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List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

77 papers 18,648 46 p-index 79 g-index

79 citations 10.1 6.46 ext. papers ext. citations avg, IF L-index

#	Paper	IF	Citations
77	Laboratory evolution of a sortase enzyme that modifies amyloid-[protein. <i>Nature Chemical Biology</i> , 2021 , 17, 317-325	11.7	10
76	Dynamics of plasma biomarkers in Down syndrome: the relative levels of AB2 decrease with age, whereas NT1 tau and NfL increase. <i>Alzheimers Research and Therapy</i> , 2020 , 12, 27	9	12
75	Amyloid Eprotein and beyond: the path forward in Alzheimerß disease. <i>Current Opinion in Neurobiology</i> , 2020 , 61, 116-124	7.6	56
74	Transcriptomic correlates of neurite degeneration due to human brain-derived Aland protection by clinical anti-Alantibodies. <i>Alzheimers and Dementia</i> , 2020 , 16, e043057	1.2	
73	PrP is a central player in toxicity mediated by soluble aggregates of neurodegeneration-causing proteins. <i>Acta Neuropathologica</i> , 2020 , 139, 503-526	14.3	55
72	Target engagement in an alzheimer trial: Crenezumab lowers amyloid lbligomers in cerebrospinal fluid. <i>Annals of Neurology</i> , 2019 , 86, 215-224	9.4	41
71	Identification of neurotoxic cross-linked amyloid-ldimers in the Alzheimerß brain. <i>Brain</i> , 2019 , 142, 144	1-1:457	40
70	Soluble tau aggregates inhibit synaptic long-term depression and amyloid Facilitated LTD in vivo. <i>Neurobiology of Disease</i> , 2019 , 127, 582-590	7.5	12
69	A vicious cycle of 🖾 myloid-dependent neuronal hyperactivation. <i>Science</i> , 2019 , 365, 559-565	33.3	206
68	miR-212 and miR-132 Are Downregulated in Neurally Derived Plasma Exosomes of Alzheimer Patients. <i>Frontiers in Neuroscience</i> , 2019 , 13, 1208	5.1	61
67	PrP-grafted antibodies bind certain amyloid Eprotein aggregates, but do not prevent toxicity. <i>Brain Research</i> , 2019 , 1710, 125-135	3.7	13
66	Learnings about the complexity of extracellular tau aid development of a blood-based screen for Alzheimer® disease. <i>Alzheimer</i> ® and <i>Dementia</i> , 2019 , 15, 487-496	1.2	60
65	Diffusible, highly bioactive oligomers represent a critical minority of soluble Alın Alzheimerß disease brain. <i>Acta Neuropathologica</i> , 2018 , 136, 19-40	14.3	47
64	Detection of Aggregation-Competent Tau in Neuron-Derived Extracellular Vesicles. <i>International Journal of Molecular Sciences</i> , 2018 , 19,	6.3	86
63	An in vitro paradigm to assess potential anti-Alantibodies for Alzheimerß disease. <i>Nature Communications</i> , 2018 , 9, 2676	17.4	26
62	P1-106: A HEAD-TO-HEAD COMPARISON OF LEAD CLINICAL ANTI-ALANTIBODIES 2018 , 14, P312-P312		
61	P1-301: CERTAIN PLASMA N-TERMINAL TAU FRAGMENTS ARE ELEVATED IN AD AND AD-MCI COMPARED TO CONTROLS 2018 , 14, P405-P405		

60	DT-02-03: TARGET ENGAGEMENT IN AN AD TRIAL: CRENEZUMAB LOWERS AIDLIGOMER LEVELS IN CSF 2018 , 14, P1669-P1670		3
59	Cellular Prion Protein Mediates the Disruption of Hippocampal Synaptic Plasticity by Soluble Tau. <i>Journal of Neuroscience</i> , 2018 , 38, 10595-10606	6.6	45
58	Extracellular Forms of Aland Tau from iPSC Models of Alzheimer Disease Disrupt Synaptic Plasticity. <i>Cell Reports</i> , 2018 , 23, 1932-1938	10.6	43
57	Developmental Regulation of Mitochondrial Apoptosis by c-Myc Governs Age- and Tissue-Specific Sensitivity to Cancer Therapeutics. <i>Cancer Cell</i> , 2017 , 31, 142-156	24.3	123
56	Soluble Alaggregates can inhibit prion propagation. <i>Open Biology</i> , 2017 , 7,	7	9
55	Human Brain-Derived AlDligomers Bind to Synapses and Disrupt Synaptic Activity in a Manner That Requires APP. <i>Journal of Neuroscience</i> , 2017 , 37, 11947-11966	6.6	72
54	Large Soluble Oligomers of Amyloid Protein from Alzheimer Brain Are Far Less Neuroactive Than the Smaller Oligomers to Which They Dissociate. <i>Journal of Neuroscience</i> , 2017 , 37, 152-163	6.6	185
53	Peripheral Interventions Enhancing Brain Glutamate Homeostasis Relieve Amyloid Eland TNFE Mediated Synaptic Plasticity Disruption in the Rat Hippocampus. <i>Cerebral Cortex</i> , 2017 , 27, 3724-3735	5.1	13
52	Non-Fibrillar Oligomeric Amyloid-Iwithin Synapses. <i>Journal of Alzheimers</i> Disease, 2016 , 53, 787-800	4.3	43
51	The Aggregation Paths and Products of AB2 Dimers Are Distinct from Those of the AB2 Monomer. <i>Biochemistry</i> , 2016 , 55, 6150-6161	3.2	15
50	Soluble Albligomers impair hippocampal LTP by disrupting glutamatergic/GABAergic balance. <i>Neurobiology of Disease</i> , 2016 , 85, 111-121	7.5	92
49	A critical appraisal of the pathogenic protein spread hypothesis of neurodegeneration. <i>Nature Reviews Neuroscience</i> , 2016 , 17, 251-60	13.5	198
48	Tau immunization: a cautionary tale?. Neurobiology of Aging, 2015, 36, 1316-32	5.6	25
47	The aqueous phase of Alzheimer ß disease brain contains assemblies built from ~4 and ~7 kDa All species. <i>Alzheimers</i> and Dementia, 2015 , 11, 1286-305	1.2	41
46	A highly sensitive novel immunoassay specifically detects low levels of soluble Albligomers in human cerebrospinal fluid. <i>Alzheimers Research and Therapy</i> , 2015 , 7, 14	9	63
45	Autoregulated paracellular clearance of amyloid-lacross the blood-brain barrier. <i>Science Advances</i> , 2015 , 1, e1500472	14.3	73
44	N-Terminal Extensions Retard A&2 Fibril Formation but Allow Cross-Seeding and Coaggregation with A&2. <i>Journal of the American Chemical Society</i> , 2015 , 137, 14673-85	16.4	51
43	C-Terminally Truncated Forms of Tau, But Not Full-Length Tau or Its C-Terminal Fragments, Are Released from Neurons Independently of Cell Death. <i>Journal of Neuroscience</i> , 2015 , 35, 10851-65	6.6	106

42	A human monoclonal IgG that binds alassemblies and diverse amyloids exhibits anti-amyloid activities in vitro and in vivo. <i>Journal of Neuroscience</i> , 2015 , 35, 6265-76	6.6	18
41	Anti-Alantibodies incapable of reducing cerebral Albligomers fail to attenuate spatial reference memory deficits in J20 mice. <i>Neurobiology of Disease</i> , 2015 , 82, 372-384	7.5	31
40	F5-02-01: Getting a handle on soluble alln Alzheimerß disease brains 2015 , 11, P304-P305		
39	IgG Conformer B Binding to Amyloidogenic Aggregates. <i>PLoS ONE</i> , 2015 , 10, e0137344	3.7	3
38	Aldimers differ from monomers in structural propensity, aggregation paths and population of synaptotoxic assemblies. <i>Biochemical Journal</i> , 2014 , 461, 413-26	3.8	53
37	Secreted amyloid Eproteins in a cell culture model include N-terminally extended peptides that impair synaptic plasticity. <i>Biochemistry</i> , 2014 , 53, 3908-21	3.2	71
36	Neurotransmitter receptor and time dependence of the synaptic plasticity disrupting actions of Alzheimerß disease Alīn vivo. <i>Philosophical Transactions of the Royal Society B: Biological Sciences</i> , 2014 , 369, 20130147	5.8	17
35	Simultaneous measurement of a range of particle sizes during All-42 fibrillogenesis quantified using fluorescence correlation spectroscopy. <i>Biochemical and Biophysical Research Communications</i> , 2014 , 448, 195-9	3.4	18
34	APP homodimers transduce an amyloid-Emediated increase in release probability at excitatory synapses. <i>Cell Reports</i> , 2014 , 7, 1560-1576	10.6	82
33	Peripheral administration of a humanized anti-PrP antibody blocks Alzheimer disease All synaptotoxicity. <i>Journal of Neuroscience</i> , 2014 , 34, 6140-5	6.6	57
32	mGlu5 receptors and cellular prion protein mediate amyloid-Facilitated synaptic long-term depression in vivo. <i>Nature Communications</i> , 2014 , 5, 3374	17.4	122
31	The ELISA-measured increase in cerebrospinal fluid tau that discriminates Alzheimerß disease from other neurodegenerative disorders is not attributable to differential recognition of tau assembly forms. <i>Journal of Alzheimers Disease</i> , 2013, 33, 923-8	4.3	9
30	Alzheimer brain-derived amyloid Eprotein impairs synaptic remodeling and memory consolidation. <i>Neurobiology of Aging</i> , 2013 , 34, 1315-27	5.6	47
29	Amyloid-Ihanotubes are associated with prion protein-dependent synaptotoxicity. <i>Nature Communications</i> , 2013 , 4, 2416	17.4	97
28	The levels of water-soluble and triton-soluble Alare increased in Alzheimerß disease brain. <i>Brain Research</i> , 2012 , 1450, 138-47	3.7	43
27	Alzheimerß disease and the amyloid Eprotein. <i>Progress in Molecular Biology and Translational Science</i> , 2012 , 107, 101-24	4	96
26	Isolation of low-n amyloid Eprotein oligomers from cultured cells, CSF, and brain. <i>Methods in Molecular Biology</i> , 2011 , 670, 33-44	1.4	44
25	Soluble amyloid beta-protein dimers isolated from Alzheimer cortex directly induce Tau hyperphosphorylation and neuritic degeneration. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2011 , 108, 5819-24	11.5	641

(2003-2011)

24	Interaction between prion protein and toxic amyloid lassemblies can be therapeutically targeted at multiple sites. <i>Nature Communications</i> , 2011 , 2, 336	17.4	228
23	Alzheimerß disease brain-derived amyloid-Emediated inhibition of LTP in vivo is prevented by immunotargeting cellular prion protein. <i>Journal of Neuroscience</i> , 2011 , 31, 7259-63	6.6	193
22	beta-Secretase cleavage is not required for generation of the intracellular C-terminal domain of the amyloid precursor family of proteins. <i>FEBS Journal</i> , 2010 , 277, 1503-18	5.7	21
21	Amyloid beta-protein dimers rapidly form stable synaptotoxic protofibrils. <i>Journal of Neuroscience</i> , 2010 , 30, 14411-9	6.6	199
20	The presence of sodium dodecyl sulphate-stable Abeta dimers is strongly associated with Alzheimer-type dementia. <i>Brain</i> , 2010 , 133, 1328-41	11.2	207
19	Alzheimerß disease: synaptic dysfunction and Abeta. <i>Molecular Neurodegeneration</i> , 2009 , 4, 48	19	318
18	A facile method for expression and purification of the Alzheimer® disease-associated amyloid beta-peptide. <i>FEBS Journal</i> , 2009 , 276, 1266-81	5.7	197
17	Soluble oligomers of amyloid Beta protein facilitate hippocampal long-term depression by disrupting neuronal glutamate uptake. <i>Neuron</i> , 2009 , 62, 788-801	13.9	698
16	Amyloid-beta protein dimers isolated directly from Alzheimerß brains impair synaptic plasticity and memory. <i>Nature Medicine</i> , 2008 , 14, 837-42	50.5	2779
15	Aggregation and catabolism of disease-associated intra-Abeta mutations: reduced proteolysis of AbetaA21G by neprilysin. <i>Neurobiology of Disease</i> , 2008 , 31, 442-50	7.5	76
14	Amyloid beta protein dimer-containing human CSF disrupts synaptic plasticity: prevention by systemic passive immunization. <i>Journal of Neuroscience</i> , 2008 , 28, 4231-7	6.6	256
13	Intracerebroventricular Administration of Amyloid Eprotein Oligomers Selectively Increases Dorsal Hippocampal Dialysate Glutamate Levels in the Awake Rat. <i>Sensors</i> , 2008 , 8, 7428-7437	3.8	19
12	Natural oligomers of the Alzheimer amyloid-beta protein induce reversible synapse loss by modulating an NMDA-type glutamate receptor-dependent signaling pathway. <i>Journal of Neuroscience</i> , 2007 , 27, 2866-75	6.6	1232
11	Certain inhibitors of synthetic amyloid beta-peptide (Abeta) fibrillogenesis block oligomerization of natural Abeta and thereby rescue long-term potentiation. <i>Journal of Neuroscience</i> , 2005 , 25, 2455-62	6.6	262
10	Amyloid beta protein immunotherapy neutralizes Abeta oligomers that disrupt synaptic plasticity in vivo. <i>Nature Medicine</i> , 2005 , 11, 556-61	50.5	443
9	Natural oligomers of the amyloid-beta protein specifically disrupt cognitive function. <i>Nature Neuroscience</i> , 2005 , 8, 79-84	25.5	1436
8	Deciphering the molecular basis of memory failure in Alzheimerß disease. <i>Neuron</i> , 2004 , 44, 181-93	13.9	1004
7	gamma-Secretase cleavage and binding to FE65 regulate the nuclear translocation of the intracellular C-terminal domain (ICD) of the APP family of proteins. <i>Biochemistry</i> , 2003 , 42, 6664-73	3.2	91

6	The Many Faces of Aβ: Structures and Activity. <i>Current Medicinal Chemistry Immunology, Endocrine & Metabolic Agents</i> , 2003 , 3, 277-291		18
5	Naturally secreted oligomers of amyloid beta protein potently inhibit hippocampal long-term potentiation in vivo. <i>Nature</i> , 2002 , 416, 535-9	50.4	3560
4	An improved method of preparing the amyloid beta-protein for fibrillogenesis and neurotoxicity experiments. <i>Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis</i> , 2000 , 7, 166-78	2.7	211
3	Protofibrillar intermediates of amyloid beta-protein induce acute electrophysiological changes and progressive neurotoxicity in cortical neurons. <i>Journal of Neuroscience</i> , 1999 , 19, 8876-84	6.6	873
2	Amyloid beta-protein fibrillogenesis. Detection of a protofibrillar intermediate. <i>Journal of Biological Chemistry</i> , 1997 , 272, 22364-72	5.4	859
1	Aggregation and metal-binding properties of mutant forms of the amyloid A beta peptide of Alzheimerß disease. <i>Journal of Neurochemistry</i> , 1996 , 66, 740-7	6	94