-Î² Protein Precursor and Amyloid-Î² in Human CellsÂand Brain Samples. Journal of Alzheimer's Disease, 2018, 62, 269-278.	1.2	3
104	RNA versus protein toxicity in C9orf72 ALS/FTLD. Acta Neuropathologica, 2018, 135, 475-479.	3.9	8
105	Exploring the genetics and non-cell autonomous mechanisms underlying ALS/FTLD. Cell Death and Differentiation, 2018, 25, 648-662.	5.0	55
106	Haploinsufficiency leads to neurodegeneration in C9ORF72 ALS/FTD human induced motor neurons. Nature Medicine, 2018, 24, 313-325.	15.2	445
107	Multi-Omics for Biomarker Discovery and Target Validation in Biofluids for Amyotrophic Lateral Sclerosis Diagnosis. OMICS A Journal of Integrative Biology, 2018, 22, 52-64.	1.0	31
108	CUG initiation and frameshifting enable production of dipeptide repeat proteins from ALS/FTD C9ORF72 transcripts. Nature Communications, 2018, 9, 152.	5.8	123
109	Incorporating upper motor neuron health in ALS drug discovery. Drug Discovery Today, 2018, 23, 696-703.	3.2	18

#	Article	IF	Citations
110	A zebrafish model for C9orf72 ALS reveals RNA toxicity as a pathogenic mechanism. Acta Neuropathologica, 2018, 135, 427-443.	3.9	98
111	ALS Genes in the Genomic Era and their Implications for FTD. Trends in Genetics, 2018, 34, 404-423.	2.9	229
112	C9orf72 is essential for neurodevelopment and motility mediated by Cyclin G1. Experimental Neurology, 2018, 304, 114-124.	2.0	34
113	Morphological changes in the cerebellum as a result of ethanol treatment and cigarette smoke exposure: A study on astrogliosis, apoptosis and Purkinje cells. Neuroscience Letters, 2018, 672, 70-77.	1.0	9
114	Mouse models of ALS: Past, present and future. Brain Research, 2018, 1693, 1-10.	1.1	89
115	Expression of C9orf72-related dipeptides impairs motor function in a vertebrate model. Human Molecular Genetics, 2018, 27, 1754-1762.	1.4	44
116	Disease Mechanisms ofC9ORF72Repeat Expansions. Cold Spring Harbor Perspectives in Medicine, 2018, 8, a024224.	2.9	75
117	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. Acta Neuropathologica, 2018, 135, 459-474.	3.9	152
118	Stable transgenic C9orf72 zebrafish model key aspects of the ALS/FTD phenotype and reveal novel pathological features. Acta Neuropathologica Communications, 2018, 6, 125.	2.4	47
119	A feedback loop between dipeptide-repeat protein, TDP-43 and karyopherin-Î± mediates C9orf72-related neurodegeneration. Brain, 2018, 141, 2908-2924.	3.7	75
120	Animal models of neurodegenerative diseases. Nature Neuroscience, 2018, 21, 1370-1379.	7.1	358
121	Translational Research on Amyotrophic Lateral Sclerosis (ALS): The Preclinical SOD1 Mouse Model. Journal of Translational Neurosciences, 2018, 03, .	0.2	4
122	C9orf72 Repeat Expansion Frequency among Patients with Huntington Disease Genetic Testing. Neurodegenerative Diseases, 2018, 18, 239-253.	0.8	11
123	Repeat-associated non-ATG (RAN) translation. Journal of Biological Chemistry, 2018, 293, 16127-16141.	1.6	81
124	Encoding activities of non-coding RNAs. Theranostics, 2018, 8, 2496-2507.	4.6	42
125	From Mouse Models to Human Disease: An Approach for Amyotrophic Lateral Sclerosis. In Vivo, 2018, 32, 983-998.	0.6	17
126	The C9ORF72 Gene, Implicated in Amyotrophic Lateral Sclerosis and Frontotemporal Dementia, Encodes a Protein That Functions in Control of Endothelin and Glutamate Signaling. Molecular and Cellular Biology, 2018, 38, .	1.1	26
127	Motor neuron differentiation of iPSCs obtained from peripheral blood of a mutant TARDBP ALS patient. Stem Cell Research, 2018, 30, 61-68.	0.3	21

#	Article	IF	Citations
128	Alzheimerâ€™s Disease and Frontotemporal Lobar Degeneration: Mouse Models. , 2018, , 187-219.		1
130	Driven to decay: Excitability and synaptic abnormalities in amyotrophic lateral sclerosis. Brain Research Bulletin, 2018, 140, 318-333.	1.4	63
131	Poly(GR) impairs protein translation and stress granule dynamics in C9orf72-associated frontotemporal dementia and amyotrophic lateral sclerosis. Nature Medicine, 2018, 24, 1136-1142.	15.2	241
132	Loss of Tmem106b is unable to ameliorate frontotemporal dementia-like phenotypes in an AAV mouse model of C9ORF72-repeat induced toxicity. Acta Neuropathologica Communications, 2018, 6, 42.	2.4	20
133	Novel antibodies reveal presynaptic localization of C9orf72 protein and reduced protein levels in C9orf72 mutation carriers. Acta Neuropathologica Communications, 2018, 6, 72.	2.4	87
134	Dipeptide repeat proteins activate a heat shock response found in C9ORF72-ALS/FTLD patients. Acta Neuropathologica Communications, 2018, 6, 55.	2.4	24
135	A complex of C9ORF72 and p62 uses arginine methylation to eliminate stress granules by autophagy. Nature Communications, 2018, 9, 2794.	5.8	126
136	C9ORF72 dipeptide repeat poly-GA inclusions promote intracellular aggregation of phosphorylated TDP-43. Human Molecular Genetics, 2018, 27, 2658-2670.	1.4	39
137	C9orf72-mediated ALS and FTD: multiple pathways to disease. Nature Reviews Neurology, 2018, 14, 544-558.	4.9	478
138	Repeat-Associated Non-ATG Translation in Neurological Diseases. Cold Spring Harbor Perspectives in Biology, 2018, 10, a033019.	2.3	33
139	Clinical features and genetic characterization of two dizygotic twins with C9orf72 expansion. Neurobiology of Aging, 2018, 69, 293.e1-293.e8.	1.5	1
140	C9orf72 intermediate repeats are associated with corticobasal degeneration, increased C9orf72 expression and disruption of autophagy. Acta Neuropathologica, 2019, 138, 795-811.	3.9	50
141	Neuroinflammation in frontotemporal dementia. Nature Reviews Neurology, 2019, 15, 540-555.	4.9	159
142	Effects of anodal transcranial direct current stimulation on motor evoked potentials variability in humans. Physiological Reports, 2019, 7, e14087.	0.7	14
143	Splicing repression is a major function of TDP-43 in motor neurons. Acta Neuropathologica, 2019, 138, 813-826.	3.9	60
144	Proteomics Approaches for Biomarker and Drug Target Discovery in ALS and FTD. Frontiers in Neuroscience, 2019, 13, 548.	1.4	57
145	Motor dysfunction and neurodegeneration in a C9orf72 mouse line expressing poly-PR. Nature Communications, 2019, 10, 2906.	5.8	68
146	C-terminal and full length TDP-43 specie differ according to FTLD-TDP lesion type but not genetic mutation. Acta Neuropathologica Communications, 2019, 7, 100.	2.4	11

#	Article	IF	Citations
147	Phenotypic Suppression of ALS/FTD-Associated Neurodegeneration Highlights Mechanisms of Dysfunction. Journal of Neuroscience, 2019, 39, 8217-8224.	1.7	13
148	CRISPR/Cas9 does not facilitate stable expression of long C9orf72 dipeptides in mice. Neurobiology of Aging, 2019, 84, 235.e1-235.e8.	1.5	3
149	Targeted DNA methylation of neurodegenerative disease genes via homology directed repair. Nucleic Acids Research, 2019, 47, 11609-11622.	6.5	13
150	Pathogenic Mechanisms and Therapy Development for C9orf72 Amyotrophic Lateral Sclerosis/Frontotemporal Dementia. Neurotherapeutics, 2019, 16, 1115-1132.	2.1	30
151	Modulation of actin polymerization affects nucleocytoplasmic transport in multiple forms of amyotrophic lateral sclerosis. Nature Communications, 2019, 10, 3827.	5.8	54
152	Repeat-associated non-AUG (RAN) translation mechanisms are running into focus for GGGGCC-repeat associated ALS/FTD. Progress in Neurobiology, 2019, 183, 101697.	2.8	10
153	C9ORF72 protein function and immune dysregulation in amyotrophic lateral sclerosis. Neuroscience Letters, 2019, 713, 134523.	1.0	19
154	The Relevancy of Data Regarding the Metabolism of Iron to Our Understanding of Deregulated Mechanisms in ALS; Hypotheses and Pitfalls. Frontiers in Neuroscience, 2019, 12, 1031.	1.4	19
155	Poly(ADP-Ribosylation) in Age-Related Neurological Disease. Trends in Genetics, 2019, 35, 601-613.	2.9	22
156	Astrocytes and Microglia as Potential Contributors to the Pathogenesis of C9orf72 Repeat Expansion-Associated FTLD and ALS. Frontiers in Neuroscience, 2019, 13, 486.	1.4	47
157	Mouse models of neurodegeneration: Know your question, know your mouse. Science Translational Medicine, 2019, 11, .	5.8	51
158	Drosophila Ref1/ALYREF regulates transcription and toxicity associated with ALS/FTD disease etiologies. Acta Neuropathologica Communications, 2019, 7, 65.	2.4	20
159	C9ORF72-ALS/FTD-associated poly(GR) binds Atp5a1 and compromises mitochondrial function in vivo. Nature Neuroscience, 2019, 22, 851-862.	7.1	161
160	Genetic Convergence Brings Clarity to the Enigmatic Red Line in ALS. Neuron, 2019, 101, 1057-1069.	3.8	111
161	C9orf72 deficiency promotes motor deficits of a C9ALS/FTD mouse model in a dose-dependent manner. Acta Neuropathologica Communications, 2019, 7, 32.	2.4	50
162	Deficits in Social Behavior Precede Cognitive Decline in Middle-Aged Mice. Frontiers in Behavioral Neuroscience, 2019, 13, 55.	1.0	26
163	Aberrant deposition of stress granule-resident proteins linked to C9orf72-associated TDP-43 proteinopathy. Molecular Neurodegeneration, 2019, 14, 9.	4.4	111
164	The coming-of-age of nucleocytoplasmic transport in motor neuron disease and neurodegeneration. Cellular and Molecular Life Sciences, 2019, 76, 2247-2273.	2.4	27

#	Article	IF	Citations
165	Cre-inducible Adeno Associated Virus-mediated Expression of P301L Mutant Tau Causes Motor Deficits and Neuronal Degeneration in the Substantia Nigra. Neuroscience, 2019, 422, 65-74.	1.1	6
166	ALS Genetics, Mechanisms, and Therapeutics: Where Are We Now?. Frontiers in Neuroscience, 2019, 13, 1310.	1.4	487
167	Review: Modelling the pathology and behaviour of frontotemporal dementia. Neuropathology and Applied Neurobiology, 2019, 45, 58-80.	1.8	13
168	The Hairpin Form of r(G4C2)exp in c9ALS/FTD Is Repeat-Associated Non-ATG Translated and a Target for Bioactive Small Molecules. Cell Chemical Biology, 2019, 26, 179-190.e12.	2.5	80
169	Molecular Mechanisms of Neurodegeneration Related to C9orf72 Hexanucleotide Repeat Expansion. Behavioural Neurology, 2019, 2019, 1-18.	1.1	63
170	RNA Binding Proteins and the Pathogenesis of Frontotemporal Lobar Degeneration. Annual Review of Pathology: Mechanisms of Disease, 2019, 14, 469-495.	9.6	32
171	C9orf72-FTD/ALS pathogenesis: evidence from human neuropathological studies. Acta Neuropathologica, 2019, 137, 1-26.	3.9	53
172	Heavy Metal Neurotoxicants Induce ALS-Linked TDP-43 Pathology. Toxicological Sciences, 2019, 167, 105-115.	1.4	37
173	A Chemical Screen Identifies Compounds Limiting the Toxicity of C9ORF72 Dipeptide Repeats. Cell Chemical Biology, 2019, 26, 235-243.e5.	2.5	16
174	Motor Neuron Abnormalities Correlate with Impaired Movement in Zebrafish that Express Mutant Superoxide Dismutase 1. Zebrafish, 2019, 16, 8-14.	0.5	16
175	Redox Mechanisms in Neurodegeneration: From Disease Outcomes to Therapeutic Opportunities. Antioxidants and Redox Signaling, 2019, 30, 1450-1499.	2.5	90
176	Glia-specific autophagy dysfunction in ALS. Seminars in Cell and Developmental Biology, 2020, 99, 172-182.	2.3	39
177	Back to the origins: Human brain organoids to investigate neurodegeneration. Brain Research, 2020, 1727, 146561.	1.1	12
178	RNA toxicity in nonâ€coding repeat expansion disorders. EMBO Journal, 2020, 39, e101112.	3.5	135
179	Mutant C9orf72 human iPSCâ€derived astrocytes cause nonâ€cell autonomous motor neuron pathophysiology. Glia, 2020, 68, 1046-1064.	2.5	90
180	Modeling cell-autonomous motor neuron phenotypes in ALS using iPSCs. Neurobiology of Disease, 2020, 134, 104680.	2.1	55
181	The Development of C9orf72-Related Amyotrophic Lateral Sclerosis and Frontotemporal Dementia Disorders. Frontiers in Genetics, 2020, 11, 562758.	1.1	23
182	Preclinical models of disease and multimorbidity with focus upon cardiovascular disease and dementia. Mechanisms of Ageing and Development, 2020, 192, 111361.	2.2	7

#	Article	IF	Citations
183	Absence of Survival and Motor Deficits in 500 Repeat C9ORF72 BAC Mice. Neuron, 2020, 108, 775-783.e4.	3.8	33
184	Drosophila Glia: Models for Human Neurodevelopmental and Neurodegenerative Disorders. International Journal of Molecular Sciences, 2020, 21, 4859.	1.8	17
185	From Multi-Omics Approaches to Precision Medicine in Amyotrophic Lateral Sclerosis. Frontiers in Neuroscience, 2020, 14, 577755.	1.4	35
186	Is the ALS a motor neuron disease or a hematopoietic stem cell disease?. Progress in Brain Research, 2020, 258, 381-396.	0.9	5
187	Antisense Transcription across Nucleotide Repeat Expansions in Neurodegenerative and Neuromuscular Diseases: Progress and Mysteries. Genes, 2020, 11, 1418.	1.0	11
188	The carboxyl termini of RAN translated GGGGCC nucleotide repeat expansions modulate toxicity in models of ALS/FTD. Acta Neuropathologica Communications, 2020, 8, 122.	2.4	15
189	BV-2 Microglial Cells Overexpressing C9orf72 Hexanucleotide Repeat Expansion Produce DPR Proteins and Show Normal Functionality but No RNA Foci. Frontiers in Neurology, 2020, 11, 550140.	1.1	4
190	C9orf72 poly(GR) aggregation induces TDP-43 proteinopathy. Science Translational Medicine, 2020, 12, .	5.8	115
191	The Role of TDP-43Âin Genome Repair and beyond in Amyotrophic Lateral Sclerosis. , 0, , .		0
192	Insights into disease mechanisms and potential therapeutics for C9orf72-related amyotrophic lateral sclerosis/frontotemporal dementia. Ageing Research Reviews, 2020, 64, 101172.	5.0	5
193	RNA-mediated toxicity in C9orf72 ALS and FTD. Neurobiology of Disease, 2020, 145, 105055.	2.1	31
194	Role of the C9ORF72 Gene in the Pathogenesis of Amyotrophic Lateral Sclerosis and Frontotemporal Dementia. Neuroscience Bulletin, 2020, 36, 1057-1070.	1.5	4
195	Gene Therapy for Neurodegenerative Diseases: Slowing Down the Ticking Clock. Frontiers in Neuroscience, 2020, 14, 580179.	1.4	42
196	Female sex mitigates motor and behavioural phenotypes in TDP-43Q331K knock-in mice. Scientific Reports, 2020, 10, 19220.	1.6	9
197	In vivo stress granule misprocessing evidenced in a FUS knock-in ALS mouse model. Brain, 2020, 143, 1350-1367.	3.7	42
198	Hexanucleotide Repeat Expansions in c9FTD/ALS and SCA36 Confer Selective Patterns of Neurodegeneration InÂVivo. Cell Reports, 2020, 31, 107616.	2.9	37
199	Loss of TMEM106B leads to myelination deficits: implications for frontotemporal dementia treatment strategies. Brain, 2020, 143, 1905-1919.	3.7	44
200	Divergence, Convergence, and Therapeutic Implications: A Cell Biology Perspective of C9ORF72-ALS/FTD. Molecular Neurodegeneration, 2020, 15, 34.	4.4	32

#	Article	IF	Citations
201	Congenic expression of poly-GA but not poly-PR in mice triggers selective neuron loss and interferon responses found in C9orf72 ALS. Acta Neuropathologica, 2020, 140, 121-142.	3.9	44
202	Cellâ€toâ€cell transmission of C9orf72 polyâ€(Glyâ€Ala) triggers key features of <scp>ALS</scp> / <scp>FTD</scp>. EMBO Journal, 2020, 39, e102811.	3.5	51
203	Omics Approach to Axonal Dysfunction of Motor Neurons in Amyotrophic Lateral Sclerosis (ALS). Frontiers in Neuroscience, 2020, 14, 194.	1.4	42
204	Quadruplex targets in neurodegenerative diseases. Annual Reports in Medicinal Chemistry, 2020, , 441-483.	0.5	1
205	An ENU-induced mutation in Twist1 transactivation domain causes hindlimb polydactyly with complete penetrance and dominant-negatively impairs E2A-dependent transcription. Scientific Reports, 2020, 10, 2501.	1.6	5
206	Knocking out C9ORF72 Exacerbates Axonal Trafficking Defects Associated with Hexanucleotide Repeat Expansion and Reduces Levels of Heat Shock Proteins. Stem Cell Reports, 2020, 14, 390-405.	2.3	48
207	Reduced autophagy upon C9ORF72 loss synergizes with dipeptide repeat protein toxicity in G4C2 repeat expansion disorders. EMBO Journal, 2020, 39, e100574.	3.5	100
208	Plasma glial fibrillary acidic protein is raised in progranulin-associated frontotemporal dementia. Journal of Neurology, Neurosurgery and Psychiatry, 2020, 91, 263-270.	0.9	106
209	Hippocampal firing rates count. Nature Neuroscience, 2020, 23, 597-599.	7.1	2
210	IN VITRO AND IN VIVO MODELS OF AMYOTROPHIC LATERAL SCLEROSIS: AN UPDATED OVERVIEW. Brain Research Bulletin, 2020, 159, 32-43.	1.4	36
211	Reduced C9ORF72 function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in C9orf72. Nature Neuroscience, 2020, 23, 615-624.	7.1	157
212	Dipeptide repeat derived from C9orf72 hexanucleotide expansions forms amyloids or natively unfolded structures inÂvitro. Biochemical and Biophysical Research Communications, 2020, 526, 410-416.	1.0	7
213	Realizing the gains and losses in C9ORF72 ALS/FTD. Nature Neuroscience, 2020, 23, 596-597.	7.1	4
214	Long nonâ€coding RNAs in motor neuron development and disease. Journal of Neurochemistry, 2021, 156, 777-801.	2.1	22
215	Glial Cell Dysfunction in C9orf72-Related Amyotrophic Lateral Sclerosis and Frontotemporal Dementia. Cells, 2021, 10, 249.	1.8	16
216	Molecular conformations and dynamics of nucleotide repeats associated with neurodegenerative diseases: double helices and CAG hairpin loops. Computational and Structural Biotechnology Journal, 2021, 19, 2819-2832.	1.9	11
217	Little Helpers or Mean Rogueâ€"Role of Microglia in Animal Models of Amyotrophic Lateral Sclerosis. International Journal of Molecular Sciences, 2021, 22, 993.	1.8	8
218	Inducible expression of human C9ORF72 36Ã— G4C2 hexanucleotide repeats is sufficient to cause RAN translation and rapid muscular atrophy in mice. DMM Disease Models and Mechanisms, 2021, 14, .	1.2	11

#	Article	IF	Citations
219	Concomitant gain and loss of function pathomechanisms in C9ORF72 amyotrophic lateral sclerosis. Life Science Alliance, 2021, 4, e202000764.	1.3	11
220	Variant-selective stereopure oligonucleotides protect against pathologies associated with C9orf72-repeat expansion in preclinical models. Nature Communications, 2021, 12, 847.	5.8	48
221	p53 is a central regulator driving neurodegeneration caused by C9orf72 poly(PR). Cell, 2021, 184, 689-708.e20.	13.5	104
222	Emerging Perspectives on Dipeptide Repeat Proteins in C9ORF72 ALS/FTD. Frontiers in Cellular Neuroscience, 2021, 15, 637548.	1.8	29
223	Multi-phaseted problems of TDP-43 in selective neuronal vulnerability in ALS. Cellular and Molecular Life Sciences, 2021, 78, 4453-4465.	2.4	6
224	The progress inC9orf72research: ALS/FTD pathogenesis, functions and structure. Small GTPases, 2022, 13, 56-76.	0.7	6
225	Localization of RNAs in the nucleus: cis- and trans- regulation. RNA Biology, 2021, 18, 2073-2086.	1.5	10
226	The Cryo-EM Effect: Structural Biology of Neurodegenerative Disease Proteostasis Factors. Journal of Neuropathology and Experimental Neurology, 2021, 80, 494-513.	0.9	4
227	Non-neuronal cells in amyotrophic lateral sclerosis â€" from pathogenesis to biomarkers. Nature Reviews Neurology, 2021, 17, 333-348.	4.9	78
229	Where and Why Modeling Amyotrophic Lateral Sclerosis. International Journal of Molecular Sciences, 2021, 22, 3977.	1.8	20
231	C9ORF72: What It Is, What It Does, and Why It Matters. Frontiers in Cellular Neuroscience, 2021, 15, 661447.	1.8	61
232	Widespread displacement of DNAâ€•and RNAâ€binding factors underlies toxicity of arginineâ€rich cellâ€penetrating peptides. EMBO Journal, 2021, 40, e103311.	3.5	21
233	Expression of C9orf72 hexanucleotide repeat expansion leads to formation of RNA foci and dipeptide repeat proteins but does not influence autophagy or proteasomal function in neuronal cells. Biochimica Et Biophysica Acta - Molecular Cell Research, 2021, 1868, 119021.	1.9	5
234	Early weight instability is associated with cognitive decline and poor survival in amyotrophic lateral sclerosis. Brain Research Bulletin, 2021, 171, 10-15.	1.4	9
235	Mini-Review: Induced pluripotent stem cells and the search for new cell-specific ALS therapeutic targets. Neuroscience Letters, 2021, 755, 135911.	1.0	20
236	UBQLN2-HSP70 axis reduces poly-Gly-Ala aggregates and alleviates behavioral defects in the C9ORF72 animal model. Neuron, 2021, 109, 1949-1962.e6.	3.8	24
237	The Skeletal Muscle Emerges as a New Disease Target in Amyotrophic Lateral Sclerosis. Journal of Personalized Medicine, 2021, 11, 671.	1.1	20
238	Nanoscopic investigation of C9orf72 poly-GA oligomers on nuclear membrane disruption by a photoinducible platform. Communications Chemistry, 2021, 4, .	2.0	2

#	Article	IF	Citations
239	FTLD Patientâ€“Derived Fibroblasts Show Defective Mitochondrial Function and Accumulation of p62. Molecular Neurobiology, 2021, 58, 5438-5458.	1.9	4
240	Prion-Like Proteins in Phase Separation and Their Link to Disease. Biomolecules, 2021, 11, 1014.	1.8	26
241	Exploring the alternative: Fish, flies and worms as preclinical models for ALS. Neuroscience Letters, 2021, 759, 136041.	1.0	8
242	NRF2 as a therapeutic opportunity to impact in the molecular roadmap of ALS. Free Radical Biology and Medicine, 2021, 173, 125-141.	1.3	21
243	Elucidating the Role of Cerebellar Synaptic Dysfunction in C9orf72-ALS/FTD â€" a Systematic Review and Meta-Analysis. Cerebellum, 2022, 21, 681-714.	1.4	3
244	The porphyrin TMPyP4 inhibits elongation during the noncanonical translation of the FTLD/ALS-associated GGGGCC repeat in the C9orf72 gene. Journal of Biological Chemistry, 2021, 297, 101120.	1.6	17
245	NEAT1 lncRNA and amyotrophic lateral sclerosis. Neurochemistry International, 2021, 150, 105175.	1.9	12
246	Hypothalamic symptoms of frontotemporal dementia disorders. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2021, 182, 269-280.	1.0	9
247	Unraveling molecular biology of C9ORF72 repeat expansions in amyotrophic lateral sclerosis-frontotemporal dementia: Implications for therapy. , 2021, , 19-47.		0
248	Chimeric Peptide Species Contribute to Divergent Dipeptide Repeat Pathology in c9ALS/FTD and SCA36. Neuron, 2020, 107, 292-305.e6.	3.8	51
249	From Reproducibility to Translation in Neurodegenerative Disease. ILAR Journal, 2017, 58, 106-114.	1.8	11
256	Disparity of outcomes: the limits of modeling amyotrophic lateral sclerosis in murine models and translating results clinically. Journal of Controversies in Biomedical Research, 2015, 1, 4-22.	0.5	2
257	Modeling neuromuscular junctions in vitro: A review of the current progress employing human induced pluripotent stem cells. AIMS Cell and Tissue Engineering, 2018, 2, 91-118.	0.4	5
258	Mislocalization, aggregation formation and defect in proteolysis in ALS. AIMS Molecular Science, 2016, 3, 246-268.	0.3	2
259	Toward precision medicine in amyotrophic lateral sclerosis. Annals of Translational Medicine, 2016, 4, 27.	0.7	10
260	La accesibilidad de los portales web de las universidades pÃºblicas andaluzas. Revista Espanola De Documentacion Cientifica, 2017, 40, 169.	0.1	10
262	Modeling C9orf72-Related Frontotemporal Dementia and Amyotrophic Lateral Sclerosis in Drosophila. Frontiers in Cellular Neuroscience, 2021, 15, 770937.	1.8	4
263	A C. elegans model of C9orf72-associated ALS/FTD uncovers a conserved role for eIF2D in RAN translation. Nature Communications, 2021, 12, 6025.	5.8	27

#	Article	IF	Citations
264	Dual-isoform hUBE3A gene transfer improves behavioral and seizure outcomes in Angelman syndrome model mice. JCI Insight, 2021, 6, .	2.3	12
266	Poly(GP) proteins: a potential pharmacodynamic marker in ALS and FTD. Annals of Translational Medicine, 2017, 5, 504-504.	0.7	0
267	Solving the Puzzle of Neurodegeneration. , 2018, , 1-22.		2
268	Amyotrophic lateral sclerosis: characteristics of the immunophenotype of hematopoietic precursor cells as a potential biomarker for early diagnostics of fatal disease. Genes and Cells, 2019, 14, 72-79.	0.2	1
272	A behavioral screen for mediators of age-dependent TDP-43 neurodegeneration identifies SF2/SRSF1 among a group of potent suppressors in both neurons and glia. PLoS Genetics, 2021, 17, e1009882.	1.5	14
273	Repeat length increases disease penetrance and severity in C9orf72 ALS/FTD BAC transgenic mice. Human Molecular Genetics, 2021, 29, 3900-3918.	1.4	7
274	New opportunities for treatment of neurodegenerative disease through the modulation of TDP-43. , 2022, , 183-250.		1
275	Proteostasis deregulation as a driver of C9ORF72 pathogenesis. Journal of Neurochemistry, 2021, 159, 941.	2.1	2
276	Dysfunction of RNA/RNA-Binding Proteins in ALS Astrocytes and Microglia. Cells, 2021, 10, 3005.	1.8	6
278	Nearly 30 Years of Animal Models to Study Amyotrophic Lateral Sclerosis: A Historical Overview and Future Perspectives. International Journal of Molecular Sciences, 2021, 22, 12236.	1.8	40
279	C9orf72 ALS/FTD dipeptide repeat protein levels are reduced by small molecules that inhibit PKA or enhance protein degradation. EMBO Journal, 2022, 41, e105026.	3.5	13
280	An Integrated Approach to Studying Rare Neuromuscular Diseases Using Animal and Human Cell-Based Models. Frontiers in Cell and Developmental Biology, 2021, 9, 801819.	1.8	2
281	RAN proteins in neurodegenerative disease: Repeating themes and unifying therapeutic strategies. Current Opinion in Neurobiology, 2022, 72, 160-170.	2.0	10
282	HDAC6 Interacts With Poly (GA) and Modulates its Accumulation in c9FTD/ALS. Frontiers in Cell and Developmental Biology, 2021, 9, 809942.	1.8	4
283	C9orf72 hexanucleotide repeat expansion leads to altered neuronal and dendritic spine morphology and synaptic dysfunction. Neurobiology of Disease, 2022, 162, 105584.	2.1	5
284	TDP-43 pathology: From noxious assembly to therapeutic removal. Progress in Neurobiology, 2022, 211, 102229.	2.8	30
285	Reduced mtDNA Copy Number in the Prefrontal Cortex of C9ORF72 Patients. Molecular Neurobiology, 2022, 59, 1230-1237.	1.9	4
287	Pathophysiology of stress granules: An emerging link to diseases (Review). International Journal of Molecular Medicine, 2022, 49, .	1.8	14

#	Article	IF	Citations
288	SOD1 in ALS: Taking Stock in Pathogenic Mechanisms and the Role of Glial and Muscle Cells. Antioxidants, 2022, 11, 614.	2.2	26
289	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.	1.8	5
290	Modelling amyotrophic lateral sclerosis in rodents. Nature Reviews Neuroscience, 2022, 23, 231-251.	4.9	17
291	The role of inflammation in neurodegeneration: novel insights into the role of the immune system in C9orf72 HRE-mediated ALS/FTD. Molecular Neurodegeneration, 2022, 17, 22.	4.4	24
292	Mutation in protein disulfide isomerase A3 causes neurodevelopmental defects by disturbing endoplasmic reticulum proteostasis. EMBO Journal, 2022, 41, e105531.	3.5	11
293	Transcriptomic analysis of frontotemporal lobar degeneration with TDP-43 pathology reveals cellular alterations across multiple brain regions. Acta Neuropathologica, 2022, 143, 383-401.	3.9	20
294	Cerebellar pathology in motor neuron disease: neuroplasticity and neurodegeneration. Neural Regeneration Research, 2022, 17, 2335.	1.6	14
298	Genetic and Epigenetic Interplay Define Disease Onset and Severity in Repeat Diseases. Frontiers in Aging Neuroscience, 2022, 14, 750629.	1.7	4
300	Drug screen in iPSC-Neurons identifies nucleoside analogs as inhibitors of (G4C2)n expression in C9orf72 ALS/FTD. Cell Reports, 2022, 39, 110913.	2.9	7
301	NUP62 localizes to ALS/FTLD pathological assemblies and contributes to TDP-43 insolubility. Nature Communications, 2022, 13, .	5.8	26
302	Poly(ADP-ribose) promotes toxicity of C9ORF72 arginine-rich dipeptide repeat proteins. Science Translational Medicine, 2022, 14, .	5.8	9
303	Staufen Impairs Autophagy in Neurodegeneration. Annals of Neurology, 2023, 93, 398-416.	2.8	4
304	Two FTD-ALS genes converge on the endosomal pathway to induce TDP-43 pathology and degeneration. Science, 2022, 378, 94-99.	6.0	32
305	C9orf72 regulates the unfolded protein response and stress granule formation by interacting with eIF2Î±. Theranostics, 2022, 12, 7289-7306.	4.6	6
306	Comprehensive evaluation of human-derived anti-poly-GA antibodies in cellular and animal models of C9orf72 disease. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, .	3.3	3
307	Models of Neurodegenerative Diseases. Learning Materials in Biosciences, 2023, , 179-209.	0.2	0
308	Dorsomedial prefrontal hypoexcitability underlies lost empathy in frontotemporal dementia. Neuron, 2023, 111, 797-806.e6.	3.8	6
309	Therapeutic reduction of GGGGCC repeat RNA levels by hnRNPA3 suppresses neurodegeneration in Drosophila models of C9orf72-linked ALS/FTD. Human Molecular Genetics, 2023, 32, 1673-1682.	1.4	7

#	Article	IF	Citations
310	Negative regulation of TREM2-mediated C9orf72 poly-GA clearance by the NLRP3 inflammasome. Cell Reports, 2023, 42, 112133.	2.9	7
311	C9ORF72 knockdown triggers FTD-like symptoms and cell pathology in mice. Frontiers in Cellular Neuroscience, 0, 17, .	1.8	2
312	Antisense, but not sense, repeat expanded RNAs activate PKR/eIF2Î±-dependent ISR in C9ORF72 FTD/ALS. ELife, 0, 12, .	2.8	3
321	Neurogenetic motor disorders. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2023, , 183-250.	1.0	0