Jochen Walter

List of Publications by Year
in descending order

130 papers	17,970 citations	24978 57 h-index	16605 123 g-index
144 all docs	144 docs citations	144 times ranked	30646 citing authors

#	Article	IF	Citations
1	FTY720 decreases ceramides levels in the brain and prevents memory impairments in a mouse model of familial Alzheimerâ€™s disease expressing APOE4. Biomedicine and Pharmacotherapy, 2022, 152, 113240.	2.5	5
2	A reporter cell system for the triggering receptor expressed on myeloid cells 2 reveals differential effects of diseaseâ€associated variants on receptor signaling and activation by antibodies against the stalk region. Glia, 2021, 69, 1126-1139.	2.5	5
3	Wild-type sTREM2 blocks Aî² aggregation and neurotoxicity, but the Alzheimer's R47H mutant increases Aî² aggregation. Journal of Biological Chemistry, 2021, 296, 100631.	1.6	33
4	CERTL reduces C16 ceramide, amyloid-î² levels, and inflammation in a model of Alzheimerâ€™s disease. Alzheimer's Research and Therapy, 2021, 13, 45.	3.0	16
5	In vivo Characterization of Biochemical Variants of Amyloid-î² in Subjects with Idiopathic Normal Pressure Hydrocephalus and Alzheimerâ€™s Disease Neuropathological Change. Journal of Alzheimer's Disease, 2021, 80, 1003-1012.	1.2	3
6	Epigenetic and gene expression changes of neuronal cells from MSA patients are pronounced in enzymes for cell metabolism and calcium-regulated protein kinases. Acta Neuropathologica, 2021, 142, 781-783.	3.9	1
7	Differential interaction with <scp>TREM2</scp> modulates microglial uptake of modified Aî² species. Glia, 2021, 69, 2917-2932.	2.5	9
8	Carboxy-terminal fragment of amyloid precursor protein mediates lipid droplet accumulation upon î³-secretase inhibition. Biochemical and Biophysical Research Communications, 2021, 570, 137-142.	1.0	3
9	TREM2 modulates differential deposition of modified and non-modified Aî² species in extracellular plaques and intraneuronal deposits. Acta Neuropathologica Communications, 2021, 9, 168.	2.4	12
10	Effects of Sex, Age, and Apolipoprotein E Genotype on Brain Ceramides and Sphingosine-1-Phosphate in Alzheimerâ€™s Disease and Control Mice. Frontiers in Aging Neuroscience, 2021, 13, 765252.	1.7	7
11	A rare heterozygous <i>TREM2</i> coding variant identified in familial clustering of dementia affects an intrinsically disordered protein region and function of TREM2. Human Mutation, 2020, 41, 169-181.	1.1	4
12	Sphingolipids in Alzheimer's disease, how can we target them?. Advanced Drug Delivery Reviews, 2020, 159, 214-231.	6.6	53
13	Phosphorylated Aî² peptides in human Down syndrome brain and different Alzheimerâ€™s-like mouse models. Acta Neuropathologica Communications, 2020, 8, 118.	2.4	14
14	The coarse-grained plaque: a divergent Aî² plaque-type in early-onset Alzheimerâ€™s disease. Acta Neuropathologica, 2020, 140, 811-830.	3.9	45
15	Ceramide analog [18F]F-HPA-12 detects sphingolipid disbalance in the brain of Alzheimerâ€™s disease transgenic mice by functioning as a metabolic probe. Scientific Reports, 2020, 10, 19354.	1.6	9
16	Impact of the presence of Aî² N3pE and Aî² pSer8 in Aî² aggregates on the induction of Aî² seeding and spreading in the brains of APP23 mice. Alzheimer's and Dementia, 2020, 16, e038224.	0.4	0
17	A novel type of amyloidâ€beta plaques identified in earlyâ€onset AD. Alzheimer's and Dementia, 2020, 16, e040626.	0.4	0
18	Novel Phosphorylation-State Specific Antibodies Reveal Differential Deposition of Ser26 Phosphorylated Aî² Species in a Mouse Model of Alzheimerâ€™s Disease. Frontiers in Molecular Neuroscience, 2020, 13, 619639.	1.4	7

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19	Importance of Î³-secretase in the regulation of liver X receptor and cellular lipid metabolism. Life Science Alliance, 2020, 3, e201900521.	1.3	9
20	Implication of protein glycosylation impairment in cellular cholesterol accumulation caused by Presenilin deficiency. FASEB Journal, 2020, 34, 1-1.	0.2	0
21	TREM2 triggers microglial density and ageâ€related neuronal loss. Glia, 2019, 67, 539-550.	2.5	84
22	Dietary Sargassum fusiforme improves memory and reduces amyloid plaque load in an Alzheimerâ€™s disease mouse model. Scientific Reports, 2019, 9, 4908.	1.6	51
23	Presenilins and Î³-Secretase in Membrane Proteostasis. Cells, 2019, 8, 209.	1.8	45
24	Different aspects of Alzheimerâ€™s disease-related amyloid Î²-peptide pathology and their relationship to amyloid positron emission tomography imaging and dementia. Acta Neuropathologica Communications, 2019, 7, 178.	2.4	29
25	P3â€151: GAMMAâ€SECRETASE INHIBITION INDUCES LIPID DROPLET ACCUMULATION VIA APPâ€CTF ACCUMULATION. Alzheimer's and Dementia, 2018, 14, P1126.	0.4	0
26	3 .Neuropathologie und molekulare Mechanismen. , 2018, , 35-122.		1
27	Deposition of phosphorylated amyloidâ€Î² in brains of aged nonhuman primates and canines. Brain Pathology, 2018, 28, 427-430.	2.1	8
28	Modified amyloid variants in pathological subgroups of <i>Î²</i>â€amyloidosis. Annals of Clinical and Translational Neurology, 2018, 5, 815-831.	1.7	18
29	Pleiotropic Effect of Human ApoE4 on Cerebral Ceramide and Saturated Fatty Acid Levels. Journal of Alzheimer's Disease, 2017, 60, 769-781.	1.2	7
30	Altered Sphingolipid Balance in Capillary Cerebral Amyloid Angiopathy. Journal of Alzheimer's Disease, 2017, 60, 795-807.	1.2	26
31	Intramembranous processing by Î³â€secretase regulates reverse signaling of ephrinâ€B2 in migration of microglia. Glia, 2017, 65, 1103-1118.	2.5	13
32	Synthesis, Radiosynthesis, and Preliminary in vitro and in vivo Evaluation of the Fluorinated Ceramide Trafficking Inhibitor (HPA-12) for Brain Applications. Journal of Alzheimer's Disease, 2017, 60, 783-794.	1.2	11
33	Î³â€Secretase in microglia â€“ implications for neurodegeneration and neuroinflammation. Journal of Neurochemistry, 2017, 143, 445-454.	2.1	15
34	Microglia-derived ASC specks cross-seed amyloid-Î² in Alzheimerâ€™s disease. Nature, 2017, 552, 355-361.	13.7	664
35	[P3â€“164]: FUNCTIONAL CHARACTERIZATION OF A RARE GENETIC VARIANT IN PHOSPHOLIPASE CÎ³2 WHICH IS ASSOCIATED WITH A BENEFICIAL EFFECT ON THE PROGRESSION OF ALZHEIMER's DISEASE. Alzheimer's and Dementia, 2017, 13, P997.	0.4	0
36	Phosphorylation modifies the molecular stability of Î²-amyloid deposits. Nature Communications, 2016, 7, 11359.	5.8	70

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37	The intact Kunitz domain protects the amyloid precursor protein from being processed by matriptase-2. Biological Chemistry, 2016, 397, 777-790.	1.2	10
38	Phosphorylation of the amyloid Î²-peptide at Ser26 stabilizes oligomeric assembly and increases neurotoxicity. Acta Neuropathologica, 2016, 131, 525-537.	3.9	84
39	Phosphorylation Interferes with Maturation of Amyloid-Î² Fibrillar Structure in the N Terminus. Journal of Biological Chemistry, 2016, 291, 16059-16067.	1.6	22
40	Sphingosine 1-phosphate lyase ablation disrupts presynaptic architecture and function via an ubiquitin- proteasome mediated mechanism. Scientific Reports, 2016, 6, 37064.	1.6	43
41	Generation of aggregation prone N-terminally truncated amyloid Î² peptides by meprin Î² depends on the sequence specificity at the cleavage site. Molecular Neurodegeneration, 2016, 11, 19.	4.4	65
42	Functional involvement of Î³-secretase in signaling of the triggering receptor expressed on myeloid cells-2 (TREM2). Journal of Neuroinflammation, 2016, 13, 17.	3.1	28
43	Trehalose Alters Subcellular Trafficking and the Metabolism of the Alzheimer-associated Amyloid Precursor Protein. Journal of Biological Chemistry, 2016, 291, 10528-10540.	1.6	53
44	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	4.3	4,701
45	The Triggering Receptor Expressed on Myeloid Cells 2: A Molecular Link of Neuroinflammation and Neurodegenerative Diseases. Journal of Biological Chemistry, 2016, 291, 4334-4341.	1.6	61
46	P2-049: Functional characterization of a novel TREM2 coding variant linked to familial Alzheimer's disease. , 2015, 11, P500-P500.		2
47	Lithium Decreases Glial Fibrillary Acidic Protein in a Mouse Model of Alexander Disease. PLoS ONE, 2015, 10, e0138132.	1.1	16
48	Twenty Years of Presenilinsâ€"Important Proteins in Health and Disease. Molecular Medicine, 2015, 21, S41-S48.	1.9	5
49	Investigation of <scp>A</scp>Î² phosphorylated at serine 8 (p<scp>A</scp>Î²) in <scp>A</scp>lzheimer's disease, dementia with <scp>L</scp>ewy bodies and vascular dementia. Neuropathology and Applied Neurobiology, 2015, 41, 428-444.	1.8	16
50	Impact of amyloid Î² aggregate maturation on antibody treatment in APP23 mice. Acta Neuropathologica Communications, 2015, 3, 41.	2.4	13
51	Neuropathology and biochemistry of AÎ² and its aggregates in Alzheimerâ€™s disease. Acta Neuropathologica, 2015, 129, 167-182.	3.9	224
52	Serotonin stimulates secretion of exosomes from microglia cells. Glia, 2015, 63, 626-634.	2.5	160
53	Sphingosine-1-Phosphate: Boon and Bane for the Brain. Cellular Physiology and Biochemistry, 2014, 34, 148-157.	1.1	47
54	Turn Plasticity Distinguishes Different Modes of Amyloid-Î² Aggregation. Journal of the American Chemical Society, 2014, 136, 4913-4919.	6.6	39

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55	Interplay between phosphorylation and palmitoylation mediates plasma membrane targeting and sorting of GAP43. Molecular Biology of the Cell, 2014, 25, 3284-3299.	0.9	44
56	Biochemical stages of amyloid-Î² peptide aggregation and accumulation in the human brain and their association with symptomatic and pathologically preclinical Alzheimerâ€™s disease. Brain, 2014, 137, 887-903.	3.7	136
57	Deficiency of Sphingosine-1-phosphate Lyase Impairs Lysosomal Metabolism of the Amyloid Precursor Protein. Journal of Biological Chemistry, 2014, 289, 16761-16772.	1.6	50
58	GGA1 overexpression attenuates amyloidogenic processing of the amyloid precursor protein in Niemann-Pick type C cells. Biochemical and Biophysical Research Communications, 2014, 450, 160-165.	1.0	4
59	Early intraneuronal accumulation and increased aggregation of phosphorylated Abeta in a mouse model of Alzheimerâ€™s disease. Acta Neuropathologica, 2013, 125, 699-709.	3.9	79
60	The type of AÎ²-related neuronal degeneration differs between amyloid precursor protein (APP23) and amyloid Î²-peptide (APP48) transgenic mice. Acta Neuropathologica Communications, 2013, 1, 77.	2.4	12
61	APP Processing in Human Pluripotent Stem Cell-Derived Neurons Is Resistant to NSAID-Based Î³-Secretase Modulation. Stem Cell Reports, 2013, 1, 491-498.	2.3	58
62	Sequential Proteolytic Processing of the Triggering Receptor Expressed on Myeloid Cells-2 (TREM2) Protein by Ectodomain Shedding and Î³-Secretase-dependent Intramembranous Cleavage. Journal of Biological Chemistry, 2013, 288, 33027-33036.	1.6	236
63	Mutations in phospholipase DDHD2 cause autosomal recessive hereditary spastic paraplegia (SPG54). European Journal of Human Genetics, 2013, 21, 1214-1218.	1.4	63
64	Fishing for function â€“ distinct roles of Bace1 and Bace2 in Zebrafish development. Journal of Neurochemistry, 2013, 127, 435-437.	2.1	4
65	Cross-talk of membrane lipids and Alzheimer-related proteins. Molecular Neurodegeneration, 2013, 8, 34.	4.4	64
66	γ-Secretase, Apolipoprotein E and Cellular Cholesterol Metabolism. Current Alzheimer Research, 2012, 9, 189-199.	0.7	8
67	TLR2 Is a Primary Receptor for Alzheimerâ€™s Amyloid Î² Peptide To Trigger Neuroinflammatory Activation. Journal of Immunology, 2012, 188, 1098-1107.	0.4	346
68	Phosphorylation of Amyloid-Î² Peptide at Serine 8 Attenuates Its Clearance via Insulin-degrading and Angiotensin-converting Enzymes. Journal of Biological Chemistry, 2012, 287, 8641-8651.	1.6	64
69	Glycogen Synthase Kinase 3 Inhibition Promotes Lysosomal Biogenesis and Autophagic Degradation of the Amyloid-Î² Precursor Protein. Molecular and Cellular Biology, 2012, 32, 4410-4418.	1.1	147
70	Dispersible amyloid Î²-protein oligomers, protofibrils, and fibrils represent diffusible but not soluble aggregates: their role in neurodegeneration in amyloid precursor protein (APP) transgenic mice. Neurobiology of Aging, 2012, 33, 2641-2660.	1.5	50
71	Presenilin-1 L166P Mutant Human Pluripotent Stem Cellâ€“Derived Neurons Exhibit Partial Loss of Î³-Secretase Activity in Endogenous Amyloid-Î² Generation. American Journal of Pathology, 2012, 180, 2404-2416.	1.9	104
72	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	4.3	3,122

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73	Sphingolipids: Critical players in Alzheimerâ€™s disease. Progress in Lipid Research, 2012, 51, 378-393.	5.3	143
74	Statins in Unconventional Secretion of Insulin-Degrading Enzyme and Degradation of the Amyloid-Î² Peptide. Neurodegenerative Diseases, 2012, 10, 309-312.	0.8	22
75	BRI2 Protein Regulates Î²-Amyloid Degradation by Increasing Levels of Secreted Insulin-degrading Enzyme (IDE). Journal of Biological Chemistry, 2011, 286, 37446-37457.	1.6	37
76	Nitration of Tyrosine 10 Critically Enhances Amyloid Î² Aggregation and Plaque Formation. Neuron, 2011, 71, 833-844.	3.8	259
77	Extracellular phosphorylation of the amyloid Î²-peptide promotes formation of toxic aggregates during the pathogenesis of Alzheimer's disease. EMBO Journal, 2011, 30, 2255-2265.	3.5	160
78	Functional Relevance of a Novel SlyX Motif in Non-conventional Secretion of Insulin-degrading Enzyme. Journal of Biological Chemistry, 2011, 286, 22711-22715.	1.6	23
79	Sphingolipid Storage Affects Autophagic Metabolism of the Amyloid Precursor Protein and Promotes AÎ² Generation. Journal of Neuroscience, 2011, 31, 1837-1849.	1.7	82
80	Sphingolipid storage impairs autophagic clearance of Alzheimer-associated proteins. Autophagy, 2011, 7, 645-646.	4.3	31
81	Phosphorylation of amyloid beta (AÎ²) peptides â€" A trigger for formation of toxic aggregates in Alzheimer's disease. Aging, 2011, 3, 803-812.	1.4	142
82	Proteolytic processing of the serine protease matriptase-2: identification of the cleavage sites required for its autocatalytic release from the cell surface. Biochemical Journal, 2010, 430, 87-95.	1.7	56
83	Statins Promote the Degradation of Extracellular Amyloid Î²-Peptide by Microglia via Stimulation of Exosome-associated Insulin-degrading Enzyme (IDE) Secretion. Journal of Biological Chemistry, 2010, 285, 37405-37414.	1.6	176
84	Identification of Low Molecular Weight Pyroglutamate AÎ² Oligomers in Alzheimer Disease. Journal of Biological Chemistry, 2010, 285, 41517-41524.	1.6	91
85	Locus ceruleus controls Alzheimer's disease pathology by modulating microglial functions through norepinephrine. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 6058-6063.	3.3	408
86	Histone Deacetylase Inhibitor Valproic Acid Inhibits Cancer Cell Proliferation via Down-regulation of the Alzheimer Amyloid Precursor Protein. Journal of Biological Chemistry, 2010, 285, 10678-10689.	1.6	104
87	Casein Kinase 2 Dependent Phosphorylation of Neprilysin Regulates Receptor Tyrosine Kinase Signaling to Akt. PLoS ONE, 2010, 5, e13134.	1.1	22
88	Presenilin 1 Affects Focal Adhesion Site Formation and Cell Force Generation via c-Src Transcriptional and Posttranslational Regulation. Journal of Biological Chemistry, 2009, 284, 10138-10149.	1.6	16
89	CK2-dependent phosphorylation determines cellular localization and stability of ataxin-3. Human Molecular Genetics, 2009, 18, 3334-3343.	1.4	88
90	Adaptation of neuronal cells to chronic oxidative stress is associated with altered cholesterol and sphingolipid homeostasis and lysosomal function. Journal of Neurochemistry, 2009, 111, 669-682.	2.1	46

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91	Interactions between APP secretases and inflammatory mediators. Journal of Neuroinflammation, 2008, 5, 25.	3.1	144
92	Cerebral Small Vessel Disease-Induced Apolipoprotein E Leakage Is Associated With Alzheimer Disease and the Accumulation of Amyloid Î²-Protein in Perivascular Astrocytes. Journal of Neuropathology and Experimental Neurology, 2008, 67, 842-856.	0.9	70
93	Loss of Î³-Secretase Function Impairs Endocytosis of Lipoprotein Particles and Membrane Cholesterol Homeostasis. Journal of Neuroscience, 2008, 28, 12097-12106.	1.7	62
94	A Structural Switch of Presenilin 1 by Glycogen Synthase Kinase 3Î²-mediated Phosphorylation Regulates the Interaction with Î²-Catenin and Its Nuclear Signaling. Journal of Biological Chemistry, 2007, 282, 14083-14093.	1.6	26
95	Control of Amyloid-Î²-Peptide Generation by Subcellular Trafficking of the Î²-Amyloid Precursor Protein and Î²-Secretase. Neurodegenerative Diseases, 2006, 3, 247-254.	0.8	15
96	RNA aptamers selectively modulate protein recruitment to the cytoplasmic domain of Â-secretase BACE1 in vitro. Rna, 2006, 12, 1650-1660.	1.6	51
97	Nonsteroidal anti-inflammatory drugs repress Â-secretase gene promoter activity by the activation of PPARÂ. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 443-448.	3.3	365
98	Down-regulation of Endogenous Amyloid Precursor Protein Processing due to Cellular Aging. Journal of Biological Chemistry, 2006, 281, 2405-2413.	1.6	61
99	GGA1 Is Expressed in the Human Brain and Affects the Generation of Amyloid Â-Peptide. Journal of Neuroscience, 2006, 26, 12838-12846.	1.7	82
100	Modulation of Proteolytic Processing by Glycosphingolipids Generates Amyloid Î²-Peptide. , 2006, , 319-328.		1
101	Inhibition of Glycosphingolipid Biosynthesis Reduces Secretion of the Î²-Amyloid Precursor Protein and Amyloid Î²-Peptide*[boxs]. Journal of Biological Chemistry, 2005, 280, 28110-28117.	1.6	115
102	GGA proteins regulate retrograde transport of BACE1 from endosomes to the trans-Golgi network. Molecular and Cellular Neurosciences, 2005, 29, 453-461.	1.0	117
103	Focal glial activation coincides with increased BACE1 activation and precedes amyloid plaque deposition in APP[V717I] transgenic mice. Journal of Neuroinflammation, 2005, 2, 22.	3.1	257
104	Phosphorylation of Presenilin 1 at the Caspase Recognition Site Regulates Its Proteolytic Processing and the Progression of Apoptosis. Journal of Biological Chemistry, 2004, 279, 1585-1593.	1.6	55
105	Identification of a Î²-Secretase Activity, Which Truncates Amyloid Î²-Peptide after Its Presenilin-dependent Generation. Journal of Biological Chemistry, 2003, 278, 5531-5538.	1.6	62
106	Presenilin-dependent Intramembrane Proteolysis of CD44 Leads to the Liberation of Its Intracellular Domain and the Secretion of an AÎ²-like Peptide. Journal of Biological Chemistry, 2002, 277, 44754-44759.	1.6	253
107	Apical Sorting of Î²-Secretase Limits Amyloid Î²-Peptide Production. Journal of Biological Chemistry, 2002, 277, 5637-5643.	1.6	64
108	Secretases as targets for beta-amyloid lowering drugs. Drug Development Research, 2002, 56, 201-210.	1.4	10

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109	A non-amyloidogenic function of BACE-2 in the secretory pathway. Journal of Neurochemistry, 2002, 81, 1011-1020.	2.1	99
110	The cell biology of Alzheimer's disease: uncovering the secrets of secretases. Current Opinion in Neurobiology, 2001, 11, 585-590.	2.0	163
111	Phosphorylation Regulates Intracellular Trafficking of Î²-Secretase. Journal of Biological Chemistry, 2001, 276, 14634-14641.	1.6	248
112	The Phosphorylation of Presenilin Proteins. , 2000, 32, 317-332.		2
113	Brain Expression of Presenilins in Sporadic and Early-onset, Familial Alzheimerâ€™s Disease. Molecular Medicine, 2000, 6, 878-891.	1.9	35
114	Separation of presenilin function in amyloid beta -peptide generation and endoproteolysis of Notch. Proceedings of the National Academy of Sciences of the United States of America, 2000, 97, 5913-5918.	3.3	84
115	Phosphorylation of the Î²-Amyloid Precursor Protein at the Cell Surface by Ectocasein Kinases 1 and 2. Journal of Biological Chemistry, 2000, 275, 23523-23529.	1.6	45
116	Maturation and Pro-peptide Cleavage of Î²-Secretase. Journal of Biological Chemistry, 2000, 275, 30849-30854.	1.6	229
117	Constitutive Phosphorylation of the Parkinson's Disease Associated Î±-Synuclein. Journal of Biological Chemistry, 2000, 275, 390-397.	1.6	450
118	A Loss of Function Mutant of the Presenilin Homologue SEL-12 Undergoes Aberrant Endoproteolysis in Caenorhabditis elegans and Increases AÎ²42 Generation in Human Cells. Journal of Biological Chemistry, 2000, 275, 40925-40932.	1.6	36
119	Phosphorylation of presenilin-2 regulates its cleavage by caspases and retards progression of apoptosis. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 1391-1396.	3.3	116
120	Zebrafish (Danio rerio) Presenilin Promotes Aberrant Amyloid Î²-Peptide Production and Requires a Critical Aspartate Residue for Its Function in Amyloidogenesisâ€. Biochemistry, 1999, 38, 13602-13609.	1.2	118
121	Alzheimer's Disease Associated Presenilin-1 Holoprotein and Its 18â^'20 kDa C-Terminal Fragment Are Death Substrates for Proteases of the Caspase Familyâ€. Biochemistry, 1998, 37, 2263-2270.	1.2	69
122	Proteolytic Fragments of the Alzheimer's Disease Associated Presenilins-1 and -2 Are Phosphorylated in Vivo by Distinct Cellular Mechanismsâ€. Biochemistry, 1998, 37, 5961-5967.	1.2	60
123	Truncated presenilin 2 derived from differentially spliced mRNAs does not affect the ratio of amyloid Î²-peptide 1-42/1-40. NeuroReport, 1998, 9, 3293-3299.	0.6	17
124	Mutant Presenilin 2 Transgenic Mouse: Effect on an Ageâ€Dependent Increase of Amyloid Î²â€Protein 42 in the Brain. Journal of Neurochemistry, 1998, 71, 313-322.	2.1	81
125	Ectodomain Phosphorylation of Î²-Amyloid Precursor Protein at Two Distinct Cellular Locations. Journal of Biological Chemistry, 1997, 272, 1896-1903.	1.6	69
126	The presenilin 2 mutation (N141I) linked to familial Alzheimer disease (Volga German families) increases the secretion of amyloid Â protein ending at the 42nd (or 43rd) residue. Proceedings of the National Academy of Sciences of the United States of America, 1997, 94, 2025-2030.	3.3	378

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127	Proteolytic processing of the Alzheimer disease-associated presenilin-1 generates an in vivo substrate for protein kinase C. Proceedings of the National Academy of Sciences of the United States of America, 1997, 94, 5349-5354.	3.3	115
128	Cellular Expression and Proteolytic Processing of Presenilin Proteins Is Developmentally Regulated During Neuronal Differentiation. Journal of Neurochemistry, 1997, 69, 2432-2440.	2.1	79
129	The Alzheimerâ€™s Disease-Associated Presenilins Are Differentially Phosphorylated Proteins Located Predominantly within the Endoplasmic Reticulum. Molecular Medicine, 1996, 2, 673-691.	1.9	230
130	Induced Release of Cell Surface Protein Kinase Yields CK1- and CK2-like Enzymes in Tandem. Journal of Biological Chemistry, 1996, 271, 111-119.	1.6	60