Dominic M Walsh

List of Publications by Year
in descending order

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Version: 2024-02-01

77	22,038	36203	79541
papers	citations	51 h-index	73 g-index

79	79	79	17629
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Naturally secreted oligomers of amyloid Î² protein potently inhibit hippocampal long-term potentiation in vivo. Nature, 2002, 416, 535-539.	13.7	3,979
2	Amyloid-Î² protein dimers isolated directly from Alzheimer's brains impair synaptic plasticity and memory. Nature Medicine, 2008, 14, 837-842.	15.2	3,225
3	Natural oligomers of the amyloid-Î² protein specifically disrupt cognitive function. Nature Neuroscience, 2005, 8, 79-84.	7.1	1,595
4	Natural Oligomers of the Alzheimer Amyloid-Â Protein Induce Reversible Synapse Loss by Modulating an NMDA-Type Glutamate Receptor-Dependent Signaling Pathway. Journal of Neuroscience, 2007, 27, 2866-2875.	1.7	1,445
5	Deciphering the Molecular Basis of Memory Failure in Alzheimer's Disease. Neuron, 2004, 44, 181-193.	3.8	1,127
6	Amyloid Î²-Protein Fibrillogenesis. Journal of Biological Chemistry, 1997, 272, 22364-22372.	1.6	967
7	Protofibrillar Intermediates of Amyloid Î²-Protein Induce Acute Electrophysiological Changes and Progressive Neurotoxicity in Cortical Neurons. Journal of Neuroscience, 1999, 19, 8876-8884.	1.7	926
8	Soluble Oligomers of Amyloid Î² Protein Facilitate Hippocampal Long-Term Depression by Disrupting Neuronal Glutamate Uptake. Neuron, 2009, 62, 788-801.	3.8	818
9	Soluble amyloid Î²-protein dimers isolated from Alzheimer cortex directly induce Tau hyperphosphorylation and neuritic degeneration. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 5819-5824.	3.3	770
10	Amyloid Î² protein immunotherapy neutralizes AÎ² oligomers that disrupt synaptic plasticity in vivo. Nature Medicine, 2005, 11, 556-561.	15.2	485
11	A vicious cycle of Î² amyloidâ€“dependent neuronal hyperactivation. Science, 2019, 365, 559-565.	6.0	407
12	Alzheimer's disease: synaptic dysfunction and AÎ². Molecular Neurodegeneration, 2009, 4, 48.	4.4	388
13	Amyloid Î² Protein Dimer-Containing Human CSF Disrupts Synaptic Plasticity: Prevention by Systemic Passive Immunization. Journal of Neuroscience, 2008, 28, 4231-4237.	1.7	293
14	Certain Inhibitors of Synthetic Amyloid Â-Peptide (AÂ) Fibrillogenesis Block Oligomerization of Natural AÂ and Thereby Rescue Long-Term Potentiation. Journal of Neuroscience, 2005, 25, 2455-2462.	1.7	286
15	Interaction between prion protein and toxic amyloid Î² assemblies can be therapeutically targeted at multiple sites. Nature Communications, 2011, 2, 336.	5.8	263
16	Large Soluble Oligomers of Amyloid Î²-Protein from Alzheimer Brain Are Far Less Neuroactive Than the Smaller Oligomers to Which They Dissociate. Journal of Neuroscience, 2017, 37, 152-163.	1.7	262
17	A critical appraisal of the pathogenic protein spread hypothesis of neurodegeneration. Nature Reviews Neuroscience, 2016, 17, 251-260.	4.9	251
18	A facile method for expression and purification of the Alzheimerâ€™s diseaseâ€associated amyloid Î²â€peptide. FEBS Journal, 2009, 276, 1266-1281.	2.2	237

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19	An improved method of preparing the amyloid Î²-protein for fibrillogenesis and neurotoxicity experiments. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 2000, 7, 166-178.	1.4	232
20	Amyloid Î²-Protein Dimers Rapidly Form Stable Synaptotoxic Protofibrils. Journal of Neuroscience, 2010, 30, 14411-14419.	1.7	232
21	The presence of sodium dodecyl sulphate-stable AÎ² dimers is strongly associated with Alzheimer-type dementia. Brain, 2010, 133, 1328-1341.	3.7	229
22	Alzheimer's Disease Brain-Derived Amyloid-Î²-Mediated Inhibition of LTPIn VivoIs Prevented by Immunotargeting Cellular Prion Protein. Journal of Neuroscience, 2011, 31, 7259-7263.	1.7	215
23	Developmental Regulation of Mitochondrial Apoptosis by c-Myc Governs Age- and Tissue-Specific Sensitivity to Cancer Therapeutics. Cancer Cell, 2017, 31, 142-156.	7.7	190
24	mGlu5 receptors and cellular prion protein mediate amyloid-Î²-facilitated synaptic long-term depression in vivo. Nature Communications, 2014, 5, 3374.	5.8	157
25	Detection of Aggregation-Competent Tau in Neuron-Derived Extracellular Vesicles. International Journal of Molecular Sciences, 2018, 19, 663.	1.8	140
26	C-Terminally Truncated Forms of Tau, But Not Full-Length Tau or Its C-Terminal Fragments, Are Released from Neurons Independently of Cell Death. Journal of Neuroscience, 2015, 35, 10851-10865.	1.7	131
27	miR-212 and miR-132 Are Downregulated in Neurally Derived Plasma Exosomes of Alzheimerâ€™s Patients. Frontiers in Neuroscience, 2019, 13, 1208.	1.4	129
28	Soluble AÎ² oligomers impair hippocampal LTP by disrupting glutamatergic/GABAergic balance. Neurobiology of Disease, 2016, 85, 111-121.	2.1	120
29	Autoregulated paracellular clearance of amyloid-Î² across the blood-brain barrier. Science Advances, 2015, 1, e1500472.	4.7	113
30	Amyloid-Î² nanotubes are associated with prion protein-dependent synaptotoxicity. Nature Communications, 2013, 4, 2416.	5.8	112
31	PrP is a central player in toxicity mediated by soluble aggregates of neurodegeneration-causing proteins. Acta Neuropathologica, 2020, 139, 503-526.	3.9	110
32	APP Homodimers Transduce an Amyloid-Î²-Mediated Increase in Release Probability at Excitatory Synapses. Cell Reports, 2014, 7, 1560-1576.	2.9	109
33	Human Brain-Derived AÎ² Oligomers Bind to Synapses and Disrupt Synaptic Activity in a Manner That Requires APP. Journal of Neuroscience, 2017, 37, 11947-11966.	1.7	108
34	Alzheimer's Disease and the Amyloid Î²-Protein. Progress in Molecular Biology and Translational Science, 2012, 107, 101-124.	0.9	106
35	Aggregation and Metalâ€Binding Properties of Mutant Forms of the Amyloid AÎ² Peptide of Alzheimer's Disease. Journal of Neurochemistry, 1996, 66, 740-747.	2.1	105
36	Diffusible, highly bioactive oligomers represent a critical minority of soluble AÎ² in Alzheimerâ€™s disease brain. Acta Neuropathologica, 2018, 136, 19-40.	3.9	100

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37	Î³-Secretase Cleavage and Binding to FE65 Regulate the Nuclear Translocation of the Intracellular C-Terminal Domain (ICD) of the APP Family of Proteinsâ€. Biochemistry, 2003, 42, 6664-6673.	1.2	94
38	Learnings about the complexity of extracellular tau aid development of a bloodâ€based screen for Alzheimer's disease. Alzheimer's and Dementia, 2019, 15, 487-496.	0.4	94
39	Aggregation and catabolism of disease-associated intra-AÎ² mutations: reduced proteolysis of AÎ²A21G by neprilysin. Neurobiology of Disease, 2008, 31, 442-450.	2.1	88
40	Amyloid Î²-protein and beyond: the path forward in Alzheimerâ€™s disease. Current Opinion in Neurobiology, 2020, 61, 116-124.	2.0	87
41	Secreted Amyloid Î²-Proteins in a Cell Culture Model Include N-Terminally Extended Peptides That Impair Synaptic Plasticity. Biochemistry, 2014, 53, 3908-3921.	1.2	85
42	A highly sensitive novel immunoassay specifically detects low levels of soluble AÎ² oligomers in human cerebrospinal fluid. Alzheimer's Research and Therapy, 2015, 7, 14.	3.0	78
43	Identification of neurotoxic cross-linked amyloid-Î² dimers in the Alzheimerâ€™s brain. Brain, 2019, 142, 1441-1457.	3.7	74
44	AÎ² dimers differ from monomers in structural propensity, aggregation paths and population of synaptotoxic assemblies. Biochemical Journal, 2014, 461, 413-426.	1.7	71
45	Target engagement in an alzheimer trial: Crenezumab lowers amyloid Î² oligomers in cerebrospinal fluid. Annals of Neurology, 2019, 86, 215-224.	2.8	70
46	Peripheral Administration of a Humanized Anti-PrP Antibody Blocks Alzheimer's Disease AÎ² Synaptotoxicity. Journal of Neuroscience, 2014, 34, 6140-6145.	1.7	68
47	Cellular Prion Protein Mediates the Disruption of Hippocampal Synaptic Plasticity by Soluble TauIn Vivo. Journal of Neuroscience, 2018, 38, 10595-10606.	1.7	66
48	Non-Fibrillar Oligomeric Amyloid-Î² within Synapses. Journal of Alzheimer's Disease, 2016, 53, 787-800.	1.2	65
49	Extracellular Forms of AÎ² and Tau from iPSC Models of Alzheimerâ€™s Disease Disrupt Synaptic Plasticity. Cell Reports, 2018, 23, 1932-1938.	2.9	60
50	N-Terminal Extensions Retard AÎ²42 Fibril Formation but Allow Cross-Seeding and Coaggregation with AÎ²42. Journal of the American Chemical Society, 2015, 137, 14673-14685.	6.6	58
51	Isolation of Low-n Amyloid Î²-Protein Oligomers from Cultured Cells, CSF, and Brain. Methods in Molecular Biology, 2010, 670, 33-44.	0.4	54
52	Alzheimer brain-derived amyloid Î²-protein impairs synaptic remodeling and memory consolidation. Neurobiology of Aging, 2013, 34, 1315-1327.	1.5	54
53	The aqueous phase of Alzheimer's disease brain contains assemblies built from â^¼4 and â^¼7 kDa AÎ² species. Alzheimer's and Dementia, 2015, 11, 1286-1305.	0.4	54
54	An in vitro paradigm to assess potential anti-AÎ² antibodies for Alzheimerâ€™s disease. Nature Communications, 2018, 9, 2676.	5.8	50

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55	The levels of water-soluble and triton-soluble AÎ² are increased in Alzheimer's disease brain. Brain Research, 2012, 1450, 138-147.	1.1	47
56	Anti-AÎ² antibodies incapable of reducing cerebral AÎ² oligomers fail to attenuate spatial reference memory deficits in J20 mice. Neurobiology of Disease, 2015, 82, 372-384.	2.1	37
57	Laboratory evolution of a sortase enzyme that modifies amyloid-Î² protein. Nature Chemical Biology, 2021, 17, 317-325.	3.9	34
58	Tau immunization: a cautionary tale?. Neurobiology of Aging, 2015, 36, 1316-1332.	1.5	28
59	Simultaneous measurement of a range of particle sizes during AÎ²1â€“42 fibrillogenesis quantified using fluorescence correlation spectroscopy. Biochemical and Biophysical Research Communications, 2014, 448, 195-199.	1.0	25
60	Soluble tau aggregates inhibit synaptic long-term depression and amyloid Î²-facilitated LTD in vivo. Neurobiology of Disease, 2019, 127, 582-590.	2.1	25
61	Neurotransmitter receptor and time dependence of the synaptic plasticity disrupting actions of Alzheimer's disease AÎ² in vivo. Philosophical Transactions of the Royal Society B: Biological Sciences, 2014, 369, 20130147.	1.8	23
62	A Human Monoclonal IgG That Binds AÎ² Assemblies and Diverse Amyloids Exhibits Anti-Amyloid ActivitiesIn VitroandIn Vivo. Journal of Neuroscience, 2015, 35, 6265-6276.	1.7	23
63	Î²â€Secretase cleavage is not required for generation of the intracellular Câ€terminal domain of the amyloid precursor family of proteins. FEBS Journal, 2010, 277, 1503-1518.	2.2	22
64	The Aggregation Paths and Products of AÎ²42 Dimers Are Distinct from Those of the AÎ²42 Monomer. Biochemistry, 2016, 55, 6150-6161.	1.2	22
65	Intracerebroventricular Administration of Amyloid Î²-protein Oligomers Selectively Increases Dorsal Hippocampal Dialysate Glutamate Levels in the Awake Rat. Sensors, 2008, 8, 7428-7437.	2.1	20
66	Dynamics of plasma biomarkers in Down syndrome: the relative levels of AÎ²42 decrease with age, whereas NT1 tau and NfL increase. Alzheimer's Research and Therapy, 2020, 12, 27.	3.0	20
67	The Many Faces of Aβ: Structures and Activity. Current Medicinal Chemistry Immunology, Endocrine & Metabolic Agents, 2003, 3, 277-291.	0.2	19
68	Peripheral Interventions Enhancing Brain Glutamate Homeostasis Relieve Amyloid Î²- and TNFÎ±- Mediated Synaptic Plasticity Disruption in the Rat Hippocampus. Cerebral Cortex, 2017, 27, 3724-3735.	1.6	17
69	PrP-grafted antibodies bind certain amyloid Î²-protein aggregates, but do not prevent toxicity. Brain Research, 2019, 1710, 125-135.	1.1	14
70	Soluble AÎ² aggregates can inhibit prion propagation. Open Biology, 2017, 7, 170158.	1.5	11
71	The ELISA-Measured Increase in Cerebrospinal Fluid Tau that Discriminates Alzheimer's Disease from other Neurodegenerative Disorders is not Attributable to Differential Recognition of Tau Assembly Forms. Journal of Alzheimer's Disease, 2013, 33, 923-928.	1.2	10
72	IgG Conformer's Binding to Amyloidogenic Aggregates. PLoS ONE, 2015, 10, e0137344.	1.1	5

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73	DTâ€02â€03: TARGET ENGAGEMENT IN AN AD TRIAL: CRENEZUMAB LOWERS AÎ² OLIGOMER LEVELS IN CSF. Alzheimer's and Dementia, 2018, 14, P1669.	0.4	3
74	F5-02-01: Getting a handle on soluble aÃŸ in Alzheimer's disease brains. , 2015, 11, P304-P305.		0
75	P1â€106: A HEADâ€TOâ€HEAD COMPARISON OF LEAD CLINICAL ANTIâ€AÎ² ANTIBODIES. Alzheimer's and Dementia, 2018, 14, P312.	0.4	0
76	P1â€301: CERTAIN PLASMA Nâ€TERMINAL TAU FRAGMENTS ARE ELEVATED IN AD AND ADâ€MCI COMPARED TO CONTROLS. Alzheimer's and Dementia, 2018, 14, P405.	0.4	0
77	Transcriptomic correlates of neurite degeneration due to human brainâ€derived AÎ² and protection by clinical antiâ€AÎ² antibodies. Alzheimer's and Dementia, 2020, 16, e043057.	0.4	0