Stuart M Pickering-Brown

List of Publications by Year
in descending order

165 papers	25,885 citations	17440 63 h-index	6996 154 g-index
167 all docs	167 docs citations	167 times ranked	19799 citing authors

#	Article	IF	Citations
1	Genetic evaluation of dementia with Lewy bodies implicates distinct disease subgroups. Brain, 2022, 145, 1757-1762.	7.6	17
2	Prion-like Î±-synuclein pathology in the brain of infants with Krabbe disease. Brain, 2022, 145, 1257-1263.	7.6	9
3	C9orf72 dipeptides disrupt the nucleocytoplasmic transport machinery and cause TDP-43 mislocalisation to the cytoplasm. Scientific Reports, 2022, 12, 4799.	3.3	19
4	Pathogenic Huntingtin Repeat Expansions in Patients with Frontotemporal Dementia and Amyotrophic Lateral Sclerosis. Neuron, 2021, 109, 448-460.e4.	8.1	56
5	Amyloid-PETâ€"Positive Patient With bvFTD. Neurology: Clinical Practice, 2021, 11, e952-e955.	1.6	4
6	Genome sequencing analysis identifies new loci associated with Lewy body dementia and provides insights into its genetic architecture. Nature Genetics, 2021, 53, 294-303.	21.4	198
7	Gene Expression Imputation Across Multiple Tissue Types Provides Insight Into the Genetic Architecture of Frontotemporal Dementia and Its Clinical Subtypes. Biological Psychiatry, 2021, 89, 825-835.	1.3	10
8	Association of Variants in the SPTLC1 Gene With Juvenile Amyotrophic Lateral Sclerosis. JAMA Neurology, 2021, 78, 1236.	9.0	46
9	Age at symptom onset and death and disease duration in genetic frontotemporal dementia: an international retrospective cohort study. Lancet Neurology, The, 2020, 19, 145-156.	10.2	175
10	Cognition and behaviour in frontotemporal dementia with and without amyotrophic lateral sclerosis. Journal of Neurology, Neurosurgery and Psychiatry, 2020, 91, 1304-1311.	1.9	15
11	Mendelian randomization implies no direct causal association between leukocyte telomere length and amyotrophic lateral sclerosis. Scientific Reports, 2020, 10, 12184.	3.3	4
12	C9orf72, age at onset, and ancestry help discriminate behavioral from language variants in FTLD cohorts. Neurology, 2020, 95, e3288-e3302.	1.1	7
13	Co-expression of C9orf72 related dipeptide-repeats over 1000 repeat units reveals age- and combination-specific phenotypic profiles in Drosophila. Acta Neuropathologica Communications, 2020, 8, 158.	5.2	23
14	Extracellular Vesicles Isolated from Human Induced Pluripotent Stem Cell-Derived Neurons Contain a Transcriptional Network. Neurochemical Research, 2020, 45, 1711-1728.	3.3	11
15	The cellular expression and proteolytic processing of the amyloid precursor protein is independent of TDP-43. Bioscience Reports, 2020, 40, .	2.4	5
16	Analysis of neurodegenerative disease-causing genes in dementia with Lewy bodies. Acta Neuropathologica Communications, 2020, 8, 5.	5.2	27
17	CRISPR/Cas9 does not facilitate stable expression of long C9orf72 dipeptides in mice. Neurobiology of Aging, 2019, 84, 235.e1-235.e8.	3.1	3
18	Heritability and genetic variance of dementia with Lewy bodies. Neurobiology of Disease, 2019, 127, 492-501.	4.4	29

#	Article	IF	Citations
19	Genome-wide analyses as part of the international FTLD-TDP whole-genome sequencing consortium reveals novel disease risk factors and increases support for immune dysfunction in FTLD. Acta Neuropathologica, 2019, 137, 879-899.	7.7	90
20	Genetic meta-analysis of diagnosed Alzheimerâ€™s disease identifies new risk loci and implicates AÎ², tau, immunity and lipid processing. Nature Genetics, 2019, 51, 414-430.	21.4	1,962
21	A comprehensive screening of copy number variability in dementia with Lewy bodies. Neurobiology of Aging, 2019, 75, 223.e1-223.e10.	3.1	13
22	Oligogenic genetic variation of neurodegenerative disease genes in 980 postmortem human brains. Journal of Neurology, Neurosurgery and Psychiatry, 2018, 89, 813-816.	1.9	17
23	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
24	Mendelian adult-onset leukodystrophy genes in Alzheimer's disease: critical influence of CSF1R and NOTCH3. Neurobiology of Aging, 2018, 66, 179.e17-179.e29.	3.1	32
25	Genome-wide Analyses Identify KIF5A as a Novel ALS Gene. Neuron, 2018, 97, 1268-1283.e6.	8.1	517
26	Expression of C9orf72-related dipeptides impairs motor function in a vertebrate model. Human Molecular Genetics, 2018, 27, 1754-1762.	2.9	44
27	Investigating the genetic architecture of dementia with Lewy bodies: a two-stage genome-wide association study. Lancet Neurology, The, 2018, 17, 64-74.	10.2	195
28	Immunohistochemical detection of C9orf72 protein in frontotemporal lobar degeneration and motor neurone disease: patterns of immunostaining and an evaluation of commercial antibodies. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2018, 19, 102-111.	1.7	10
29	LRP10 in Î±-synucleinopathies. Lancet Neurology, The, 2018, 17, 1032-1033.	10.2	11
30	A C6orf10/LOC101929163 locus is associated with age of onset in C9orf72 carriers. Brain, 2018, 141, 2895-2907.	7.6	39
31	Mitochondrial DNA point mutations and relative copy number in 1363 disease and control human brains. Acta Neuropathologica Communications, 2017, 5, 13.	5.2	83
32	Genetic compendium of 1511 human brains available through the UK Medical Research Council Brain Banks Network Resource. Genome Research, 2017, 27, 165-173.	5.5	44
33	Heterogeneous ribonuclear protein E2 (hnRNP E2) is associated with TDP-43-immunoreactive neurites in Semantic Dementia but not with other TDP-43 pathological subtypes of Frontotemporal Lobar Degeneration. Acta Neuropathologica Communications, 2017, 5, 54.	5.2	15
34	Heterogeneous ribonuclear protein A3 (hnRNP A3) is present in dipeptide repeat protein containing inclusions in Frontotemporal Lobar Degeneration and Motor Neurone disease associated with expansions in C9orf72 gene. Acta Neuropathologica Communications, 2017, 5, 31.	5.2	20
35	Analysis of C9orf72 repeat expansions in a large international cohort of dementia with Lewy bodies. Neurobiology of Aging, 2017, 49, 214.e13-214.e15.	3.1	12
36	Mutation analysis of sporadic early-onset Alzheimer's disease using the NeuroX array. Neurobiology of Aging, 2017, 49, 215.e1-215.e8.	3.1	21

#	Article	IF	Citations
37	Identification of biological pathways regulated by PGRN and GRN peptide treatments using transcriptome analysis. European Journal of Neuroscience, 2016, 44, 2214-2225.	2.6	13
38	Genetic risk factors for the posterior cortical atrophy variant of Alzheimer's disease. Alzheimer's and Dementia, 2016, 12, 862-871.	0.8	93
39	Pathological tau deposition in Motor Neurone Disease and frontotemporal lobar degeneration associated with TDP-43 proteinopathy. Acta Neuropathologica Communications, 2016, 4, 33.	5.2	33
40	Modelling C9orf72 dipeptide repeat proteins of a physiologically relevant size. Human Molecular Genetics, 2016, 25, ddw327.	2.9	25
41	Genome-wide analysis of genetic correlation in dementia with Lewy bodies, Parkinson's and Alzheimer's diseases. Neurobiology of Aging, 2016, 38, 214.e7-214.e10.	3.1	78
42	Left hand dystonia as a recurring feature of a family carrying C9ORF72 mutation. Journal of Neurology, Neurosurgery and Psychiatry, 2016, 87, 793-795.	1.9	3
43	Psychosis associated with expansions in theC9orf72gene: the influence of a 10 base pair gene deletion: TableÂ1. Journal of Neurology, Neurosurgery and Psychiatry, 2016, 87, 562-563.	1.9	10
44	A novel Alzheimer disease locus located near the gene encoding tau protein. Molecular Psychiatry, 2016, 21, 108-117.	7.9	260
45	Accumulation of dipeptide repeat proteins predates that of <scp>TDP</scp>â€43 in frontotemporal lobar degeneration associated with hexanucleotide repeat expansions in <scp>C9ORF72</scp> gene. Neuropathology and Applied Neurobiology, 2015, 41, 601-612.	3.2	62
46	Histone deacetylases (<scp>HDACs</scp>) in frontotemporal lobar degeneration. Neuropathology and Applied Neurobiology, 2015, 41, 245-257.	3.2	11
47	Semantic Corticobasal Dementia. Alzheimer Disease and Associated Disorders, 2015, 29, 360-363.	1.3	1
48	A small deletion in C9orf72 hides a proportion of expansion carriers in FTLD. Neurobiology of Aging, 2015, 36, 1601.e1-1601.e5.	3.1	19
49	Plasma levels of progranulin and interleukin-6 in frontotemporal lobar degeneration. Neurobiology of Aging, 2015, 36, 1603.e1-1603.e4.	3.1	29
50	IsSIGMAR1a confirmed FTD/MND gene?. Brain, 2015, 138, e393-e393.	7.6	3
51	p62/SQSTM1 analysis in frontotemporal lobar degeneration. Neurobiology of Aging, 2015, 36, 1603.e5-1603.e9.	3.1	11
52	Antisense RNA foci in the motor neurons of C9ORF72-ALS patients are associated with TDP-43 proteinopathy. Acta Neuropathologica, 2015, 130, 63-75.	7.7	149
53	Distinct clinical and pathological phenotypes in frontotemporal dementia associated with MAPT, PGRN and C9orf72 mutations. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2015, 16, 497-505.	1.7	75
54	A UBQLN2 variant of unknown significance in frontotemporal lobar degeneration. Neurobiology of Aging, 2015, 36, 546.e15-546.e16.	3.1	13

#	Article	IF	Citations
55	TREM2 analysis and increased risk of Alzheimer's disease. Neurobiology of Aging, 2015, 36, 546.e9-546.e13.	3.1	37
56	Genetic analysis implicates APOE, SNCA and suggests lysosomal dysfunction in the etiology of dementia with Lewy bodies. Human Molecular Genetics, 2014, 23, 6139-6146.	2.9	178
57	Genomewide association study in cervical dystonia demonstrates possible association with sodium leak channel. Movement Disorders, 2014, 29, 245-251.	3.9	43
58	Brain distribution of dipeptide repeat proteins in frontotemporal lobar degeneration and motor neurone disease associated with expansions in C9ORF72. Acta Neuropathologica Communications, 2014, 2, 70.	5.2	103
59	No interaction between tau and <scp>TDP</scp>â€43 pathologies in either frontotemporal lobar degeneration or motor neurone disease. Neuropathology and Applied Neurobiology, 2014, 40, 844-854.	3.2	23
60	Rare coding variants in the phospholipase D3 gene confer risk for Alzheimerâ€™s disease. Nature, 2014, 505, 550-554.	27.8	425
61	C9orf72 repeat expansions cause neurodegeneration in Drosophila through arginine-rich proteins. Science, 2014, 345, 1192-1194.	12.6	632
62	Patterns of microglial cell activation in frontotemporal lobar degeneration. Neuropathology and Applied Neurobiology, 2014, 40, 686-696.	3.2	70
63	Frontotemporal dementia and its subtypes: a genome-wide association study. Lancet Neurology, The, 2014, 13, 686-699.	10.2	302
64	Pathogenesis/genetics of frontotemporal dementia and how it relates to ALS. Experimental Neurology, 2014, 262, 84-90.	4.1	78
65	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. Acta Neuropathologica Communications, 2013, 1, 68.	5.2	162
66	Frontotemporal dementia with amyotrophic lateral sclerosis: A clinical comparison of patients with and without repeat expansions in C9orf72. Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, 2013, 14, 172-176.	1.7	58
67	A Multicenter Study of Glucocerebrosidase Mutations in Dementia With Lewy Bodies. JAMA Neurology, 2013, 70, 727.	9.0	374
68	Distinct clinical and pathological characteristics of frontotemporal dementia associated with C9ORF72 mutations. Brain, 2012, 135, 693-708.	7.6	486
69	Prominent Sensorimotor Neuropathy Due to SACS Mutations Revealed by Whole-Exome Sequencing. Archives of Neurology, 2012, 69, 1351-4.	4.5	21
70	Analysis of optineurin in frontotemporal lobar degeneration. Neurobiology of Aging, 2012, 33, 425.e1-425.e2.	3.1	13
71	The chromosome 9 ALS and FTD locus is probably derived from a single founder. Neurobiology of Aging, 2012, 33, 209.e3-209.e8.	3.1	115
72	Analysis of the hexanucleotide repeat in C9ORF72 in Alzheimer's disease. Neurobiology of Aging, 2012, 33, 1846.e5-1846.e6.	3.1	38

#	Article	IF	Citations
73	Psychosis,C9ORF72and dementia with Lewy bodies: Table 1. Journal of Neurology, Neurosurgery and Psychiatry, 2012, 83, 1031-1032.	1.9	45
74	Semantic dementia associated with corticobasal syndrome: a further variant of frontotemporal lobe degeneration?. Journal of Neurology, 2012, 259, 1478-1480.	3.6	5
75	Frequency of the C9orf72 hexanucleotide repeat expansion in patients with amyotrophic lateral sclerosis and frontotemporal dementia: a cross-sectional study. Lancet Neurology, The, 2012, 11, 323-330.	10.2	1,039
76	Next generation sequencing of CLU, PICALM and CR1: pitfalls and potential solutions. International Journal of Molecular Epidemiology and Genetics, 2012, 3, 262-75.	0.4	2
77	Identification of common variants influencing risk of the tauopathy progressive supranuclear palsy. Nature Genetics, 2011, 43, 699-705.	21.4	502
78	No association of PGRN 3â€²UTR rs5848 in frontotemporal lobar degeneration. Neurobiology of Aging, 2011, 32, 754-755.	3.1	42
79	Frontotemporal lobar degeneration genome wide association study replication confirms a risk locus shared with amyotrophic lateral sclerosis. Neurobiology of Aging, 2011, 32, 758.e1-758.e7.	3.1	32
80	A Hexanucleotide Repeat Expansion in C9ORF72 Is the Cause of Chromosome 9p21-Linked ALS-FTD. Neuron, 2011, 72, 257-268.	8.1	3,833
81	UBAP1 Is a Component of an Endosome-Specific ESCRT-I Complex that Is Essential for MVB Sorting. Current Biology, 2011, 21, 1245-1250.	3.9	106
82	Pathological correlates of frontotemporal lobar degeneration in the elderly. Acta Neuropathologica, 2011, 121, 365-371.	7.7	70
83	The most common type of FTLD-FUS (aFTLD-U) is associated with a distinct clinical form of frontotemporal dementia but is not related to mutations in the FUS gene. Acta Neuropathologica, 2011, 122, 99-110.	7.7	108
84	TDP-43 pathological changes in early onset familial and sporadic Alzheimerâ€™s disease, late onset Alzheimerâ€™s disease and Downâ€™s Syndrome: association with age, hippocampal sclerosis and clinical phenotype. Acta Neuropathologica, 2011, 122, 703-713.	7.7	128
85	Genetic and Clinical Features of Progranulin-Associated Frontotemporal Lobar Degeneration. Archives of Neurology, 2011, 68, 488.	4.5	108
86	FUS pathology defines the majority of tau- and TDP-43-negative frontotemporal lobar degeneration. Acta Neuropathologica, 2010, 120, 33-41.	7.7	222
87	Phosphorylated TDP-43 pathology and hippocampal sclerosis in progressive supranuclear palsy. Acta Neuropathologica, 2010, 120, 55-66.	7.7	97
88	Effect of topographical distribution of Î±-synuclein pathology on TDP-43 accumulation in Lewy body disease. Acta Neuropathologica, 2010, 120, 789-801.	7.7	31
89	Review: Recent progress in frontotemporal lobar degeneration. Neuropathology and Applied Neurobiology, 2010, 36, 4-16.	3.2	9
90	Common variants at 7p21 are associated with frontotemporal lobar degeneration with TDP-43 inclusions. Nature Genetics, 2010, 42, 234-239.	21.4	479

#	Article	IF	Citations
91	Recent origin and spread of a common Welsh MAPT splice mutation causing frontotemporal lobar degeneration. Neurogenetics, 2009, 10, 313-318.	1.4	10
92	TDP-43 in ubiquitinated inclusions in the inferior olives in frontotemporal lobar degeneration and in other neurodegenerative diseases: a degenerative process distinct from normal ageing. Acta Neuropathologica, 2009, 118, 359-369.	7.7	30
93	Prominent phenotypic variability associated with mutations in Progranulin. Neurobiology of Aging, 2009, 30, 739-751.	3.1	166
94	Ubiquitin associated protein 1 is a risk factor for frontotemporal lobar degeneration. Neurobiology of Aging, 2009, 30, 656-665.	3.1	33
95	TDP-43 protein in plasma may index TDP-43 brain pathology in Alzheimerâ€™s disease and frontotemporal lobar degeneration. Acta Neuropathologica, 2008, 116, 141-146.	7.7	142
96	Progressive Anomia Revisited: Focal Degeneration Associated with Progranulin Gene Mutation. Neurocase, 2008, 13, 366-377.	0.6	17
97	The genetics of frontotemporal dementia. Handbook of Clinical Neurology / Edited By P J Vinken and G W Bruyn, 2008, 89, 383-392.	1.8	3
98	Frequency and clinical characteristics of progranulin mutation carriers in the Manchester frontotemporal lobar degeneration cohort: comparison with patients with MAPT and no known mutations. Brain, 2008, 131, 721-731.	7.6	178
99	Parietal Lobe Deficits in Frontotemporal Lobar Degeneration Caused by a Mutation in the Progranulin Gene. Archives of Neurology, 2008, 65, 506.	4.5	52
100	Progranulin Mediates Caspase-Dependent Cleavage of TAR DNA Binding Protein-43. Journal of Neuroscience, 2007, 27, 10530-10534.	3.6	339
101	Familial Early-Onset Dementia With Tau Intron 10Â+Â16 Mutation With Clinical Features Similar to Those of Alzheimer Disease. Archives of Neurology, 2007, 64, 1535.	4.5	35
102	Apolipoprotein E Îµ4 Allele Frequency and Age at Onset of Alzheimerâ€™s Disease. Dementia and Geriatric Cognitive Disorders, 2007, 23, 60-66.	1.5	56
103	The genetics of frontotemporal lobar degeneration. Current Opinion in Neurology, 2007, 20, 693-698.	3.6	8
104	The complex aetiology of frontotemporal lobar degeneration. Experimental Neurology, 2007, 206, 1-10.	4.1	28
105	TDP-43 gene analysis in frontotemporal lobar degeneration. Neuroscience Letters, 2007, 419, 1-4.	2.1	47
106	Phenotypic variability associated with progranulin haploinsufficiency in patients with the common 1477Câ†’T (Arg493X) mutation: an international initiative. Lancet Neurology, The, 2007, 6, 857-868.	10.2	199
107	Ubiquitinated pathological lesions in frontotemporal lobar degeneration contain the TAR DNA-binding protein, TDP-43. Acta Neuropathologica, 2007, 113, 521-533.	7.7	274
108	Progranulin and frontotemporal lobar degeneration. Acta Neuropathologica, 2007, 114, 39-47.	7.7	23

#	Article	IF	Citations
109	Frontotemporal dementia and parkinsonism linked to chromosome 17 (FTDP-17). Orphanet Journal of Rare Diseases, 2006, 1, 30.	2.7	99
110	CHMP2B mutations are not a common cause of frontotemporal lobar degeneration. Neuroscience Letters, 2006, 398, 83-84.	2.1	64
111	Comparison of extent of tau pathology in patients with frontotemporal dementia with Parkinsonism linked to chromosome 17 (FTDP-17), frontotemporal lobar degeneration with Pick bodies and early onset Alzheimer's disease. Neuropathology and Applied Neurobiology, 2006, 32, 374-387.	3.2	34
112	Mutations in progranulin cause tau-negative frontotemporal dementia linked to chromosome 17. Nature, 2006, 442, 916-919.	27.8	1,816
113	An immunohistochemical study of cases of sporadic and inherited frontotemporal lobar degeneration using 3R- and 4R-specific tau monoclonal antibodies. Acta Neuropathologica, 2006, 111, 329-340.	7.7	91
114	Heterogeneity of ubiquitin pathology in frontotemporal lobar degeneration: classification and relation to clinical phenotype. Acta Neuropathologica, 2006, 112, 539-549.	7.7	298
115	Analysis of IFT74as a candidate gene for chromosome 9p-linked ALS-FTD. BMC Neurology, 2006, 6, 44.	1.8	70
116	A family with tau-negative frontotemporal dementia and neuronal intranuclear inclusions linked to chromosome 17. Brain, 2006, 129, 853-867.	7.6	102
117	Frontotemporal dementia and parkinsonism associated with the IVS1+1G->A mutation in progranulin: a clinicopathologic study. Brain, 2006, 129, 3103-3114.	7.6	105
118	Progranulin gene mutations associated with frontotemporal dementia and progressive non-fluent aphasia. Brain, 2006, 129, 3091-3102.	7.6	185
119	The neuropathology of frontotemporal lobar degeneration caused by mutations in the progranulin gene. Brain, 2006, 129, 3081-3090.	7.6	291
120	Mutations in progranulin explain atypical phenotypes with variants in MAPT. Brain, 2006, 129, 3124-3126.	7.6	91
121	Mutations in progranulin are a major cause of ubiquitin-positive frontotemporal lobar degeneration. Human Molecular Genetics, 2006, 15, 2988-3001.	2.9	529
122	Frontotemporal dementia and the involvement of tau. , 2006, , 209-216.		0
123	Histopathological changes underlying frontotemporal lobar degeneration with clinicopathological correlation. Acta Neuropathologica, 2005, 110, 501-512.	7.7	131
124	The Effect of tau genotype on clinical features in FTDP-17. Parkinsonism and Related Disorders, 2005, 11, 205-208.	2.2	31
125	The Tau Gene Locus and Frontotemporal Dementia. Dementia and Geriatric Cognitive Disorders, 2004, 17, 258-260.	1.5	9
126	Î±-T-Catenin Is Expressed in Human Brain and Interacts With the Wnt Signaling Pathway But Is Not Responsible for Linkage to Chromosome 10 in Alzheimer's Disease. NeuroMolecular Medicine, 2004, 5, 133-146.	3.4	41

#	Article	IF	Citations
127	The neuropathology of frontotemporal lobar degeneration with respect to the cytological and biochemical characteristics of tau protein. Neuropathology and Applied Neurobiology, 2004, 30, 1-18.	3.2	72
128	Evidence of a founder effect in families with frontotemporal dementia that harbor the tau +16 splice mutation. American Journal of Medical Genetics Part A, 2004, 125B, 79-82.	2.4	24
129	A polymorphism in the angiotensin 1-converting enzyme gene is associated with damage to cerebral cortical white matter in Alzheimer's disease. Neuroscience Letters, 2004, 354, 103-106.	2.1	30
130	Frontotemporal dementia with Pickâ€type histology associated with Q336R mutation in the tau gene. Brain, 2004, 127, 1415-1426.	7.6	87
131	Identification of a truncated IL-18RÎ² mRNA: a putative regulator of IL-18 expressed in rat brain. Journal of Neuroimmunology, 2003, 145, 40-45.	2.3	36
132	Tau load is associated with apolipoproteinâ€ƒE genotype and the amount of amyloidâ€ƒÎ² protein, AÎ²40 , in sporadic and familial Alzheimer's disease. Neuropathology and Applied Neurobiology, 2003, 29, 35-44.	3.2	34
133	Tau haplotype frequency in frontotemporal lobar degeneration and amyotrophic lateral sclerosis. Experimental Neurology, 2003, 181, 12-16.	4.1	36
134	Inherited frontotemporal dementia in nine British families associated with intronic mutations in the tau gene. Brain, 2002, 125, 732-751.	7.6	116
135	A polymorphism within intron 11 of the tau gene is not increased in frequency in patients with sporadic Alzheimer's disease, nor does it influence the extent of tau pathology in the brain. Neuroscience Letters, 2002, 324, 113-116.	2.1	18
136	The angiotensin 1-converting enzyme insertion (I)/deletion (D) polymorphism does not influence the extent of amyloid or tau pathology in patients with sporadic Alzheimer's disease. Neuroscience Letters, 2002, 328, 314-318.	2.1	50
137	Pathological relationships between microglial cell activity and tau and amyloid Î² protein in patients with Alzheimer's disease. Neuroscience Letters, 2002, 331, 171-174.	2.1	51
138	Sporadic Pick's disease: A tauopathy characterized by a spectrum of pathological ? isoforms in gray and white matter. Annals of Neurology, 2002, 51, 730-739.	5.3	141
139	The Neuropathology of Frontotemporal Lobar Degeneration. Advances in Behavioral Biology, 2002, , 523-529.	0.2	0
140	Amyloid Angiopathy and Variability in Amyloid Î² Deposition Is Determined by Mutation Position in Presenilin-1-Linked Alzheimerâ€™s Disease. American Journal of Pathology, 2001, 158, 2165-2175.	3.8	170
141	The status of â€œPick's Diseaseâ€•and other tauopathies within the frontotemporal dementias. Neurobiology of Aging, 2001, 22, 109-111.	3.1	5
142	The extended haplotype of the microtubule associated protein tau gene is not associated with Pick's disease. Neuroscience Letters, 2001, 299, 156-158.	2.1	35
143	Amyloid Î² protein deposition in patients with frontotemporal lobar degeneration: relationship to age and apolipoprotein E genotype. Neuroscience Letters, 2001, 304, 161-164.	2.1	41
144	Clinical features of dementia associated with apolipoprotein ?4: discrimination with a neural network genetic algorithm. International Journal of Geriatric Psychiatry, 2001, 16, 77-81.	2.7	9

#	Article	IF	Citations
145	Pick's disease is associated with mutations in thetau gene. Annals of Neurology, 2000, 48, 859-867.	5.3	131
146	Apolipoprotein E Ïµ4 Allele Has No Effect on Age at Onset or Duration of Disease in Cases of Frontotemporal Dementia with Pick- or Microvacuolar-Type Histology. Experimental Neurology, 2000, 163, 452-456.	4.1	45
147	Molecular classification of the dementias. Lancet, The, 2000, 355, 626.	13.7	61
148	Detection of the interleukin 18 family in rat brain by RT-PCR. Molecular Brain Research, 2000, 77, 290-293.	2.3	54
149	Pick's disease is associated with mutations in the tau gene. Annals of Neurology, 2000, 48, 859-867.	5.3	7
150	5â€² Splice Site Mutations in tau Associated with the Inherited Dementia FTDP-17 Affect a Stem-Loop Structure That Regulates Alternative Splicing of Exon 10. Journal of Biological Chemistry, 1999, 274, 15134-15143.	3.4	266
151	Frequency oftau mutations in three series of non-Alzheimer's degenerative dementia. Annals of Neurology, 1999, 46, 243-248.	5.3	150
152	Apolipoprotein E genotype does not affect the age of onset of dementia in families with defined tau mutations. Neuroscience Letters, 1999, 260, 193-195.	2.1	27
153	Association of missense and 5â€²-splice-site mutations in tau with the inherited dementia FTDP-17. Nature, 1998, 393, 702-705.	27.8	3,333
154	Effect of apolipoprotein E status on clinical features of dementia. , 1998, 13, 177-185.		9
155	Preferential deposition of amyloid Î² protein (AÎ²) in the form AÎ²40 in Alzheimer's disease is associated with a gene dosage effect of the apolipoprotein E E4 allele. Neuroscience Letters, 1997, 221, 81-84.	2.1	94
156	An intronic polymorphism in the presenilin-1 gene does not influence the amount or molecular form of the amyloid Î² protein deposited in Alzheimer's disease. Neuroscience Letters, 1997, 222, 57-60.	2.1	14
157	The relative abundance of dopamine D4 receptor mRNA in post mortem brains of schizophrenics and controls. Schizophrenia Research, 1996, 20, 171-174.	2.0	29
158	ApoE2 Allele, Down's Syndrome, and Dementiaa. Annals of the New York Academy of Sciences, 1996, 777, 255-259.	3.8	19
159	Debrisoquine hydroxylase gene polymorphism frequencies in patients with amyotrophic lateral sclerosis. Neuroscience Letters, 1996, 208, 65-68.	2.1	17
160	The extent of amyloid deposition in brain in patients with Down's syndrome does not depend upon the apolipoprotein E genotype. Neuroscience Letters, 1995, 196, 105-108.	2.1	23
161	Apolipoprotein E allelic frequencies in patients with lobar atrophy. Neuroscience Letters, 1995, 188, 205-207.	2.1	37
162	Allelic variations in apolipoprotein E and prion protein genotype related to plaque formation and age of onset in sporadic Creutzfeldt-Jakob disease. Neuroscience Letters, 1995, 187, 127-129.	2.1	35

#	Article	IF	Citations
163	The abundance of mRNA for dopamine D2 receptor isoforms in brain tissue from controls and schizophrenics. Molecular Brain Research, 1994, 25, 173-175.	2.3	21
164	Apolipoprotein E Îµ2 allele promotes longevity and protects patients with DownÊ¼s syndrome from dementia. NeuroReport, 1994, 5, 2583-2585.	1.2	93
165	Genome-Wide Analyses Identify KIF5A as a Novel ALS Gene. SSRN Electronic Journal, 0, , .	0.4	4