Tammaryn Lashley

List of Publications by Year
in descending order

148	12,127	44444 50	35168 102
papers	citations	h-index	g-index

170	170	170	15673
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Variability in the type and layer distribution of cortical AÎ² pathology in familial Alzheimerâ€™s disease. Brain Pathology, 2022, 32, e13009.	2.1	12
2	HnRNP K mislocalisation in neurons of the dentate nucleus is a novel neuropathological feature of neurodegenerative disease and ageing. Neuropathology and Applied Neurobiology, 2022, 48, .	1.8	11
3	Brain region-specific susceptibility of Lewy body pathology in synucleinopathies is governed by Î±-synuclein conformations. Acta Neuropathologica, 2022, 143, 453-469.	3.9	14
4	TDP-43 loss and ALS-risk SNPs drive mis-splicing and depletion of UNC13A. Nature, 2022, 603, 131-137.	13.7	188
5	Markers of cognitive resilience and a framework for investigating clinical heterogeneity in <scp>ALS</scp> â€. Journal of Pathology, 2022, , .	2.1	0
6	Age-dependent formation of TMEM106B amyloid filaments in human brains. Nature, 2022, 605, 310-314.	13.7	88
7	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
8	Somatic copy number variant mutations in alpha-synuclein and genome-wide in brains of synucleinopathy cases. Journal of Neurology, Neurosurgery and Psychiatry, 2022, 93, A4.2-A4.	0.9	0
9	N-terminally truncated AÎ²4-x proteoforms and their relevance for Alzheimerâ€™s pathophysiology. Translational Neurodegeneration, 2022, 11, .	3.6	7
10	A Clinicopathologic Study of Movement Disorders in Frontotemporal Lobar Degeneration. Movement Disorders, 2021, 36, 632-641.	2.2	3
11	Novel clinicopathological characteristics differentiate dementia with Lewy bodies from Parkinsonâ€™s disease dementia. Neuropathology and Applied Neurobiology, 2021, 47, 143-156.	1.8	19
12	The age-dependent associations of white matter hyperintensities and neurofilament light in early- and late-stage Alzheimer's disease. Neurobiology of Aging, 2021, 97, 10-17.	1.5	18
13	Molecular forms of neurogranin in cerebrospinal fluid. Journal of Neurochemistry, 2021, 157, 816-833.	2.1	6
14	Neuroligin-1 in brain and CSF of neurodegenerative disorders: investigation for synaptic biomarkers. Acta Neuropathologica Communications, 2021, 9, 19.	2.4	17
15	Genome sequencing analysis identifies new loci associated with Lewy body dementia and provides insights into its genetic architecture. Nature Genetics, 2021, 53, 294-303.	9.4	198
16	Discriminatory ability of next-generation tau PET tracers for Alzheimerâ€™s disease. Brain, 2021, 144, 2284-2290.	3.7	29
17	HnRNP K mislocalisation is a novel protein pathology of frontotemporal lobar degeneration and ageing and leads to cryptic splicing. Acta Neuropathologica, 2021, 142, 609-627.	3.9	24
18	Comparing amyloid-Î² plaque burden with antemortem PiB PET in autosomal dominant and late-onset Alzheimer disease. Acta Neuropathologica, 2021, 142, 689-706.	3.9	15

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19	A novel presenilin 1 duplication mutation (Ile168dup) causing Alzheimer's disease associated with myoclonus, seizures and pyramidal features. Neurobiology of Aging, 2021, 103, 137.e1-137.e5.	1.5	1
20	The localization of amyloid precursor protein to ependymal cilia in vertebrates and its role in ciliogenesis and brain development in zebrafish. Scientific Reports, 2021, 11, 19115.	1.6	4
21	Amyloid pathology and synaptic loss in pathological aging. Journal of Neurochemistry, 2021, 159, 258-272.	2.1	6
22	Structure-based classification of tauopathies. Nature, 2021, 598, 359-363.	13.7	409
23	Association of clusterin with the BRI2-derived amyloid molecules ABri and ADan. Neurobiology of Disease, 2021, 158, 105452.	2.1	5
24	MOBP and HIP1 in multiple system atrophy: New Î±â€synuclein partners in glial cytoplasmic inclusions implicated in the disease pathogenesis. Neuropathology and Applied Neurobiology, 2021, 47, 640-652.	1.8	11
25	Familial Alzheimerâ€™s Disease Mutations in PSEN1 Lead to Premature Human Stem Cell Neurogenesis. Cell Reports, 2021, 34, 108615.	2.9	53
26	Heterogeneous Nuclear Ribonucleoproteins: Implications in Neurological Diseases. Molecular Neurobiology, 2021, 58, 631-646.	1.9	37
27	Extensive Anti-CoA Immunostaining in Alzheimerâ€™s Disease and Covalent Modification of Tau by a Key Cellular Metabolite Coenzyme A. Frontiers in Cellular Neuroscience, 2021, 15, 739425.	1.8	8
28	Quantitative detection of grey and white matter amyloid pathology using a combination of K114 and CRANAD-3 fluorescence. Neurobiology of Disease, 2021, 161, 105540.	2.1	8
29	FTLDâ€TDP assemblies seed neoaggregates with subtypeâ€specific features via a prionâ€like cascade. EMBO Reports, 2021, 22, e53877.	2.0	14
30	Presence of coâ€pathology in sporadic earlyâ€onset Alzheimer disease versus dominantly inherited Alzheimer disease. Alzheimer's and Dementia, 2021, 17, e055045.	0.4	0
31	The role of the RNA binding protein HnRNP K in the pathogenesis of frontotemporal lobar degeneration.. Alzheimer's and Dementia, 2021, 17 Suppl 3, e052826.	0.4	0
32	Familial Alzheimerâ€™s disease patient-derived neurons reveal distinct mutation-specific effects on amyloid beta. Molecular Psychiatry, 2020, 25, 2919-2931.	4.1	99
33	White matter DNA methylation profiling reveals deregulation of HIP1, LMAN2, MOBP, and other loci in multiple system atrophy. Acta Neuropathologica, 2020, 139, 135-156.	3.9	42
34	Structural and functional conservation of non-lumenized lymphatic endothelial cells in the mammalian leptomeninges. Acta Neuropathologica, 2020, 139, 383-401.	3.9	24
35	Corticospinal tract degeneration and temporal lobe atrophy in frontotemporal lobar degeneration TDPâ€43 type C pathology. Neuropathology and Applied Neurobiology, 2020, 46, 296-299.	1.8	6
36	A case of TDP-43 type C pathology presenting as nonfluent variant primary progressive aphasia. Neurocase, 2020, 26, 1-6.	0.2	4

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37	Two pathologically confirmed cases of novel mutations in the MAPT gene causing frontotemporal dementia. Neurobiology of Aging, 2020, 87, 141.e15-141.e20.	1.5	3
38	The role of hnRNPs in frontotemporal dementia and amyotrophic lateral sclerosis. Acta Neuropathologica, 2020, 140, 599-623.	3.9	62
39	Microglial burden, activation and dystrophy patterns in frontotemporal lobar degeneration. Journal of Neuroinflammation, 2020, 17, 234.	3.1	29
40	Neuronal intranuclear inclusion disease is genetically heterogeneous. Annals of Clinical and Translational Neurology, 2020, 7, 1716-1725.	1.7	38
41	Medin aggregation causes cerebrovascular dysfunction in aging wild-type mice. Proceedings of the National Academy of Sciences of the United States of America, 2020, 117, 23925-23931.	3.3	20
42	White matter hyperintensity increases are a feature of familial AD and are associated with increased brain atrophy. Alzheimer's and Dementia, 2020, 16, e038925.	0.4	0
43	Premature neuronal differentiation in familial Alzheimerâ€™s disease human stem cells in vitro and in postmortem brain tissue. Alzheimer's and Dementia, 2020, 16, e039793.	0.4	0
44	iPSCâ€derived engineered cerebral organoids (enCORs) as in vitro models of tauopathy. Alzheimer's and Dementia, 2020, 16, e039816.	0.4	0
45	Soluble and insoluble dipeptide repeat protein measurements in C9orf72-frontotemporal dementia brains show regional differential solubility and correlation of poly-GR with clinical severity. Acta Neuropathologica Communications, 2020, 8, 184.	2.4	25
46	In vivo staging of frontotemporal lobar degeneration TDP-43 type C pathology. Alzheimer's Research and Therapy, 2020, 12, 34.	3.0	20
47	Relevance of biomarkers across different neurodegenerative diseases. Alzheimer's Research and Therapy, 2020, 12, 56.	3.0	42
48	Epigenomics and transcriptomics analyses of multiple system atrophy brain tissue supports a role for inflammatory processes in disease pathogenesis. Acta Neuropathologica Communications, 2020, 8, 71.	2.4	5
49	Symmetric dimethylation of poly-GR correlates with disease duration in C9orf72 FTLD and ALS and reduces poly-GR phase separation and toxicity. Acta Neuropathologica, 2020, 139, 407-410.	3.9	36
50	Investigation of pathology, expression and proteomic profiles in human TREM2 variant postmortem brains with and without Alzheimerâ€™s disease. Brain Pathology, 2020, 30, 794-810.	2.1	10
51	CSF amyloid is a consistent predictor of white matter hyperintensities across the disease course from aging to Alzheimer's disease. Neurobiology of Aging, 2020, 91, 5-14.	1.5	30
52	Truncated stathmin-2 is a marker of TDP-43 pathology in frontotemporal dementia. Journal of Clinical Investigation, 2020, 130, 6080-6092.	3.9	117
53	Analysis of neurodegenerative disease-causing genes in dementia with Lewy bodies. Acta Neuropathologica Communications, 2020, 8, 5.	2.4	27
54	LATE to the PART-y. Brain, 2019, 142, e47-e47.	3.7	44

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55	Frontotemporal lobar degenerations: from basic science to clinical manifestations. Neuropathology and Applied Neurobiology, 2019, 45, 3-5.	1.8	1
56	A novel TBK1 mutation in a family with diverse frontotemporal dementia spectrum disorders. Journal of Physical Education and Sports Management, 2019, 5, a003913.	0.5	19
57	Alzheimerâ€™s disease phospholipase C-gamma-2 (PLCG2) protective variant is a functional hypermorph. Alzheimer's Research and Therapy, 2019, 11, 16.	3.0	100
58	Review: Clinical, neuropathological and genetic features of Lewy body dementias. Neuropathology and Applied Neurobiology, 2019, 45, 635-654.	1.8	26
59	Mass Spectrometric Analysis of Lewy Body-Enriched Î±-Synuclein in Parkinsonâ€™s Disease. Journal of Proteome Research, 2019, 18, 2109-2120.	1.8	49
60	ApoE4 lowers age at onset in patients with frontotemporal dementia and tauopathy independent of amyloidâ€Î²2 copathology. Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring, 2019, 11, 277-280.	1.2	24
61	Heritability and genetic variance of dementia with Lewy bodies. Neurobiology of Disease, 2019, 127, 492-501.	2.1	29
62	Heterogeneous nuclear ribonucleoproteins R and Q accumulate in pathological inclusions in FTLD-FUS. Acta Neuropathologica Communications, 2019, 7, 18.	2.4	26
63	A distinct brain beta amyloid signature in cerebral amyloid angiopathy compared to Alzheimerâ€™s disease. Neuroscience Letters, 2019, 701, 125-131.	1.0	43
64	Pyroglutamation of amyloid-Î²x-42 (AÎ²x-42) followed by AÎ²1â€“40 deposition underlies plaque polymorphism in progressing Alzheimerâ€™s disease pathology. Journal of Biological Chemistry, 2019, 294, 6719-6732.	1.6	49
65	Investigation of somatic CNVs in brains of synucleinopathy cases using targeted SNCA analysis and single cell sequencing. Acta Neuropathologica Communications, 2019, 7, 219.	2.4	35
66	TDP-43 extracted from frontotemporal lobar degeneration subject brains displays distinct aggregate assemblies and neurotoxic effects reflecting disease progression rates. Nature Neuroscience, 2019, 22, 65-77.	7.1	143
67	A comprehensive screening of copy number variability in dementia with Lewy bodies. Neurobiology of Aging, 2019, 75, 223.e1-223.e10.	1.5	13
68	Novel tau fragments in cerebrospinal fluid: relation to tangle pathology and cognitive decline in Alzheimerâ€™s disease. Acta Neuropathologica, 2019, 137, 279-296.	3.9	128
69	The intact postsynaptic protein neurogranin is reduced in brain tissue from patients with familial and sporadic Alzheimerâ€™s disease. Acta Neuropathologica, 2019, 137, 89-102.	3.9	64
70	<scp>APOE</scp> Îµ4 is also required in <scp>TREM</scp>2 R47H variant carriers for Alzheimer's disease to develop. Neuropathology and Applied Neurobiology, 2019, 45, 183-186.	1.8	12
71	Investigating the genetic architecture of dementia with Lewy bodies: a two-stage genome-wide association study. Lancet Neurology, The, 2018, 17, 64-74.	4.9	195
72	P1â€188: MODELLING AMYLOID BETA PROFILES IN IPSCâ€DERIVED CORTICAL NEURONS OF MULTIPLE FAMILIAL ALZHEIMER'S DISEASE GENOTYPES, INCLUDING A CASE STUDY OF SAME DONOR CULTURE MEDIA, CSF AND BRAIN TISSUE. Alzheimer's and Dementia, 2018, 14, P350.	0.4	0

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73	P3â€461: THE DISTRIBUTION OF CORTICAL AÎ² AND MICROGLIAL PATHOLOGY IN FAMILIAL ALZHEIMER'S DISEASE. Alzheimer's and Dementia, 2018, 14, P1295.	0.4	0
74	Hypertonic Stress Causes Cytoplasmic Translocation of Neuronal, but Not Astrocytic, FUS due to Impaired Transportin Function. Cell Reports, 2018, 24, 987-1000.e7.	2.9	49
75	The presubiculum is preserved from neurodegenerative changes in Alzheimerâ€™s disease. Acta Neuropathologica Communications, 2018, 6, 62.	2.4	9
76	Large inter- and intra-case variability of first generation tau PET ligand binding in neurodegenerative dementias. Acta Neuropathologica Communications, 2018, 6, 34.	2.4	31
77	Molecular biomarkers of Alzheimer's disease: progress and prospects. DMM Disease Models and Mechanisms, 2018, 11, .	1.2	163
78	Pathological correlates of white matter hyperintensities in a case of progranulin mutation associated frontotemporal dementia. Neurocase, 2018, 24, 166-174.	0.2	40
79	Immunohistochemical and Molecular Investigations Show Alteration in the Inflammatory Profile of Multiple System Atrophy Brain. Journal of Neuropathology and Experimental Neurology, 2018, 77, 598-607.	0.9	18
80	Somatic copy number gains of Î±-synuclein (SNCA) in Parkinsonâ€™s disease and multiple system atrophy brains. Brain, 2018, 141, 2419-2431.	3.7	63
81	The clinical, neuroanatomical, and neuropathologic phenotype of TBK1â€associated frontotemporal dementia: A longitudinal case report. Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring, 2017, 6, 75-81.	1.2	28
82	Bidirectional nucleolar dysfunction in C9orf72 frontotemporal lobar degeneration. Acta Neuropathologica Communications, 2017, 5, 29.	2.4	43
83	Expanding the genetic heterogeneity of intellectual disability. Human Genetics, 2017, 136, 1419-1429.	1.8	122
84	[P2â€“441]: PATHOLOGICAL CORRELATES OF WHITE MATTER HYPERINTENSITIES ON CADAVERIC MRI IN PROGRANULINâ€ASSOCIATED FRONTOTEMPORAL DEMENTIA. Alzheimer's and Dementia, 2017, 13, P805.	0.4	0
85	Amyloid polymorphisms constitute distinct clouds of conformational variants in different etiological subtypes of Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 13018-13023.	3.3	170
86	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.	2.4	15
87	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.	2.4	20
88	Analysis of C9orf72 repeat expansions in a large international cohort of dementia with Lewy bodies. Neurobiology of Aging, 2017, 49, 214.e13-214.e15.	1.5	12
89	[P1â€“220]: 3D CEREBRAL ORGANOIDS AS IN VITRO MODELS FOR ALZHEIMER's DISEASE. Alzheimer's and Dementia, 2017, 13, P327.	0.4	0
90	[P2â€“158]: IS THE PRESUBICULUM PROTECTED FROM NEURODEGENERATIVE CHANGES? A PATHOLOGICAL AND BIOCHEMICAL INVESTIGATION. Alzheimer's and Dementia, 2017, 13, P668.	0.4	0

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91	Neuronal and Peripheral Pentraxins Modify Glutamate Release and may Interact in Bloodâ€“Brain Barrier Failure. Cerebral Cortex, 2017, 27, 3437-3448.	1.6	34
92	P2â€152: Characterization of the Postsynaptic Protein Neurogranin in Different Brain Regions in Patients with Familial Alzheimer's Disease, Alzheimer's Disease, Pathological Aging And Healthy Controls. Alzheimer's and Dementia, 2016, 12, P672.	0.4	0
93	P1-002: AD-Associated TREM2 Variants Lead to Some Subpopulations of Microglia to be Less Abundant But More Activated. , 2016, 12, P397-P397.		0
94	The presence of heterogeneous nuclear ribonucleoproteins in frontotemporal lobar degeneration with FUS-positive inclusions. Neurobiology of Aging, 2016, 46, 192-203.	1.5	20
95	Clinical phenotype and genetic associations in autosomal dominant familial Alzheimerâ€™s disease: a case series. Lancet Neurology, The, 2016, 15, 1326-1335.	4.9	163
96	Apomorphine: A potential modifier of amyloid deposition in Parkinson's disease?. Movement Disorders, 2016, 31, 668-675.	2.2	31
97	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.	1.5	78
98	Characterization of tau positron emission tomography tracer [¹⁸F]AVâ€1451 binding to postmortem tissue in Alzheimer's disease,Âprimary tauopathies, and other dementias. Alzheimer's and Dementia, 2016, 12, 1116-1124.	0.4	161
99	Review: An update on clinical, genetic and pathological aspects of frontotemporal lobar degenerations. Neuropathology and Applied Neurobiology, 2015, 41, 858-881.	1.8	168
100	Spontaneous ARIA (Amyloid-Related Imaging Abnormalities) and Cerebral Amyloid Angiopathy Related Inflammation in Presenilin 1-Associated Familial Alzheimer's Disease. Journal of Alzheimer's Disease, 2015, 44, 1069-1074.	1.2	22
101	C9orf72 expansions in frontotemporal dementia and amyotrophic lateral sclerosis. Lancet Neurology, The, 2015, 14, 291-301.	4.9	270
102	Brain Amyloid-Beta Fragment Signatures in Pathological Ageing and Alzheimer's Disease by Hybrid Immunoprecipitation Mass Spectrometry. Neurodegenerative Diseases, 2015, 15, 50-57.	0.8	41
103	Alterations in global <scp>DNA</scp> methylation and hydroxymethylation are not detected in <scp>A</scp>lzheimer's disease. Neuropathology and Applied Neurobiology, 2015, 41, 497-506.	1.8	78
104	Genetic determinants of white matter hyperintensities and amyloid angiopathy in familial Alzheimer's disease. Neurobiology of Aging, 2015, 36, 3140-3151.	1.5	53
105	Histological evidence of chronic traumatic encephalopathy in a large series of neurodegenerative diseases. Acta Neuropathologica, 2015, 130, 891-893.	3.9	92
106	Qualitative changes in human Î³-secretase underlie familial Alzheimerâ€™s disease. Journal of Experimental Medicine, 2015, 212, 2003-2013.	4.2	134
107	A 30-unit hexanucleotide repeat expansion in C9orf72 induces pathological lesions with dipeptide-repeat proteins and RNA foci, but not TDP-43 inclusions and clinical disease. Acta Neuropathologica, 2015, 130, 599-601.	3.9	31
108	Evaluating the relationship between amyloid-Î² and Î±-synuclein phosphorylated at Ser129 in dementia with Lewy bodies and Parkinsonâ€™s disease. Alzheimer's Research and Therapy, 2014, 6, 77.	3.0	74

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109	Genetic analysis implicates APOE, SNCA and suggests lysosomal dysfunction in the etiology of dementia with Lewy bodies. Human Molecular Genetics, 2014, 23, 6139-6146.	1.4	178
110	A pathogenic progranulin mutation and <scp>C9orf72</scp> repeat expansion in a family with frontotemporal dementia. Neuropathology and Applied Neurobiology, 2014, 40, 502-513.	1.8	37
111	Neuropathological features of genetically confirmed DYT1 dystonia: investigating disease-specific inclusions. Acta Neuropathologica Communications, 2014, 2, 159.	2.4	21
112	The Significance of Î±-Synuclein, Amyloid-Î² and Tau Pathologies in Parkinson's Disease Progression and Related Dementia. Neurodegenerative Diseases, 2014, 13, 154-156.	0.8	83
113	Widespread RNA metabolism impairment in sporadic inclusion body myositis TDP43-proteinopathy. Neurobiology of Aging, 2014, 35, 1491-1498.	1.5	36
114	Homozygosity for the C9orf72 GGGGCC repeat expansion in frontotemporal dementia. Acta Neuropathologica, 2013, 126, 401-409.	3.9	126
115	A Novel Prion Disease Associated with Diarrhea and Autonomic Neuropathy. New England Journal of Medicine, 2013, 369, 1904-1914.	13.9	113
116	TREM2 Variants in Alzheimer's Disease. New England Journal of Medicine, 2013, 368, 117-127.	13.9	2,385
117	RANTing about C9orf72. Neuron, 2013, 77, 597-598.	3.8	19
118	Genetic Analysis of Inherited Leukodystrophies. JAMA Neurology, 2013, 70, 875.	4.5	147
119	C9orf72 frontotemporal lobar degeneration is characterised by frequent neuronal sense and antisense RNA foci. Acta Neuropathologica, 2013, 126, 845-857.	3.9	289
120	Frontotemporal dementia with the C9ORF72 hexanucleotide repeat expansion: clinical, neuroanatomical and neuropathological features. Brain, 2012, 135, 736-750.	3.7	392
121	O1â€05â€01: Frontotemporal dementia with the C9ORF72 hexanucleotide repeat expansion: Clinical, neuroanatomical and neuropathological features. Alzheimer's and Dementia, 2012, 8, P92.	0.4	0
122	Globular glial tauopathies (GGT) presenting with motor neuron disease or frontotemporal dementia: an emerging group of 4-repeat tauopathies. Acta Neuropathologica, 2011, 122, 415-428.	3.9	67
123	Transportin1: a marker of FTLD-FUS. Acta Neuropathologica, 2011, 122, 591-600.	3.9	58
124	A comparative clinical, pathological, biochemical and genetic study of fused in sarcoma proteinopathies. Brain, 2011, 134, 2548-2564.	3.7	76
125	Clinical and neuroanatomical signatures of tissue pathology in frontotemporal lobar degeneration. Brain, 2011, 134, 2565-2581.	3.7	306
126	Lewy- and Alzheimer-type pathologies in Parkinson's disease dementia: which is more important?. Brain, 2011, 134, 1493-1505.	3.7	497

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127	The clinical and neuroanatomical phenotype of FUS associated frontotemporal lobar degeneration. Journal of Neurology, Neurosurgery and Psychiatry, 2011, 82, 1405-1407.	0.9	32
128	Cerebral amyloidosis: amyloid subunits, mutants and phenotypes. Cellular and Molecular Life Sciences, 2010, 67, 581-600.	2.4	52
129	Common variants at 7p21 are associated with frontotemporal lobar degeneration with TDP-43 inclusions. Nature Genetics, 2010, 42, 234-239.	9.4	479
130	Genetics and molecular pathogenesis of sporadic and hereditary cerebral amyloid angiopathies. Acta Neuropathologica, 2009, 118, 115-130.	3.9	255
131	Cortical Î±-synuclein load is associated with amyloid-Î² plaque burden in a subset of Parkinsonâ€™s disease patients. Acta Neuropathologica, 2008, 115, 417-425.	3.9	146
132	Expression of BRI2 mRNA and protein in normal human brain and familial British dementia: its relevance to the pathogenesis of disease. Neuropathology and Applied Neurobiology, 2008, 34, 492-505.	1.8	28
133	Mutations in TTBK2, encoding a kinase implicated in tau phosphorylation, segregate with spinocerebellar ataxia type 11. Nature Genetics, 2007, 39, 1434-1436.	9.4	185
134	Genetic Alterations of the BRI2 gene: Familial British and Danish Dementias. Brain Pathology, 2006, 16, 71-79.	2.1	40
135	Molecular chaperons, amyloid and preamyloid lesions in the BRI2 gene-related dementias: a morphological study. Neuropathology and Applied Neurobiology, 2006, 32, 492-504.	1.8	29
136	Chromosome 13 dementias. Cellular and Molecular Life Sciences, 2005, 62, 1814-1825.	2.4	59
137	Familial Danish Dementia. Journal of Biological Chemistry, 2005, 280, 36883-36894.	1.6	59
138	P1-262 Familial British and Danish dementias: BRI2 gene and protein expression by human cerebral cells. Neurobiology of Aging, 2004, 25, S170-S171.	1.5	0
139	P1-264 The possible origin of the amyloid peptides in the BRI2 gene-related dementias. Neurobiology of Aging, 2004, 25, S171.	1.5	0
140	Cerebral Amyloid Angiopathies: A Pathologic, Biochemical, and Genetic View. Journal of Neuropathology and Experimental Neurology, 2003, 62, 885-898.	0.9	245
141	Complement Activation in Chromosome 13 Dementias. Journal of Biological Chemistry, 2002, 277, 49782-49790.	1.6	59
142	Familial Danish Dementia: A Novel Form of Cerebral Amyloidosis Associated with Deposition of Both Amyloid-Dan and Amyloid-Beta. Journal of Neuropathology and Experimental Neurology, 2002, 61, 254-267.	0.9	116
143	Familial British dementia (FBD): a cerebral amyloidosis with systemic amyloid deposition. Neuropathology and Applied Neurobiology, 2002, 28, 148-148.	1.8	4
144	Regional Distribution of Amyloid-Bri Deposition and Its Association with Neurofibrillary Degeneration in Familial British Dementia. American Journal of Pathology, 2001, 158, 515-526.	1.9	127

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145	Chromosome 13 dementia syndromes as models of neurodegeneration. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 2001, 8, 277-284.	1.4	29
146	Systemic Amyloid Deposits in Familial British Dementia. Journal of Biological Chemistry, 2001, 276, 43909-43914.	1.6	73
147	Variant Alzheimer's disease with spastic paraparesis and cotton wool plaques is caused by PS-1 mutations that lead to exceptionally high amyloid-? concentrations. Annals of Neurology, 2000, 48, 806-808.	2.8	135
148	Variant Alzheimer's disease with spastic paraparesis and cotton wool plaques is caused by PSâ€1 mutations that lead to exceptionally high amyloidâ€Î² concentrations. Annals of Neurology, 2000, 48, 806-808.	2.8	3