Philip C Wong

List of Publications by Year
in descending order

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107 papers	14,521 citations	44042 48 h-index	39638 94 g-index
116 all docs	116 docs citations	116 times ranked	16401 citing authors

#	Article	IF	Citations
1	Presenilin Is Essential for ApoE Secretion, a Novel Role of Presenilin Involved in Alzheimer's Disease Pathogenesis. Journal of Neuroscience, 2022, 42, 1574-1586.	1.7	8
2	Loss of TDP-43 function and rimmed vacuoles persist after T cell depletion in a xenograft model of sporadic inclusion body myositis. Science Translational Medicine, 2022, 14, eabi9196.	5.8	27
3	Human brain sialoglycan ligand for CD33, a microglial inhibitory Siglec implicated in Alzheimerâ€™s disease. Journal of Biological Chemistry, 2022, 298, 101960.	1.6	13
4	Aberrant neural activity in prefrontal pyramidal neurons lacking TDP-43 precedes neuron loss. Progress in Neurobiology, 2022, 215, 102297.	2.8	3
5	D-Glucose uptake and clearance in the tauopathy Alzheimerâ€™s disease mouse brain detected by on-resonance variable delay multiple pulse MRI. Journal of Cerebral Blood Flow and Metabolism, 2021, 41, 1013-1025.	2.4	27
6	Hemizygous deletion of Tbk1 worsens neuromuscular junction pathology in TDP-43 transgenic mice. Experimental Neurology, 2021, 335, 113496.	2.0	15
7	Brain metabolism in tau and amyloid mouse models of Alzheimer's disease: An MRI study. NMR in Biomedicine, 2021, 34, e4568.	1.6	11
8	Early detection of Alzheimer's disease using creatine chemical exchange saturation transfer magnetic resonance imaging. NeuroImage, 2021, 236, 118071.	2.1	20
9	Loss of TDP-43 in male germ cells causes meiotic failure and impairs fertility in mice. Journal of Biological Chemistry, 2021, 297, 101231.	1.6	8
10	Upregulation of ATG7 attenuates motor neuron dysfunction associated with depletion of TARDBP/TDP-43. Autophagy, 2020, 16, 672-682.	4.3	24
11	Splicing repression is a major function of TDP-43 in motor neurons. Acta Neuropathologica, 2019, 138, 813-826.	3.9	60
12	Longitudinal diffusion tensor magnetic resonance imaging analysis at the cohort level reveals disturbed cortical and callosal microstructure with spared corticospinal tract in the TDP-43G298S ALS mouse model. Translational Neurodegeneration, 2019, 8, 27.	3.6	13
13	Protein aggregation linked to Alzheimer's disease revealed by saturation transfer MRI. NeuroImage, 2019, 188, 380-390.	2.1	50
14	Reversible induction of TDP-43 granules in cortical neurons after traumatic injury. Experimental Neurology, 2018, 299, 15-25.	2.0	41
15	Tdp-43 cryptic exons are highly variable between cell types. Molecular Neurodegeneration, 2017, 12, 13.	4.4	63
16	Cryptic exon incorporation occurs in Alzheimerâ€™s brain lacking TDP-43 inclusion but exhibiting nuclear clearance of TDP-43. Acta Neuropathologica, 2017, 133, 923-931.	3.9	58
17	An LHX1-Regulated Transcriptional Network Controls Sleep/Wake Coupling and Thermal Resistance of the Central Circadian Clockworks. Current Biology, 2017, 27, 128-136.	1.8	36
18	Challenges and Advances in Gene Therapy Approaches for Neurodegenerative Disorders. Current Gene Therapy, 2017, 17, 187-193.	0.9	9

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19	O5-03-04: G-Secretase Modulator Enhances the Ab-Lowering Effect of Bace1 Inhibitor in Mouse Models of Alzheimerâ€™s Disease. , 2016, 12, P383-P384.		0
20	PTBP1 and PTBP2 Repress Nonconserved Cryptic Exons. Cell Reports, 2016, 17, 104-113.	2.9	53
21	Motor neuron disease, TDP-43 pathology, and memory deficits in mice expressing ALSâ€“FTD-linked UBQLN2 mutations. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, E7580-E7589.	3.3	77
22	Depletion of TDP-43 decreases fibril and plaque Î²-amyloid and exacerbates neurodegeneration in an Alzheimerâ€™s mouse model. Acta Neuropathologica, 2016, 132, 859-873.	3.9	43
23	The neuritic plaque facilitates pathological conversion of tau in an Alzheimerâ€™s disease mouse model. Nature Communications, 2016, 7, 12082.	5.8	56
24	Serotonin 2B receptor slows disease progression and prevents degeneration of spinal cord mononuclear phagocytes in amyotrophic lateral sclerosis. Acta Neuropathologica, 2016, 131, 465-480.	3.9	41
25	TDP-43 repression of nonconserved cryptic exons is compromised in ALS-FTD. Science, 2015, 349, 650-655.	6.0	419
26	Intraventricular Delivery of siRNA Nanoparticles to the Central Nervous System. Molecular Therapy - Nucleic Acids, 2015, 4, e242.	2.3	43
27	Low dietary protein content alleviates motor symptoms in mice with mutant dynactin/dynein-mediated neurodegeneration. Human Molecular Genetics, 2015, 24, 2228-2240.	1.4	22
28	Defective Age-Dependent Metaplasticity in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2015, 35, 11346-11357.	1.7	54
29	GGGGCC repeat expansion in C9orf72 compromises nucleocytoplasmic transport. Nature, 2015, 525, 129-133.	13.7	692
30	Alzheimer Disease. , 2015, , 321-338.		9
31	Postsynaptic Target Specific Synaptic Dysfunctions in the CA3 Area of BACE1 Knockout Mice. PLoS ONE, 2014, 9, e92279.	1.1	25
32	Function, therapeutic potential and cell biology of <scp>BACE</scp> proteases: current status and future prospects. Journal of Neurochemistry, 2014, 130, 4-28.	2.1	269
33	Treatment with bexarotene, a compound that increases apolipoprotein-E, provides no cognitive benefit in mutant APP/PS1 mice. Molecular Neurodegeneration, 2013, 8, 18.	4.4	75
34	Altered astrocytic expression of TDP-43 does not influence motor neuron survival. Experimental Neurology, 2013, 250, 250-259.	2.0	49
35	The dynactin p150 subunit: cell biology studies of sequence changes found in ALS/MND and Parkinsonian Syndromes. Journal of Neural Transmission, 2013, 120, 785-798.	1.4	35
36	Motor Neuron Diseases. , 2012, , 801-814.		3

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37	Neurobiology of Alzheimerâ€™s Disease. , 2012, , 815-828.		3
38	Specific domains in anterior pharynx-defective 1 determine its intramembrane interactions with nicastrin and presenilin. Neurobiology of Aging, 2012, 33, 277-285.	1.5	7
39	Rodent models of TDP-43: Recent advances. Brain Research, 2012, 1462, 26-39.	1.1	99
40	Selectivity to amyloid-Î² precursor protein cleavage provides hope against Alzheimer's. Alzheimer's Research and Therapy, 2011, 3, 7.	3.0	2
41	Amyloid Precursor Protein Processing and Alzheimer's Disease. Annual Review of Neuroscience, 2011, 34, 185-204.	5.0	1,455
42	Arc/Arg3.1 Regulates an Endosomal Pathway Essential for Activity-Dependent Î²-Amyloid Generation. Cell, 2011, 147, 615-628.	13.5	183
43	Reduced BACE1 Activity Enhances Clearance of Myelin Debris and Regeneration of Axons in the Injured Peripheral Nervous System. Journal of Neuroscience, 2011, 31, 5744-5754.	1.7	76
44	Increased Expression of PS1 Is Sufficient to Elevate the Level and Activity of Î³-Secretase In Vivo. PLoS ONE, 2011, 6, e28179.	1.1	24
45	An Overview of APP Processing Enzymes and Products. NeuroMolecular Medicine, 2010, 12, 1-12.	1.8	515
46	Deletion of TDP-43 down-regulates Tbc1d1 , a gene linked to obesity, and alters body fat metabolism. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 16320-16324.	3.3	255
47	Mossy Fiber Long-Term Potentiation Deficits in BACE1 Knock-Outs Can Be Rescued by Activation of Î±7 Nicotinic Acetylcholine Receptors. Journal of Neuroscience, 2010, 30, 13808-13813.	1.7	26
48	Modeling an Anti-Amyloid Combination Therapy for Alzheimerâ€™s Disease. Science Translational Medicine, 2010, 2, 13ra1.	5.8	42
49	Altered distributions of Gemini of coiled bodies and mitochondria in motor neurons of TDP-43 transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 16325-16330.	3.3	283
50	S-Palmitoylation of Î³-Secretase Subunits Nicastrin and APH-1. Journal of Biological Chemistry, 2009, 284, 1373-1384.	1.6	61
51	Alzheimer Disease AÎ² Production in the Absence of S-Palmitoylation-dependent Targeting of BACE1 to Lipid Rafts. Journal of Biological Chemistry, 2009, 284, 3793-3803.	1.6	137
52	Single Chain Variable Fragment against Nicastrin Inhibits the Î³-Secretase Activity. Journal of Biological Chemistry, 2009, 284, 27838-27847.	1.6	19
53	APH1 Polar Transmembrane Residues Regulate the Assembly and Activity of Presenilin Complexes. Journal of Biological Chemistry, 2009, 284, 16298-16307.	1.6	30
54	The Î²-Secretase Enzyme BACE in Health and Alzheimer's Disease: Regulation, Cell Biology, Function, and Therapeutic Potential. Journal of Neuroscience, 2009, 29, 12787-12794.	1.7	498

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55	Progressive behavioral deficits in DJ-1-deficient mice are associated with normal nigrostriatal function. Neurobiology of Disease, 2008, 29, 505-514.	2.1	89
56	Translational Control of BACE1 May Go Awry in Alzheimer's Disease. Neuron, 2008, 60, 941-943.	3.8	12
57	Glu332 in the Nicastrin Ectodomain Is Essential for Î³-Secretase Complex Maturation but Not for Its Activity. Journal of Biological Chemistry, 2008, 283, 20096-20105.	1.6	97
58	BACE1 Knock-Outs Display Deficits in Activity-Dependent Potentiation of Synaptic Transmission at Mossy Fiber to CA3 Synapses in the Hippocampus. Journal of Neuroscience, 2008, 28, 8677-8681.	1.7	98
59	Motor Neuron Disease Occurring in a Mutant Dynactin Mouse Model Is Characterized by Defects in Vesicular Trafficking. Journal of Neuroscience, 2008, 28, 1997-2005.	1.7	166
60	Messenger RNA Oxidation Occurs Early in Disease Pathogenesis and Promotes Motor Neuron Degeneration in ALS. PLoS ONE, 2008, 3, e2849.	1.1	178
61	Alzheimer's Betaâ€Secretase in Health and Disease. FASEB Journal, 2008, 22, 119.2.	0.2	0
62	Epidermal Growth Factor Receptor and Notch Pathways Participate in the Tumor Suppressor Function of Î³-Secretase. Journal of Biological Chemistry, 2007, 282, 32264-32273.	1.6	82
63	Moderate Reduction of Î³-Secretase Attenuates Amyloid Burden and Limits Mechanism-Based Liabilities. Journal of Neuroscience, 2007, 27, 10849-10859.	1.7	78
64	BACE1 inhibition reduces endogenous Abeta and alters APP processing in wild-type mice. Journal of Neurochemistry, 2006, 99, 1555-1563.	2.1	101
65	Amyotrophic Lateral Sclerosis 2-Deficiency Leads to Neuronal Degeneration in Amyotrophic Lateral Sclerosis through Altered AMPA Receptor Trafficking. Journal of Neuroscience, 2006, 26, 11798-11806.	1.7	79
66	Alzheimer Disease: Therapeutic Targets for Clinical Trials. Retina, 2005, 25, S80-S81.	1.0	1
67	Selected genetically engineered models relevant to human neurodegenerative disease. , 2005, , 176-195.		1
68	Alzheimer's Disease: Clinical Features, Neuropathologies and Biochemical Abnormalities, Genetics, Models, and Experimental Therapeutics. , 2005, , 445-458.		3
69	Axonal Transport, Amyloid Precursor Protein, Kinesin-1, and the Processing Apparatus: Revisited. Journal of Neuroscience, 2005, 25, 2386-2395.	1.7	221
70	Loss of ALS2 Function Is Insufficient to Trigger Motor Neuron Degeneration in Knock-Out Mice But Predisposes Neurons to Oxidative Stress. Journal of Neuroscience, 2005, 25, 7567-7574.	1.7	104
71	APH-1a Is the Principal Mammalian APH-1 Isoform Present in Â-Secretase Complexes during Embryonic Development. Journal of Neuroscience, 2005, 25, 192-198.	1.7	105
72	BACE1, a Major Determinant of Selective Vulnerability of the Brain to Amyloid-Â Amyloidogenesis, is Essential for Cognitive, Emotional, and Synaptic Functions. Journal of Neuroscience, 2005, 25, 11693-11709.	1.7	490

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73	Nicastrin Is Critical for Stability and Trafficking but Not Association of Other Presenilin/Î³-Secretase Components. Journal of Biological Chemistry, 2005, 280, 17020-17026.	1.6	105
74	Role of Alzheimer's disease models in designing and testing experimental therapeutics. Drug Discovery Today: Disease Models, 2005, 2, 305-312.	1.2	1
75	Association of Î³-Secretase with Lipid Rafts in Post-Golgi and Endosome Membranes. Journal of Biological Chemistry, 2004, 279, 44945-44954.	1.6	372
76	Binding Sites of Â-Secretase Inhibitors in Rodent Brain: Distribution, Postnatal Development, and Effect of Deafferentation. Journal of Neuroscience, 2004, 24, 2942-2952.	1.7	42
77	Toxicity of Familial ALS-Linked SOD1 Mutants from Selective Recruitment to Spinal Mitochondria. Neuron, 2004, 43, 5-17.	3.8	497
78	Early events of target deprivation/axotomyâ€induced neuronal apoptosis in vivo: oxidative stress, DNA damage, p53 phosphorylation and subcellular redistribution of death proteins. Journal of Neurochemistry, 2003, 85, 234-247.	2.1	71
79	Nicastrin Is Required for Assembly of Presenilin/Î³-Secretase Complexes to Mediate Notch Signaling and for Processing and Trafficking of Î²-Amyloid Precursor Protein in Mammals. Journal of Neuroscience, 2003, 23, 3272-3277.	1.7	174
80	Genetically engineered models of neurodegenerative diseases. , 2002, , 1841-1862.		1
81	Genetically engineered mouse models of neurodegenerative diseases. Nature Neuroscience, 2002, 5, 633-639.	7.1	219
82	Reply to 'Is ALS caused by an altered oxidative activity of mutant superoxide dismutase?'. Nature Neuroscience, 2002, 5, 919-920.	7.1	12
83	Mutant SOD1 causes motor neuron disease independent of copper chaperoneâ€“mediated copper loading. Nature Neuroscience, 2002, 5, 301-307.	7.1	253
84	Histological Evidence of Protein Aggregation in Mutant SOD1 Transgenic Mice and in Amyotrophic Lateral Sclerosis Neural Tissues. Neurobiology of Disease, 2001, 8, 933-941.	2.1	374
85	Genetically Engineered Models Relevant to Neurodegenerative Disorders: Their Value for Understanding Disease Mechanisms and Designing/Testing Experimental Therapeutics. Journal of Molecular Neuroscience, 2001, 17, 233-257.	1.1	14
86	BACE1 is the major Î²-secretase for generation of AÎ² peptides by neurons. Nature Neuroscience, 2001, 4, 233-234.	7.1	1,023
87	The Brain's Susceptibility to Amyloid Plaques. Science, 2001, 293, 1434b-1434.	6.0	15
88	The Value of Transgenic Models for the Study of Neurodegenerative Diseases. Annals of the New York Academy of Sciences, 2000, 920, 179-191.	1.8	51
89	Alzheimer%#x2019;s Disease and Genetically Engineered Animal Models. , 1999, , 187-214.		0
90	Transgenic Mouse Models of Alzheimer's Disease and Amyotrophic Lateral Sclerosis. Brain Pathology, 1998, 8, 735-757.	2.1	27

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91	The genetic and molecular mechanisms of motor neuron disease. Current Opinion in Neurobiology, 1998, 8, 791-799.	2.0	68
92	Effects of PS1 Deficiency on Membrane Protein Trafficking in Neurons. Neuron, 1998, 21, 1213-1221.	3.8	359
93	An Alzheimer's Disease-Linked PS1 Variant Rescues the Developmental Abnormalities of PS1-Deficient Embryos. Neuron, 1998, 20, 603-609.	3.8	134
94	Axonal Transport of Mutant Superoxide Dismutase 1 and Focal Axonal Abnormalities in the Proximal Axons of Transgenic Mice. Neurobiology of Disease, 1998, 5, 27-35.	2.1	96
95	Protective effect of neurofilament heavy gene overexpression in motor neuron disease induced by mutant superoxide dismutase. Proceedings of the National Academy of Sciences of the United States of America, 1998, 95, 9626-9630.	3.3	193
96	Familial Amyotrophic Lateral Sclerosis and Alzheimerâ€™s Disease. Advances in Experimental Medicine and Biology, 1998, , 145-159.	0.8	7
97	Transgenic Models of Amyotrophic Lateral Sclerosis and Alzheimer's Disease. , 1998, , 107-123.		0
98	Presenilin 1 is required for Notch 1 and Dll1 expression in the paraxial mesoderm. Nature, 1997, 387, 288-292.	13.7	730
99	Perspectives on the Mechanisms of Familial Amyotrophic Lateral Sclerosis Caused by Mutations in Superoxide Dismutase 1. , 1997, , 295-314.		0
100	Transgenic models of neurodegenerative diseases. Current Opinion in Neurobiology, 1996, 6, 651-660.	2.0	30
101	Transgenic and gene-targeting approaches to model disorders of motor neurons. Seminars in Neuroscience, 1996, 8, 163-169.	2.3	2
102	Inherited Neurodegenerative Diseases and Transgenic Models. Brain Pathology, 1996, 6, 467-480.	2.1	9
103	Motor neuron disease caused by mutations in superoxide dismutase 1. Current Opinion in Neurology, 1995, 8, 294-302.	1.8	34
104	Superoxide Dismutase 1 Subunits with Mutations Linked to Familial Amyotrophic Lateral Sclerosis Do Not Affect Wild-type Subunit Function. Journal of Biological Chemistry, 1995, 270, 3234-3238.	1.6	142
105	An adverse property of a familial ALS-linked SOD1 mutation causes motor neuron disease characterized by vacuolar degeneration of mitochondria. Neuron, 1995, 14, 1105-1116.	3.8	1,394
106	Alzheimer's disease, model systems and experimental therapeutics. , 0, , 565-586.		0
107	Î²-secretase: Physiological Role and Target Validation. , 0, , 59-76.		0