

# Tania F Gendron

## List of Publications by Year in descending order

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Version: 2024-02-01

78  
papers

10,747  
citations

44069

48  
h-index

69250

77  
g-index

84  
all docs

84  
docs citations

84  
times ranked

8071  
citing authors

#	ARTICLE	IF	CITATIONS
1	Comprehensive cross-sectional and longitudinal analyses of plasma neurofilament light across FTD spectrum disorders. <i>Cell Reports Medicine</i> , 2022, 3, 100607.	6.5	21
2	Sensitive ELISA-based detection method for the mitophagy marker p-S65-Ub in human cells, autopsy brain, and blood samples. <i>Autophagy</i> , 2021, 17, 2613-2628.	9.1	29
3	Alterations of mesenchymal stromal cells in cerebrospinal fluid: insights from transcriptomics and an ALS clinical trial. <i>Stem Cell Research and Therapy</i> , 2021, 12, 187.	5.5	8
4	Deep vein thrombosis and pulmonary embolism among hospitalized coronavirus disease 2019â€“positive patients predicted for higher mortality and prolonged intensive care unit and hospital stays in a multisite healthcare system. <i>Journal of Vascular Surgery: Venous and Lymphatic Disorders</i> , 2021, 9, 1361-1370.e1.	1.6	17
5	Long-read targeted sequencing uncovers clinicopathological associations for <i>C9orf72</i> -linked diseases. <i>Brain</i> , 2021, 144, 1082-1088.	7.6	17
6	Serum neurofilament light protein correlates with unfavorable clinical outcomes in hospitalized patients with COVID-19. <i>Science Translational Medicine</i> , 2021, 13, .	12.4	67
7	A <i>C. elegans</i> model of <i>C9orf72</i> -associated ALS/FTD uncovers a conserved role for eIF2D in RAN translation. <i>Nature Communications</i> , 2021, 12, 6025.	12.8	27
8	Ribonuclease recruitment using a small molecule reduced c9ALS/FTD r(G <sub>4</sub> C <sub>2</sub> ) Tj ETQq0,0,0 rgBT /Overlock 1	12.4	39
9	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
10	Toward allele-specific targeting therapy and pharmacodynamic marker for spinocerebellar ataxia type 3. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	32
11	Plasma neurofilament light predicts mortality in patients with stroke. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	51
12	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
13	<i>C9orf72</i> poly(GR) aggregation induces TDP-43 proteinopathy. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	115
14	Cross-sectional and longitudinal measures of chitinase proteins in amyotrophic lateral sclerosis and expression of CHI3L1 in activated astrocytes. <i>Journal of Neurology, Neurosurgery and Psychiatry</i> , 2020, 91, 350-358.	1.9	54
15	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
16	Dipeptide repeat proteins inhibit homology-directed DNA double strand break repair in <i>C9ORF72</i> ALS/FTD. <i>Molecular Neurodegeneration</i> , 2020, 15, 13.	10.8	58
17	Nucleocytoplasmic Proteomic Analysis Uncovers eRF1 and Nonsense-Mediated Decay as Modifiers of ALS/FTD <i>C9orf72</i> Toxicity. <i>Neuron</i> , 2020, 106, 90-107.e13.	8.1	58
18	Reduced <i>C9ORF72</i> function exacerbates gain of toxicity from ALS/FTD-causing repeat expansion in <i>C9orf72</i> . <i>Nature Neuroscience</i> , 2020, 23, 615-624.	14.8	157

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19	Chimeric Peptide Species Contribute to Divergent Dipeptide Repeat Pathology in c9ALS/FTD and SCA36. <i>Neuron</i> , 2020, 107, 292-305.e6.	8.1	51
20	Truncated stathmin-2 is a marker of TDP-43 pathology in frontotemporal dementia. <i>Journal of Clinical Investigation</i> , 2020, 130, 6080-6092.	8.2	117
21	RPS25 is required for efficient RAN translation of C9orf72 and other neurodegenerative disease-associated nucleotide repeats. <i>Nature Neuroscience</i> , 2019, 22, 1383-1388.	14.8	87
22	Extensive transcriptomic study emphasizes importance of vesicular transport in C9orf72 expansion carriers. <i>Acta Neuropathologica Communications</i> , 2019, 7, 150.	5.2	40
23	Aberrant deposition of stress granule-resident proteins linked to C9orf72-associated TDP-43 proteinopathy. <i>Molecular Neurodegeneration</i> , 2019, 14, 9.	10.8	111
24	Heterochromatin anomalies and double-stranded RNA accumulation underlie <i>C9orf72</i> poly(PR) toxicity. <i>Science</i> , 2019, 363, .	12.6	181
25	Transcription elongation factor AFF2/FMR2 regulates expression of expanded GGGGCC repeat-containing C9ORF72 allele in ALS/FTD. <i>Nature Communications</i> , 2019, 10, 5466.	12.8	40
26	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
27	Poly(GP), neurofilament and grey matter deficits in <i>C9orf72</i> expansion carriers. <i>Annals of Clinical and Translational Neurology</i> , 2018, 5, 583-597.	3.7	48
28	CUG initiation and frameshifting enable production of dipeptide repeat proteins from ALS/FTD C9ORF72 transcripts. <i>Nature Communications</i> , 2018, 9, 152.	12.8	123
29	A zebrafish model for C9orf72 ALS reveals RNA toxicity as a pathogenic mechanism. <i>Acta Neuropathologica</i> , 2018, 135, 427-443.	7.7	98
30	Cover Image, Volume 177B, Number 1, January 2018. <i>American Journal of Medical Genetics Part B: Neuropsychiatric Genetics</i> , 2018, 177, i.	1.7	0
31	Disease Mechanisms of <i>C9ORF72</i> Repeat Expansions. <i>Cold Spring Harbor Perspectives in Medicine</i> , 2018, 8, a024224.	6.2	75
32	OPTN p.Met468Arg and ATXN2 intermediate length polyQ extension in families with C9orf72 mediated amyotrophic lateral sclerosis and frontotemporal dementia. <i>American Journal of Medical Genetics Part B: Neuropsychiatric Genetics</i> , 2018, 177, 75-85.	1.7	12
33	Biomarkers for Amyotrophic Lateral Sclerosis and Frontotemporal Dementia Associated With Hexanucleotide Expansion Mutations in C9orf72. <i>Frontiers in Neurology</i> , 2018, 9, 1063.	2.4	28
34	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
35	Loss of Tmem106b is unable to ameliorate frontotemporal dementia-like phenotypes in an AAV mouse model of C9ORF72-repeat induced toxicity. <i>Acta Neuropathologica Communications</i> , 2018, 6, 42.	5.2	20
36	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

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37	Amyotrophic Lateral Sclerosis: An Update for 2018. <i>Mayo Clinic Proceedings</i> , 2018, 93, 1617-1628.	3.0	227
38	Long-read sequencing across the C9orf72 GGGGCC™ repeat expansion: implications for clinical use and genetic discovery efforts in human disease. <i>Molecular Neurodegeneration</i> , 2018, 13, 46.	10.8	111
39	Spinal poly-GA inclusions in a C9orf72 mouse model trigger motor deficits and inflammation without neuron loss. <i>Acta Neuropathologica</i> , 2017, 134, 241-254.	7.7	99
40	In-depth clinico-pathological examination of RNA foci in a large cohort of C9ORF72 expansion carriers. <i>Acta Neuropathologica</i> , 2017, 134, 255-269.	7.7	76
41	Phosphorylated neurofilament heavy chain: A biomarker of survival for C9ORF72-associated amyotrophic lateral sclerosis. <i>Annals of Neurology</i> , 2017, 82, 139-146.	5.3	88
42	Poly(GP) proteins are a useful pharmacodynamic marker for C9ORF72-associated amyotrophic lateral sclerosis. <i>Science Translational Medicine</i> , 2017, 9, .	12.4	179
43	ARHGEF28 p.Lys280Metfs40Ter in an amyotrophic lateral sclerosis family with a C9orf72 expansion. <i>Neurology: Genetics</i> , 2017, 3, e190.	1.9	6
44	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
45	A C9ORF72 BAC mouse model recapitulates key epigenetic perturbations of ALS/FTD. <i>Molecular Neurodegeneration</i> , 2017, 12, 46.	10.8	22
46	Abnormal expression of homeobox genes and transthyretin in C9ORF72 expansion carriers. <i>Neurology: Genetics</i> , 2017, 3, e161.	1.9	12
47	Gain of Toxicity from ALS/FTD-Linked Repeat Expansions in C9ORF72 Is Alleviated by Antisense Oligonucleotides Targeting GGGGCC-Containing RNAs. <i>Neuron</i> , 2016, 90, 535-550.	8.1	437
48	Poly(GR) in C9ORF72-Related ALS/FTD Compromises Mitochondrial Function and Increases Oxidative Stress and DNA Damage in iPSC-Derived Motor Neurons. <i>Neuron</i> , 2016, 92, 383-391.	8.1	323
49	Spt4 selectively regulates the expression of C9orf72 sense and antisense mutant transcripts. <i>Science</i> , 2016, 353, 708-712.	12.6	116
50	Timing and significance of pathological features in C9orf72 expansion-associated frontotemporal dementia. <i>Brain</i> , 2016, 139, 3202-3216.	7.6	136
51	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
52	C9ORF72 poly(GA) aggregates sequester and impair HR23 and nucleocytoplasmic transport proteins. <i>Nature Neuroscience</i> , 2016, 19, 668-677.	14.8	268
53	Distinct brain transcriptome profiles in C9orf72-associated and sporadic ALS. <i>Nature Neuroscience</i> , 2015, 18, 1175-1182.	14.8	330
54	C9ORF72 repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. <i>Science</i> , 2015, 348, 1151-1154.	12.6	332

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55	Differential Toxicity of Nuclear RNA Foci versus Dipeptide Repeat Proteins in a <i>Drosophila</i> Model of C9ORF72 FTD/ALS. <i>Neuron</i> , 2015, 87, 1207-1214.	8.1	176
56	Novel clinical associations with specific C9ORF72 transcripts in patients with repeat expansions in C9ORF72. <i>Acta Neuropathologica</i> , 2015, 130, 863-876.	7.7	104
57	Cerebellar c9RAN proteins associate with clinical and neuropathological characteristics of C9ORF72 repeat expansion carriers. <i>Acta Neuropathologica</i> , 2015, 130, 559-573.	7.7	89
58	Quantitative analysis and clinico-pathological correlations of different dipeptide repeat protein pathologies in C9ORF72 mutation carriers. <i>Acta Neuropathologica</i> , 2015, 130, 845-861.	7.7	204
59	C9orf72 BAC Transgenic Mice Display Typical Pathologic Features of ALS/FTD. <i>Neuron</i> , 2015, 88, 892-901.	8.1	249
60	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
61	<sc>TDP</sc>â€1, the <i><sc>C</sc>aenorhabditis elegans</i> ortholog of <sc>TDP</sc>â€43, limits the accumulation of doubleâ€stranded <sc>RNA</sc>. <i>EMBO Journal</i> , 2014, 33, 2947-2966.	7.8	62
62	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
63	Mechanisms of toxicity in C9FTLD/ALS. <i>Acta Neuropathologica</i> , 2014, 127, 359-376.	7.7	134
64	Characterization of DNA hypermethylation in the cerebellum of c9FTD/ALS patients. <i>Brain Research</i> , 2014, 1584, 15-21.	2.2	70
65	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
66	Targeting RNA Foci in iPSC-Derived Motor Neurons from ALS Patients with a <i>C9ORF72</i> Repeat Expansion. <i>Science Translational Medicine</i> , 2013, 5, 208ra149.	12.4	586
67	Dipeptide repeat proteins are present in the p62 positive inclusions in patients with frontotemporal lobar degeneration and motor neurone disease associated with expansions in C9ORF72. <i>Acta Neuropathologica Communications</i> , 2013, 1, 68.	5.2	162
68	Unconventional Translation of C9ORF72 GGGGCC Expansion Generates Insoluble Polypeptides Specific to c9FTD/ALS. <i>Neuron</i> , 2013, 77, 639-646.	8.1	962
69	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
70	Reduced C9orf72 gene expression in c9FTD/ALS is caused by histone trimethylation, an epigenetic event detectable in blood. <i>Acta Neuropathologica</i> , 2013, 126, 895-905.	7.7	263
71	Does Obesity-Induced Â Phosphorylation Tip the Scale Toward Dementia?. <i>Diabetes</i> , 2013, 62, 1365-1366.	0.6	4
72	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

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73	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
74	TARDBP Mutation Analysis in TDP-43 Proteinopathies and Deciphering the Toxicity of Mutant TDP-43. <i>Journal of Alzheimer's Disease</i> , 2012, 33, S35-S45.	2.6	43
75	Rodent Models of TDP-43 Proteinopathy: Investigating the Mechanisms of TDP-43-Mediated Neurodegeneration. <i>Journal of Molecular Neuroscience</i> , 2011, 45, 486-499.	2.3	59
76	The role of tau in neurodegeneration. <i>Molecular Neurodegeneration</i> , 2009, 4, 13.	10.8	353
77	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
78	Ethanol enhances tau accumulation in neuroblastoma cells that inducibly express tau. <i>Neuroscience Letters</i> , 2008, 443, 67-71.	2.1	21