Masato Hasegawa

List of Publications by Year
in descending order

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103	15,345	36271	30058
papers	citations	51 h-index	103 g-index

111	111	111	12434
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	TDP-43 is a component of ubiquitin-positive tau-negative inclusions in frontotemporal lobar degeneration and amyotrophic lateral sclerosis. Biochemical and Biophysical Research Communications, 2006, 351, 602-611.	1.0	2,248
2	Î±-Synuclein is phosphorylated in synucleinopathy lesions. Nature Cell Biology, 2002, 4, 160-164.	4.6	1,739
3	Prion-like spreading of pathological Î±-synuclein in brain. Brain, 2013, 136, 1128-1138.	3.7	691
4	Phosphorylated TDPâ€43 in frontotemporal lobar degeneration and amyotrophic lateral sclerosis. Annals of Neurology, 2008, 64, 60-70.	2.8	630
5	Reconsideration of Amyloid Hypothesis and Tau Hypothesis in Alzheimer's Disease. Frontiers in Neuroscience, 2018, 12, 25.	1.4	591
6	Inhibition of Heparin-induced Tau Filament Formation by Phenothiazines, Polyphenols, and Porphyrins. Journal of Biological Chemistry, 2005, 280, 7614-7623.	1.6	479
7	Structures of Î±-synuclein filaments from multiple system atrophy. Nature, 2020, 585, 464-469.	13.7	446
8	Tau proteins with FTDPâ€17 mutations have a reduced ability to promote microtubule assembly. FEBS Letters, 1998, 437, 207-210.	1.3	440
9	Prion-like Properties of Pathological TDP-43 Aggregates from Diseased Brains. Cell Reports, 2013, 4, 124-134.	2.9	418
10	Structure-based classification of tauopathies. Nature, 2021, 598, 359-363.	13.7	409
11	Frontotemporal Dementia and Corticobasal Degeneration in a Family with a P301S Mutation in Tau. Journal of Neuropathology and Experimental Neurology, 1999, 58, 667-677.	0.9	381
12	Novel tau filament fold in corticobasal degeneration. Nature, 2020, 580, 283-287.	13.7	381
13	Phosphorylated Î±-Synuclein Is Ubiquitinated in Î±-Synucleinopathy Lesions. Journal of Biological Chemistry, 2002, 277, 49071-49076.	1.6	365
14	Accumulation of Phosphorylated Î±-Synuclein in Aging Human Brain. Journal of Neuropathology and Experimental Neurology, 2003, 62, 644-654.	0.9	312
15	Phosphorylated TDP-43 in Alzheimerâ€™s disease and dementia with Lewy bodies. Acta Neuropathologica, 2009, 117, 125-136.	3.9	294
16	Misfolded proteinase Kâ€“resistant hyperphosphorylated Î±-synuclein in aged transgenic mice with locomotor deterioration and in human Î±-synucleinopathies. Journal of Clinical Investigation, 2002, 110, 1429-1439.	3.9	292
17	Seeded Aggregation and Toxicity of Î±-Synuclein and Tau. Journal of Biological Chemistry, 2010, 285, 34885-34898.	1.6	288
18	TDP-43 is deposited in the Guam parkinsonism-dementia complex brains. Brain, 2007, 130, 1386-1394.	3.7	210

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19	Hyperphosphorylation of Tau in PHF. Neurobiology of Aging, 1995, 16, 365-371.	1.5	207
20	Pathological alpha-synuclein propagates through neural networks. Acta Neuropathologica Communications, 2014, 2, 88.	2.4	203
21	Misfolded proteinase Kâ€“resistant hyperphosphorylated Î±-synuclein in aged transgenic mice with locomotor deterioration and in human Î±-synucleinopathies. Journal of Clinical Investigation, 2002, 110, 1429-1439.	3.9	195
22	Alzheimer-like Changes in Microtubule-associated Protein Tau Induced by Sulfated Glycosaminoglycans. Journal of Biological Chemistry, 1997, 272, 33118-33124.	1.6	184
23	Identification of amino-terminally cleaved tau fragments that distinguish progressive supranuclear palsy from corticobasal degeneration. Annals of Neurology, 2004, 55, 72-79.	2.8	170
24	Biochemical classification of tauopathies by immunoblot, protein sequence and mass spectrometric analyses of sarkosyl-insoluble and trypsin-resistant tau. Acta Neuropathologica, 2016, 131, 267-280.	3.9	167
25	Regulation of Mitochondrial Transport and Inter-Microtubule Spacing by Tau Phosphorylation at the Sites Hyperphosphorylated in Alzheimer's Disease. Journal of Neuroscience, 2012, 32, 2430-2441.	1.7	156
26	Propagation of pathological Î±-synuclein in marmoset brain. Acta Neuropathologica Communications, 2017, 5, 12.	2.4	142
27	Structure of pathological TDP-43 filaments from ALS with FTLD. Nature, 2022, 601, 139-143.	13.7	129
28	Ubiquitination of Î±-Synucleinâ€. Biochemistry, 2005, 44, 361-368.	1.2	122
29	Mass spectrometric analysis of accumulated TDP-43 in amyotrophic lateral sclerosis brains. Scientific Reports, 2016, 6, 23281.	1.6	118
30	Progranulin regulates lysosomal function and biogenesis through acidification of lysosomes. Human Molecular Genetics, 2017, 26, ddx011.	1.4	110
31	Phosphorylated and cleaved TDP-43 in ALS, FTLD and other neurodegenerative disorders and in cellular models of TDP-43 proteinopathy. Neuropathology, 2010, 30, 170-181.	0.7	109
32	Phosphorylated TDP-43 pathology and hippocampal sclerosis in progressive supranuclear palsy. Acta Neuropathologica, 2010, 120, 55-66.	3.9	97
33	Molecular analysis and biochemical classification of TDP-43 proteinopathy. Brain, 2012, 135, 3380-3391.	3.7	95
34	Seeded assembly inÂvitro does not replicate the structures of Î±â€synuclein filaments from multiple system atrophy. FEBS Open Bio, 2021, 11, 999-1013.	1.0	95
35	Conversion of Wild-type Î±-Synuclein into Mutant-type Fibrils and Its Propagation in the Presence of A30P Mutant. Journal of Biological Chemistry, 2009, 284, 7940-7950.	1.6	94
36	Accumulation of phosphorylated TDP-43 in brains of patients with argyrophilic grain disease. Acta Neuropathologica, 2009, 117, 151-158.	3.9	91

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37	Templated Aggregation of TAR DNA-binding Protein of 43 kDa (TDP-43) by Seeding with TDP-43 Peptide Fibrils. Journal of Biological Chemistry, 2016, 291, 8896-8907.	1.6	89
38	The Effect of Fragmented Pathogenic Î±-Synuclein Seeds on Prion-like Propagation. Journal of Biological Chemistry, 2016, 291, 18675-18688.	1.6	88
39	Age-dependent formation of TMEM106B amyloid filaments in human brains. Nature, 2022, 605, 310-314.	13.7	88
40	Plasma phosphorylated-TDP-43 protein levels correlate with brain pathology in frontotemporal lobar degeneration. Acta Neuropathologica, 2009, 118, 647-658.	3.9	82
41	Neuronal and glial inclusions in frontotemporal dementia with or without motor neuron disease are immunopositive for p62. Neuroscience Letters, 2003, 342, 41-44.	1.0	81
42	Methylene Blue Reduced Abnormal Tau Accumulation in P301L Tau Transgenic Mice. PLoS ONE, 2012, 7, e52389.	1.1	79
43	The basis of clinicopathological heterogeneity in TDP-43 proteinopathy. Acta Neuropathologica, 2019, 138, 751-770.	3.9	78
44	Cysteine misincorporation in bacterially expressed human Î±-synuclein. FEBS Letters, 2006, 580, 1775-1779.	1.3	74
45	Potent prion-like behaviors of pathogenic Î±-synuclein and evaluation of inactivation methods. Acta Neuropathologica Communications, 2018, 6, 29.	2.4	73
46	Fibrillogenic Nuclei Composed of P301L Mutant Tau Induce Elongation of P301L Tau but Not Wild-type Tau*. Journal of Biological Chemistry, 2007, 282, 20309-20318.	1.6	71
47	Î±-Synuclein Fibrils Exhibit Gain of Toxic Function, Promoting Tau Aggregation and Inhibiting Microtubule Assembly. Journal of Biological Chemistry, 2016, 291, 15046-15056.	1.6	67
48	Extracellular association of APP and tau fibrils induces intracellular aggregate formation of tau. Acta Neuropathologica, 2015, 129, 895-907.	3.9	65
49	Extensive deamidation at asparagine residue 279 accounts for weak immunoreactivity of tau with RD4 antibody in Alzheimerâ€™s disease brain. Acta Neuropathologica Communications, 2013, 1, 54.	2.4	61
50	Pathological Endogenous Î±-Synuclein Accumulation in Oligodendrocyte Precursor Cells Potentially Induces Inclusions in Multiple System Atrophy. Stem Cell Reports, 2018, 10, 356-365.	2.3	61
51	Molecular Mechanisms in the Pathogenesis of Alzheimerâ€™s disease and Tauopathies-Prion-Like Seeded Aggregation and Phosphorylation. Biomolecules, 2016, 6, 24.	1.8	53
52	Isomerase Pin1 Stimulates Dephosphorylation of Tau Protein at Cyclin-dependent Kinase (Cdk5)-dependent Alzheimer Phosphorylation Sites. Journal of Biological Chemistry, 2013, 288, 7968-7977.	1.6	52
53	Prion-like mechanisms and potential therapeutic targets in neurodegenerative disorders. , 2017, 172, 22-33.		52
54	Wild-Type Monomeric Î±-Synuclein Can Impair Vesicle Endocytosis and Synaptic Fidelity via Tubulin Polymerization at the Calyx of Held. Journal of Neuroscience, 2017, 37, 6043-6052.	1.7	51

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55	The twenty-four KDa C-terminal tau fragment increases with aging in tauopathy mice: implications of prion-like properties. Human Molecular Genetics, 2015, 24, 6403-6416.	1.4	50
56	Molecular Dissection of TDP-43 Proteinopathies. Journal of Molecular Neuroscience, 2011, 45, 480-485.	1.1	49
57	Mutations in CHCHD2 cause Î±-synuclein aggregation. Human Molecular Genetics, 2019, 28, 3895-3911.	1.4	48
58	Comparison of Common and Disease-Specific Post-translational Modifications of Pathological Tau Associated With a Wide Range of Tauopathies. Frontiers in Neuroscience, 2020, 14, 581936.	1.4	47
59	Progranulin Reduction Is Associated With Increased Tau Phosphorylation in P301L Tau Transgenic Mice. Journal of Neuropathology and Experimental Neurology, 2015, 74, 158-165.	0.9	46
60	Î±-Synuclein fibrils subvert lysosome structure and function for the propagation of protein misfolding between cells through tunneling nanotubes. PLoS Biology, 2021, 19, e3001287.	2.6	45
61	Î±-synuclein strains that cause distinct pathologies differentially inhibit proteasome. ELife, 2020, 9, .	2.8	45
62	LATE to the PART-y. Brain, 2019, 142, e47-e47.	3.7	44
63	Isoform-independent and -dependent phosphorylation of microtubule-associated protein tau in mouse brain during postnatal development. Journal of Biological Chemistry, 2018, 293, 1781-1793.	1.6	36
64	TDP-43 Prions. Cold Spring Harbor Perspectives in Medicine, 2018, 8, a024463.	2.9	34
65	Pathological tau deposition in Motor Neurone Disease and frontotemporal lobar degeneration associated with TDP-43 proteinopathy. Acta Neuropathologica Communications, 2016, 4, 33.	2.4	33
66	Accumulation of multiple neurodegenerative disease-related proteins in familial frontotemporal lobar degeneration associated with granulin mutation. Scientific Reports, 2017, 7, 1513.	1.6	33
67	Epitope mapping of antibodies against TDP-43 and detection of protease-resistant fragments of pathological TDP-43 in amyotrophic lateral sclerosis and frontotemporal lobar degeneration. Biochemical and Biophysical Research Communications, 2012, 417, 116-121.	1.0	27
68	Tau isoform expression and phosphorylation in marmoset brains. Journal of Biological Chemistry, 2019, 294, 11433-11444.	1.6	27
69	Î±-Synuclein: Experimental Pathology. Cold Spring Harbor Perspectives in Medicine, 2016, 6, a024273.	2.9	26
70	TDP-43 pathology in familial British dementia. Acta Neuropathologica, 2009, 118, 303-311.	3.9	23
71	Ultrastructural and biochemical classification of pathogenic tau, Î±-synuclein and TDP-43. Acta Neuropathologica, 2022, 143, 613-640.	3.9	22
72	Quantitative and combinatory determination of in situ phosphorylation of tau and its FTDP-17 mutants. Scientific Reports, 2016, 6, 33479.	1.6	21

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73	The Abundance of Nonphosphorylated Tau in Mouse and Human Tauopathy Brains Revealed by the Use of Phos-Tag Method. American Journal of Pathology, 2016, 186, 398-409.	1.9	20
74	Structurally Distinct Î±â€Synuclein Fibrils Induce Robust Parkinsonian Pathology. Movement Disorders, 2020, 35, 256-267.	2.2	20
75	N-Methyl-D-Aspartate Receptor Link to the MAP Kinase Pathway in Cortical and Hippocampal Neurons and Microglia Is Dependent on Calcium Sensors and Is Blocked by Î±-Synuclein, Tau, and Phospho-Tau in Non-transgenic and Transgenic APPSw,Ind Mice. Frontiers in Molecular Neuroscience, 2018, 11, 273.	1.4	19
76	Prion-like propagation of Î±-synuclein in neurodegenerative diseases. Progress in Molecular Biology and Translational Science, 2019, 168, 323-348.	0.9	18
77	Human NPCs can degrade Î±â€“syn fibrils and transfer them preferentially in a cell contact-dependent manner possibly through TNT-like structures. Neurobiology of Disease, 2019, 132, 104609.	2.1	17
78	Human tauopathy-derived tau strains determine the substrates recruited for templated amplification. Brain, 2021, 144, 2333-2348.	3.7	17
79	Distinct phosphorylation profiles of tau in brains of patients with different tauopathies. Neurobiology of Aging, 2021, 108, 72-79.	1.5	17
80	Phosphorylation of endogenous Î±-synuclein induced by extracellular seeds initiates at the pre-synaptic region and spreads to the cell body. Scientific Reports, 2022, 12, 1163.	1.6	17
81	Effect of L-DOPA/Benserazide on Propagation of Pathological Î±-Synuclein. Frontiers in Neuroscience, 2019, 13, 595.	1.4	15
82	Motor neuron TDP-43 proteinopathy in progressive supranuclear palsy and corticobasal degeneration. Brain, 2022, 145, 2769-2784.	3.7	15
83	Tau progression in single severe frontal traumatic brain injury in human brains. Journal of the Neurological Sciences, 2019, 407, 116495.	0.3	13
84	An autopsy case of globular glial tauopathy presenting with clinical features of motor neuron disease with dementia and iron deposition in the motor cortex. Neuropathology, 2018, 38, 372-379.	0.7	12
85	Factors associated with development and distribution of granular/fuzzy astrocytes in neurodegenerative diseases. Brain Pathology, 2020, 30, 811-830.	2.1	12
86	Development of a novel tau propagation mouse model endogenously expressing 3 and 4 repeat tau isoforms. Brain, 2022, 145, 349-361.	3.7	11
87	Independent distribution between tauopathy secondary to subacute sclerotic panencephalitis and measles virus: An immunohistochemical analysis in autopsy cases including cases treated with aggressive antiviral therapies. Brain Pathology, 2022, 32, e13069.	2.1	11
88	Asparagine residue 368 is involved in Alzheimer's disease tau strainâ€“specific aggregation. Journal of Biological Chemistry, 2020, 295, 13996-14014.	1.6	10
89	Globular glial tauopathy Type I presenting with behavioral variant frontotemporal dementia. Neuropathology, 2020, 40, 515-525.	0.7	9
90	Tau Protein and Frontotemporal Dementias. Advances in Experimental Medicine and Biology, 2021, 1281, 177-199.	0.8	8

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91	Experimental models of prionâ€like protein propagation. Neuropathology, 2020, 40, 460-466.	0.7	6
92	Progression of phosphorylated Î±â€synuclein in Macaca fuscata. Brain Pathology, 2021, 31, e12952.	2.1	6
93	Amyotrophic lateral sclerosis with speech apraxia, predominant upper motor neuron signs, and prominent iron accumulation in the frontal operculum and precentral gyrus. Neuropathology, 2021, 41, 324-331.	0.7	6
94	Dextran sulphate-induced tau assemblies cause endogenous tau aggregation and propagation in wild-type mice. Brain Communications, 2020, 2, fcaa091.	1.5	6
95	The hot cross bun sign in corticobasal degeneration. Neuropathology, 2021, 41, 376-380.	0.7	4
96	Structures of tau and Î±-synuclein filaments from brains of patients with neurodegenerative diseases. Neurochemistry International, 2022, 158, 105362.	1.9	3
97	Common Marmoset Model of. Methods in Molecular Biology, 2021, 2322, 131-139.	0.4	2
98	Electron Microscopic Analysis of. Methods in Molecular Biology, 2021, 2322, 17-25.	0.4	2
99	An autopsy case of progressive supranuclear palsy. Pallidoâ€nigroâ€luysian type with argyrophilic grains clinically presenting with personality and behavioral changes. Neuropathology, 2022, 42, 447-452.	0.7	2
100	An autopsy case of corticobasal syndrome due to asymmetric degeneration of the motor cortex and substantia nigra with <scp>TDP</scp>â€43 proteinopathy, associated with <scp>Alzheimer's</scp> disease pathology. Neuropathology, 2021, 41, 214-225.	0.7	1
101	An autopsy case of corticobasal degeneration with inferior olivary hypertrophy. Neuropathology, 2021, 41, 226-235.	0.7	1
102	An autopsy case of A lzheimer's disease with amygdalaâ€predominant L ewy pathology presenting with frontotemporal dementiaâ€like psychiatric symptoms. Neuropathology, 2022, , .	0.7	0
103	Frontotemporal Lobar Degeneration With Unclassifiable 4-Repeat Tauopathy Mimicking Globular Glial Tauopathy. Journal of Neuropathology and Experimental Neurology, 2022, 81, 581-584.	0.9	0