Fabrizio Tagliavini

List of Publications by Year
in descending order

401 papers	22,675 citations	7096 78 h-index	13771 129 g-index
429 all docs	429 docs citations	429 times ranked	15972 citing authors

#	Article	IF	Citations
1	Neurotoxicity of a prion protein fragment. Nature, 1993, 362, 543-546.	27.8	935
2	New insights into the genetic etiology of Alzheimerâ€™s disease and related dementias. Nature Genetics, 2022, 54, 412-436.	21.4	700
3	Presymptomatic cognitive and neuroanatomical changes in genetic frontotemporal dementia in the Genetic Frontotemporal dementia Initiative (GENFI) study: a cross-sectional analysis. Lancet Neurology, The, 2015, 14, 253-262.	10.2	432
4	Identification of a second bovine amyloidotic spongiform encephalopathy: Molecular similarities with sporadic Creutzfeldt-Jakob disease. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 3065-3070.	7.1	402
5	Frontotemporal Dementia and Corticobasal Degeneration in a Family with a P301S Mutation in Tau. Journal of Neuropathology and Experimental Neurology, 1999, 58, 667-677.	1.7	381
6	Aging-related tau astrogliopathy (ARTAG): harmonized evaluation strategy. Acta Neuropathologica, 2016, 131, 87-102.	7.7	380
7	Staging of Neurofibrillary Pathology in Alzheimer's Disease: A Study of the BrainNet Europe Consortium. Brain Pathology, 2008, 18, 484-496.	4.1	361
8	A Recessive Mutation in the APP Gene with Dominant-Negative Effect on Amyloidogenesis. Science, 2009, 323, 1473-1477.	12.6	357
9	Apoptosis mediated neurotoxicity induced by chronic application of Î² amyloid fragment 25â€“35. NeuroReport, 1993, 4, 523-526.	1.2	355
10	Sporadic human prion diseases: molecular insights and diagnosis. Lancet Neurology, The, 2012, 11, 618-628.	10.2	319
11	Frontotemporal dementia and its subtypes: a genome-wide association study. Lancet Neurology, The, 2014, 13, 686-699.	10.2	302
12	Reversion of prion protein conformational changes by synthetic b-sheet breaker peptides. Lancet, The, 2000, 355, 192-197.	13.7	280
13	Preamyloid deposits in the cerebral cortex of patients with Alzheimer's disease and nondemented individuals. Neuroscience Letters, 1988, 93, 191-196.	2.1	274
14	Vascular variant of prion protein cerebral amyloidosis with tau-positive neurofibrillary tangles: the phenotype of the stop codon 145 mutation in PRNP.. Proceedings of the National Academy of Sciences of the United States of America, 1996, 93, 744-748.	7.1	270
15	Uncovering the heterogeneity and temporal complexity of neurodegenerative diseases with Subtype and Stage Inference. Nature Communications, 2018, 9, 4273.	12.8	263
16	Down patients: Extracellular preamyloid deposits precede neuritic degeneration and senile plaques. Neuroscience Letters, 1989, 97, 232-238.	2.1	242
17	Synthetic peptides homologous to prion protein residues 106-147 form amyloid-like fibrils in vitro.. Proceedings of the National Academy of Sciences of the United States of America, 1993, 90, 9678-9682.	7.1	242
18	Molecular Characteristics of a Protease-Resistant, Amyloidogenic and Neurotoxic Peptide Homologous to Residues 106-126 of the Prion Protein. Biochemical and Biophysical Research Communications, 1993, 194, 1380-1386.	2.1	212

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19	Fatal familial insomnia. Neurology, 1992, 42, 312-312.	1.1	211
20	Antiâ€amyloidogenic activity of tetracyclines: studies in vitro. FEBS Letters, 2001, 487, 404-407.	2.8	205
21	Variably proteaseâ€sensitive prionopathy: A new sporadic disease of the prion protein. Annals of Neurology, 2010, 68, 162-172.	5.3	203
22	Evaluation of Quinacrine Treatment for Prion Diseases. Journal of Virology, 2003, 77, 8462-8469.	3.4	190
23	Prion Protein Amyloidosis. Brain Pathology, 1996, 6, 127-145.	4.1	185
24	Sporadic Creutzfeldt-Jakob disease: Co-occurrence of different types of PrP ^{Sc} in the same brain. Neurology, 1999, 53, 2173-2173.	1.1	185
25	Tetracyclines affect prion infectivity. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 10849-10854.	7.1	184
26	Consensus classification of human prion disease histotypes allows reliable identification of molecular subtypes: an inter-rater study among surveillance centres in Europe and USA. Acta Neuropathologica, 2012, 124, 517-529.	7.7	184
27	Phenotypic Variability of Gerstmann-Straussler-Scheinker Disease is Associated with Prion Protein Heterogeneity. Journal of Neuropathology and Experimental Neurology, 1998, 57, 979-988.	1.7	182
28	Antiâ€“amyloid Î² autoantibodies in cerebral amyloid angiopathyâ€“related inflammation: Implications for amyloidâ€modifying therapies. Annals of Neurology, 2013, 73, 449-458.	5.3	179
29	Substitutions at Codon 22 of Alzheimer's AÎ² Peptide Induce Diverse Conformational Changes and Apoptotic Effects in Human Cerebral Endothelial Cells. Journal of Biological Chemistry, 2000, 275, 27110-27116.	3.4	178
30	Amyloid protein of Gerstmann-StrÃussler-Scheinker disease (Indiana kindred) is an 11 kd fragment of prion protein with an N-terminal glycine at codon 58.. EMBO Journal, 1991, 10, 513-519.	7.8	174
31	Prions in the Urine of Patients with Variant Creutzfeldtâ€“Jakob Disease. New England Journal of Medicine, 2014, 371, 530-539.	27.0	171
32	Effectiveness of Anthracycline Against Experimental Prion Disease in Syrian Hamsters. Science, 1997, 276, 1119-1121.	12.6	168
33	Endogenous Proteolytic Cleavage of Normal and Disease-Associated Isoforms of the Human Prion Protein in Neural and Non-Neural Tissues. American Journal of Pathology, 1998, 153, 1561-1572.	3.8	165
34	Gerstmannâ€Strä ̈usslerâ€Scheinker disease II. Neurofibrillary tangles and plaques with PrPâ€amyloid coexist in an affected family. Neurology, 1989, 39, 1453-1453.	1.1	161
35	Doxycycline in Creutzfeldt-Jakob disease: a phase 2, randomised, double-blind, placebo-controlled trial. Lancet Neurology, The, 2014, 13, 150-158.	10.2	157
36	Tetracycline affects abnormal properties of synthetic PrP peptides and PrPSc in vitro11Edited by J. Karn. Journal of Molecular Biology, 2000, 300, 1309-1322.	4.2	155

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37	Codeposition of Cystatin C with Amyloid-Î² Protein in the Brain of Alzheimer Disease Patients. Journal of Neuropathology and Experimental Neurology, 2001, 60, 94-104.	1.7	154
38	Detection of Misfolded AÎ² Oligomers for Sensitive Biochemical Diagnosis of Alzheimerâ€™s Disease. Cell Reports, 2014, 7, 261-268.	6.4	154
39	Amyloid fibrils in Gerstmann-StrÃussler-Scheinker disease (Indiana and Swedish Kindreds) express only PrP peptides encoded by the mutant allele. Cell, 1994, 79, 695-703.	28.9	152
40	Patterns of gray matter atrophy in genetic frontotemporal dementia: results from the GENFI study. Neurobiology of Aging, 2018, 62, 191-196.	3.1	151
41	Conversion of the BASE Prion Strain into the BSE Strain: The Origin of BSE?. PLoS Pathogens, 2007, 3, e31.	4.7	146
42	Gerstmannâ€šStrÃusslerâ€šScheinker Disease and the Indiana Kindred. Brain Pathology, 1995, 5, 61-75.	4.1	145
43	Common variants in Alzheimerâ€™s disease and risk stratification by polygenic risk scores. Nature Communications, 2021, 12, 3417.	12.8	140
44	Amyloid Î² plaque-associated proteins C1q and SAP enhance the AÎ²1â€“42 peptide-induced cytokine secretion by adult human microglia in vitro. Acta Neuropathologica, 2003, 105, 135-144.	7.7	129
45	Serum neurofilament light chain in genetic frontotemporal dementia: a longitudinal, multicentre cohort study. Lancet Neurology, The, 2019, 18, 1103-1111.	10.2	128
46	Pre-symptomatic detection of prions by cyclic amplification of protein misfolding. FEBS Letters, 2005, 579, 638-642.	2.8	127
47	Basal nucleus of meynert. Journal of the Neurological Sciences, 1983, 62, 243-260.	0.6	125
48	Detection of prions in blood from patients with variant Creutzfeldt-Jakob disease. Science Translational Medicine, 2016, 8, 370ra183.	12.4	120
49	A 7-kDa Prion Protein (PrP) Fragment, an Integral Component of the PrP Region Required for Infectivity, Is the Major Amyloid Protein in Gerstmann-StrÃussler-Scheinker Disease A117V. Journal of Biological Chemistry, 2001, 276, 6009-6015.	3.4	119
50	A Neurotoxic Prion Protein Fragment Induces Rat Astroglial Proliferation and Hypertrophy. European Journal of Neuroscience, 1994, 6, 1415-1422.	2.6	112
51	Immune-related genetic enrichment in frontotemporal dementia: An analysis of genome-wide association studies. PLoS Medicine, 2018, 15, e1002487.	8.4	111
52	Identification of evolutionarily conserved gene networks mediating neurodegenerative dementia. Nature Medicine, 2019, 25, 152-164.	30.7	111
53	Microglial cells respond to amyloidogenic PrP peptide by the production of inflammatory cytokines. NeuroReport, 1999, 10, 723-729.	1.2	109
54	Cerebrospinal fluid realâ€time quakingâ€induced conversion is a robust and reliable test for sporadic creutzfeldtâ€“jakob disease: An international study. Annals of Neurology, 2016, 80, 160-165.	5.3	107

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55	Genetic architecture of sporadic frontotemporal dementia and overlap with Alzheimer's and Parkinson's diseases. Journal of Neurology, Neurosurgery and Psychiatry, 2017, 88, 152-164.	1.9	107
56	Efficient RT-QuIC seeding activity for Î±-synuclein in olfactory mucosa samples of patients with Parkinsonâ€™s disease and multiple system atrophy. Translational Neurodegeneration, 2019, 8, 24.	8.0	106
57	Plasma glial fibrillary acidic protein is raised in progranulin-associated frontotemporal dementia. Journal of Neurology, Neurosurgery and Psychiatry, 2020, 91, 263-270.	1.9	106
58	Î±-Synuclein Amyloids Hijack Prion Protein to Gain Cell Entry, Facilitate Cell-to-Cell Spreading and Block Prion Replication. Scientific Reports, 2017, 7, 10050.	3.3	105
59	Deep Learning Representation from Electroencephalography of Early-Stage Creutzfeldt-Jakob Disease and Features for Differentiation from Rapidly Progressive Dementia. International Journal of Neural Systems, 2017, 27, 1650039.	5.2	104
60	Activation of microglial cells by PrP and Î²-amyloid fragments raises intracellular calcium through L-type voltage sensitive calcium channels. Brain Research, 1999, 818, 168-170.	2.2	101
61	Molecular determinants of the physicochemical properties of a critical prion protein region comprising residues 106â€“126. Biochemical Journal, 1999, 342, 207-214.	3.7	100
62	Mutant Prion Protein Expression Causes Motor and Memory Deficits and Abnormal Sleep Patterns in a Transgenic Mouse Model. Neuron, 2008, 60, 598-609.	8.1	97
63	Potential genetic modifiers of disease risk and age at onset in patients with frontotemporal lobar degeneration and GRN mutations: a genome-wide association study. Lancet Neurology, The, 2018, 17, 548-558.	10.2	97
64	The Efficacy of Tetracyclines in Peripheral and Intracerebral Prion Infection. PLoS ONE, 2008, 3, e1888.	2.5	94
65	A soluble form of prion protein in human cerebrospinal fluid: Implications for prion-related encephalopathies. Biochemical and Biophysical Research Communications, 1992, 184, 1398-1404.	2.1	90
66	Apoptosis-mediated neurotoxicity induced by Î²-amyloid and PRP fragments. Molecular and Chemical Neuropathology, 1996, 28, 163-171.	1.0	90
67	Cerebral preamyloid deposits and congophilic angiopathy in aged dogs. Neuroscience Letters, 1990, 114, 178-183.	2.1	89
68	A new function of microtubule-associated protein tau: Involvement in chromosome stability. Cell Cycle, 2008, 7, 1788-1794.	2.6	89
69	Anti-AÎ² autoantibodies in the CSF of a patient with CAA-related inflammation: A case report. Neurology, 2011, 76, 842-844.	1.1	88
70	Optimal Plasma Progranulin Cutoff Value for Predicting Null Progranulin Mutations in Neurodegenerative Diseases: A Multicenter Italian Study. Neurodegenerative Diseases, 2012, 9, 121-127.	1.4	88
71	Alzheimer patients and Down patients: Cerebral preamyloid deposits differ ultrastructurally and histochemically from the amyloid of senile plaques. Neuroscience Letters, 1989, 105, 294-299.	2.1	87
72	Hereditary Cerebral Hemorrhage With Amyloidosis Associated With the E693K Mutation of APP. Archives of Neurology, 2010, 67, 987-95.	4.5	87

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73	PRP27â€“30Is a Normal Soluble Prion Protein Fragment Released by Human Platelets. Biochemical and Biophysical Research Communications, 1996, 223, 572-577.	2.1	86
74	Inter-laboratory comparison of neuropathological assessments of Î²-amyloid protein: a study of the BrainNet Europe consortium. Acta Neuropathologica, 2008, 115, 533-546.	7.7	86
75	Prion protein preamyloid and amyloid deposits in Gerstmann-Straussler-Scheinker disease, Indiana kindred.. Proceedings of the National Academy of Sciences of the United States of America, 1992, 89, 9349-9353.	7.1	84
76	Prion Proteins with Different Conformations Accumulate in Gerstmann-StrÃussler-Scheinker Disease Caused by A117V and F198S Mutations. American Journal of Pathology, 2001, 158, 2201-2207.	3.8	83
77	The basal nucleus of Meynert in patients with progressive supranuclear palsy. Neuroscience Letters, 1984, 44, 37-42.	2.1	82
78	NEURONAL COUNTS IN BASAL NUCLEUS OF MEYNERT IN ALZHEIMER DISEASE AND IN SIMPLE SENILE DEMENTIA. Lancet, The, 1983, 321, 469-470.	13.7	81
79	Tau protein directly interacts with the amyloid Î²-protein precursor: Implications for Alzheimer's disease. Nature Medicine, 1995, 1, 365-369.	30.7	81
80	Iatrogenic Creutzfeldt-Jakob disease with Amyloid-Î² pathology: an international study. Acta Neuropathologica Communications, 2018, 6, 5.	5.2	79
81	Chronic wasting disease and atypical forms of bovine spongiform encephalopathy and scrapie are not transmissible to mice expressing wild-type levels of human prion protein. Journal of General Virology, 2012, 93, 1624-1629.	2.9	78
82	Intracellular Calcium Rise through L-Type Calcium Channels, as Molecular Mechanism for Prion Protein Fragment 106-126-Induced Astroglial Proliferation. Biochemical and Biophysical Research Communications, 1996, 228, 397-405.	2.1	76
83	A New Face for Old Antibiotics: Tetracyclines in Treatment of Amyloidoses. Journal of Medicinal Chemistry, 2013, 56, 5987-6006.	6.4	76
84	Structural Properties of Gerstmann-StrÃussler-Scheinker Disease Amyloid Protein. Journal of Biological Chemistry, 2003, 278, 48146-48153.	3.4	75
85	Intraspecies Transmission of BASE Induces Clinical Dullness and Amyotrophic Changes. PLoS Pathogens, 2008, 4, e1000075.	4.7	75
86	Prion protein fragment 106-126 induces apoptotic cell death and impairment of L-type voltage-sensitive calcium channel activity in the GH3 cell line. , 1998, 54, 341-352.		73
87	Neurofibrillary tangles of the Indiana kindred of Gerstmann-StraÂ¨ussler-Scheinker disease share antigenic determinants with those of Alzheimer disease. Brain Research, 1990, 530, 325-329.	2.2	71
88	Familial Gerstmann-StrÃussler-Scheinker disease with neurofibrillary tangles. Molecular Neurobiology, 1994, 8, 41-48.	4.0	71
89	Oxidative Damage to Nucleic Acids in Human Prion Disease. Neurobiology of Disease, 2002, 9, 275-281.	4.4	68
90	Molecular subtypes of Alzheimerâ€™s disease. Scientific Reports, 2018, 8, 3269.	3.3	68

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91	Clinical and neuropathological phenotype associated with the novel V189I mutation in the prion protein gene. Acta Neuropathologica Communications, 2019, 7, 1.	5.2	68
92	Hereditary prion protein amyloidoses. Clinics in Laboratory Medicine, 2003, 23, 65-85.	1.4	66
93	A family with Alzheimer disease and strokes associated with A713T mutation of the APP gene. Neurology, 2004, 63, 910-912.	1.1	66
94	Defined Î±-synuclein prion-like molecular assemblies spreading in cell culture. BMC Neuroscience, 2014, 15, 69.	1.9	66
95	CXCR4 involvement in neurodegenerative diseases. Translational Psychiatry, 2018, 8, 73.	4.8	66
96	Apoptotic Cell Death and Impairment of L-Type Voltage-Sensitive Calcium Channel Activity in Rat Cerebellar Granule Cells Treated with the Prion Protein Fragment 106â€“126. Neurobiology of Disease, 2000, 7, 299-309.	4.4	64
97	Alzheimer's disease amyloid precursor protein is present in senile plaques and cerebrospinal fluid: Immunohistochemical and biochemical characterization. Biochemical and Biophysical Research Communications, 1989, 163, 430-437.	2.1	63
98	White matter hyperintensities are seen only in GRN mutation carriers in the GENFI cohort. NeuroImage: Clinical, 2017, 15, 171-180.	2.7	63
99	A novel PSEN2 mutation associated with a peculiar phenotype. Neurology, 2008, 70, 1549-1554.	1.1	62
100	MM2â€Thalamic Creutzfeldtâ€“Jakob Disease: Neuropathological, Biochemical and Transmission Studies Identify a Distinctive Prion Strain. Brain Pathology, 2012, 22, 662-669.	4.1	62
101	Neuropathology of the recessive A673V APP mutation: Alzheimer disease with distinctive features. Acta Neuropathologica, 2010, 120, 803-812.	7.7	61
102	Transgenic Fatal Familial Insomnia Mice Indicate Prion Infectivity-Independent Mechanisms of Pathogenesis and Phenotypic Expression of Disease. PLoS Pathogens, 2015, 11, e1004796.	4.7	61
103	Spontaneous ARIA-like Events in Cerebral Amyloid Angiopathyâ€“Related Inflammation. Neurology, 2021, 97, e1809-e1822.	1.1	61
104	A Neurotoxic and Gliotrophic Fragment of the Prion Protein Increases Plasma Membrane Microviscosity. Neurobiology of Disease, 1997, 4, 47-57.	4.4	60
105	The Stimulation of Inducible Nitric-oxide Synthase by the Prion Protein Fragment 106â€“126 in Human Microglia Is Tumor Necrosis Factor-Î±-dependent and Involves p38 Mitogen-activated Protein Kinase. Journal of Biological Chemistry, 2001, 276, 25692-25696.	3.4	60
106	p38 MAP Kinase Mediates the Cell Death Induced by PrP106â€“126 in the SH-SY5Y Neuroblastoma Cells. Neurobiology of Disease, 2002, 9, 69-81.	4.4	59
107	Diagnostic differentiation of mild cognitive impairment due to Alzheimer's disease using a hippocampus-dependent test of spatial memory. Hippocampus, 2015, 25, 939-951.	1.9	59
108	Neuronal loss in the basal nucleus of meynert in progressive supranuclear palsy. Acta Neuropathologica, 1983, 61, 157-160.	7.7	58

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109	Alzheimer patients: preamyloid deposits are more widely distributed than senile plaques throughout the central nervous system. Neuroscience Letters, 1989, 103, 263-268.	2.1	58
110	An APOE Haplotype Associated with Decreased Îµ4 Expression Increases the Risk of Late Onset Alzheimer's Disease. Journal of Alzheimer's Disease, 2011, 24, 235-245.	2.6	58
111	Differential overexpression of SERPINA3 in human prion diseases. Scientific Reports, 2017, 7, 15637.	3.3	58
112	Proteinase-K-Resistant Prion Protein Isoforms in Gerstmann-StrÃussler-Scheinker Disease (Indiana) Tj ETQq0 0 0 rgBT /Overlock 10 Tf 50	1.7	57
113	Prion deposition in olfactory biopsy of sporadic Creutzfeldt-Jakob disease. Annals of Neurology, 2004, 55, 294-296.	5.3	57
114	Intracellular mechanisms mediating the neuronal death and astrogliosis induced by the prion protein fragment 106â€“126. International Journal of Developmental Neuroscience, 2000, 18, 481-492.	1.6	56
115	Conformational Plasticity of the Gerstmannâ€“StrÃusslerâ€“Scheinker Disease Peptide as Indicated by Its Multiple Aggregation Pathways. Journal of Molecular Biology, 2008, 381, 1349-1361.	4.2	56
116	Pathogenic Huntingtin Repeat Expansions in Patients with Frontotemporal Dementia and Amyotrophic Lateral Sclerosis. Neuron, 2021, 109, 448-460.e4.	8.1	56
117	Alzheimer patients and Down patients: Abnormal presynaptic terminals are related to cerebral preamyloid deposits. Neuroscience Letters, 1990, 119, 56-59.	2.1	55
118	Cognitive reserve and TMEM106B genotype modulate brain damage in presymptomatic frontotemporal dementia: a GENFI study. Brain, 2017, 140, 1784-1791.	7.6	55
119	Neuronal pentraxin 2: a synapse-derived CSF biomarker in genetic frontotemporal dementia. Journal of Neurology, Neurosurgery and Psychiatry, 2020, 91, 612-621.	1.9	55
120	A68 is a component of paired helical filaments of Gerstmann-StrÃussler-Scheinker disease, Indiana kindred. Brain Research, 1993, 616, 325-329.	2.2	54
121	Amyloid in alzheimer's disease and prion-related encephalopathies: Studies with synthetic peptides. Progress in Neurobiology, 1996, 49, 287-315.	5.7	54
122	The Peculiar Role of the A2V Mutation in Amyloid-Î² (AÎ²) 1â€“42 Molecular Assembly. Journal of Biological Chemistry, 2014, 289, 24143-24152.	3.4	54
123	Preventive study in subjects at risk of fatal familial insomnia: Innovative approach to rare diseases. Prion, 2015, 9, 75-79.	1.8	54
124	Neuropathology of Gerstmann-Strï¿½ussler-Scheinker disease. Microscopy Research and Technique, 2000, 50, 10-15.	2.2	53
125	Channels formed with a mutant prion protein PrP(82-146) homologous to a 7-kDa fragment in diseased brain of GSS patients. American Journal of Physiology - Cell Physiology, 2003, 285, C862-C872.	4.6	53
126	Redox metals and oxidative abnormalities in human prion diseases. Acta Neuropathologica, 2005, 110, 232-238.	7.7	52

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127	Tetracycline prevents AÎ² oligomer toxicity through an atypical supramolecular interaction. Organic and Biomolecular Chemistry, 2011, 9, 463-472.	2.8	52
128	Specific Recognition of Biologically Active Amyloid-Î² Oligomers by a New Surface Plasmon Resonance-based Immunoassay and an in Vivo Assay in Caenorhabditis elegans. Journal of Biological Chemistry, 2012, 287, 27796-27805.	3.4	52
129	Plasma Neurofilament Light for Prediction of Disease Progression in Familial Frontotemporal Lobar Degeneration. Neurology, 2021, 96, e2296-e2312.	1.1	52
130	Tauopathy in human and experimental variant Creutzfeldt-Jakob disease. Neurobiology of Aging, 2008, 29, 1864-1873.	3.1	51
131	Fatal familial insomnia. Neurology, 1998, 50, 688-692.	1.1	50
132	Interâ€Laboratory Assessment of PrP^{Sc} Typing in Creutzfeldtâ€“Jakob Disease: A Western Blot Study within the NeuroPrion Consortium. Brain Pathology, 2009, 19, 384-391.	4.1	50
133	Recurrent generalized seizures, visual loss, and palinopsia as phenotypic features of neuronal ceroid lipofuscinosis due to progranulin gene mutation. Epilepsia, 2014, 55, e56-9.	5.1	50
134	ÃŸPP Participates in PrP-Amyloid Plaques of Gerstmann-StrÃussler-Scheinker Disease, Indiana Kindred. Journal of Neuropathology and Experimental Neurology, 1993, 52, 64-70.	1.7	49
135	Activation effects of a prion protein fragment [PrP-(106-126)] on human leucocytes. Biochemical Journal, 1996, 320, 563-570.	3.7	49
136	Creutzfeldtâ€“Jakob disease with a novel four extra-repeat insertional mutation in the PrP gene. Neurology, 2000, 55, 405-410.	1.1	49
137	Creutzfeldtâ€Jakob Disease: Carnoy's Fixative Improves the Immunohistochemistry of the Proteinase Kâ€• Resistant Prion Protein. Brain Pathology, 2000, 10, 31-37.	4.1	49
138	Tetracyclines and Prion Infectivity. Infectious Disorders - Drug Targets, 2009, 9, 23-30.	0.8	48
139	A genome-wide screening and SNPs-to-genes approach to identify novel genetic risk factors associated with frontotemporal dementia. Neurobiology of Aging, 2015, 36, 2904.e13-2904.e26.	3.1	48
140	Parenchymal preamyloid and amyloid deposits in the brains of patients with hereditary cerebral hemorrhage with amyloidosisâ€”Dutch type. Neuroscience Letters, 1990, 118, 223-226.	2.1	47
141	Ectopic White Matter Neurons, a Developmental Abnormality That May Be Caused by thePSEN1S169L Mutation in a Case of Familial AD with Myoclonus and Seizures. Journal of Neuropathology and Experimental Neurology, 2001, 60, 1137-1152.	1.7	47
142	Loss of exosomes in progranulin-associated frontotemporal dementia. Neurobiology of Aging, 2016, 40, 41-49.	3.1	47
143	Functional network resilience to pathology in presymptomatic genetic frontotemporal dementia. Neurobiology of Aging, 2019, 77, 169-177.	3.1	47
144	Mutations in MAPT Gene Cause Chromosome Instability and Introduce Copy Number Variations Widely in the Genome. Journal of Alzheimer's Disease, 2013, 33, 969-982.	2.6	45

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145	Multicentre, cross-cultural, population-based, caseâ€“control study of physical activity as risk factor for amyotrophic lateral sclerosis. Journal of Neurology, Neurosurgery and Psychiatry, 2018, 89, 797-803.	1.9	45
146	Neurotoxicity of the Putative Transmembrane Domain of the Prion Protein. Neurobiology of Disease, 2000, 7, 644-656.	4.4	43
147	The G389R mutation in the MAPT gene presenting as sporadic corticobasal syndrome. Movement Disorders, 2008, 23, 892-895.	3.9	43
148	C9ORF72 Hexanucleotide Repeat Number in Frontotemporal Lobar Degeneration: A Genotype-Phenotype Correlation Study. Journal of Alzheimer's Disease, 2013, 38, 799-808.	2.6	43
149	Studies on peptide fragments of prion proteins. Advances in Protein Chemistry, 2001, 57, 171-201.	4.4	42
150	Mutant Presenilin 1 Increases the Expression and Activity of BACE1. Journal of Biological Chemistry, 2009, 284, 9027-9038.	3.4	42
151	The basal nucleus of Meynert in idiopathic Parkinson's disease. Acta Neurologica Scandinavica, 1984, 70, 20-28.	2.1	42
152	Progression of Behavioral Disturbances and Neuropsychiatric Symptoms in Patients With Genetic Frontotemporal Dementia. JAMA Network Open, 2021, 4, e2030194.	5.9	42
153	Therapy in Prion Diseases. Current Topics in Medicinal Chemistry, 2013, 13, 2465-2476.	2.1	41
154	The Semantic Variant of Primary Progressive Aphasia: Clinical and Neuroimaging Evidence in Single Subjects. PLoS ONE, 2015, 10, e0120197.	2.5	41
155	Detection of prion seeding activity in the olfactory mucosa of patients with Fatal Familial Insomnia. Scientific Reports, 2017, 7, 46269.	3.3	41
156	Comparison of arterial spin labeling registration strategies in the multiâ€center GENetic frontotemporal dementia initiative (GENFI). Journal of Magnetic Resonance Imaging, 2018, 47, 131-140.	3.4	41
157	Cerebral perfusion changes in presymptomatic genetic frontotemporal dementia: a GENFI study. Brain, 2019, 142, 1108-1120.	7.6	41
158	Progranulin plasma levels predict the presence of GRN mutations in asymptomatic subjects and do not correlate with brain atrophy: results from the GENFI study. Neurobiology of Aging, 2018, 62, 245.e9-245.e12.	3.1	40
159	Neuropathological and Clinical Phenotype of an Italian Alzheimer Family with M239V Mutation of Presenilin 2 Gene. Journal of Neuropathology and Experimental Neurology, 2004, 63, 199-209.	1.7	39
160	Periodic electroencephalogram complexes in a patient with variant Creutzfeldtâ€“Jakob disease. Annals of Neurology, 2006, 59, 423-427.	5.3	39
161	A Novel Italian Presenilin 2 Gene Mutation with Prevalent Behavioral Phenotype. Journal of Alzheimer's Disease, 2009, 16, 509-511.	2.6	39
162	Frontotemporal lobar degeneration: old knowledge and new insight into the pathogenetic mechanisms of tau mutations. Frontiers in Aging Neuroscience, 2015, 7, 192.	3.4	39

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