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	Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition). Autophagy, 2016, 12, 1-222.	4.3	4,701
2	Guidelines for the use and interpretation of assays for monitoring autophagy. Autophagy, 2012, 8, 445-544.	4.3	3,122
3	Microglia-derived ASC specks cross-seed amyloid-Î² in Alzheimerâ€™s disease. Nature, 2017, 552, 355-361.	13.7	664
4	Constitutive Phosphorylation of the Parkinson's Disease Associated Î±-Synuclein. Journal of Biological Chemistry, 2000, 275, 390-397.	1.6	450
5	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
6	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
7	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
8	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
9	Nitration of Tyrosine 10 Critically Enhances Amyloid Î² Aggregation and Plaque Formation. Neuron, 2011, 71, 833-844.	3.8	259
10	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
11	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
12	Phosphorylation Regulates Intracellular Trafficking of Î²-Secretase. Journal of Biological Chemistry, 2001, 276, 14634-14641.	1.6	248
13	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
14	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
15	Maturation and Pro-peptide Cleavage of Î²-Secretase. Journal of Biological Chemistry, 2000, 275, 30849-30854.	1.6	229
16	Neuropathology and biochemistry of AÎ² and its aggregates in Alzheimerâ€™s disease. Acta Neuropathologica, 2015, 129, 167-182.	3.9	224
17	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
18	The cell biology of Alzheimer's disease: uncovering the secrets of secretases. Current Opinion in Neurobiology, 2001, 11, 585-590.	2.0	163

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19	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
20	Serotonin stimulates secretion of exosomes from microglia cells. Glia, 2015, 63, 626-634.	2.5	160
21	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
22	Interactions between APP secretases and inflammatory mediators. Journal of Neuroinflammation, 2008, 5, 25.	3.1	144
23	Sphingolipids: Critical players in Alzheimerâ€™s disease. Progress in Lipid Research, 2012, 51, 378-393.	5.3	143
24	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
25	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
26	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
27	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
28	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
29	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
30	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
31	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
32	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
33	A non-amyloidogenic function of BACE-2 in the secretory pathway. Journal of Neurochemistry, 2002, 81, 1011-1020.	2.1	99
34	Identification of Low Molecular Weight Pyroglutamate AÎ² Oligomers in Alzheimer Disease. Journal of Biological Chemistry, 2010, 285, 41517-41524.	1.6	91
35	CK2-dependent phosphorylation determines cellular localization and stability of ataxin-3. Human Molecular Genetics, 2009, 18, 3334-3343.	1.4	88
36	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

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37	Phosphorylation of the amyloid Î²-peptide at Ser26 stabilizes oligomeric assembly and increases neurotoxicity. Acta Neuropathologica, 2016, 131, 525-537.	3.9	84
38	TREM2 triggers microglial density and ageâ€related neuronal loss. Glia, 2019, 67, 539-550.	2.5	84
39	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
40	Sphingolipid Storage Affects Autophagic Metabolism of the Amyloid Precursor Protein and Promotes AÎ² Generation. Journal of Neuroscience, 2011, 31, 1837-1849.	1.7	82
41	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
42	Cellular Expression and Proteolytic Processing of Presenilin Proteins Is Developmentally Regulated During Neuronal Differentiation. Journal of Neurochemistry, 1997, 69, 2432-2440.	2.1	79
43	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
44	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
45	Phosphorylation modifies the molecular stability of Î²-amyloid deposits. Nature Communications, 2016, 7, 11359.	5.8	70
46	Ectodomain Phosphorylation of Î²-Amyloid Precursor Protein at Two Distinct Cellular Locations. Journal of Biological Chemistry, 1997, 272, 1896-1903.	1.6	69
47	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
48	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
49	Apical Sorting of Î²-Secretase Limits Amyloid Î²-Peptide Production. Journal of Biological Chemistry, 2002, 277, 5637-5643.	1.6	64
50	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
51	Cross-talk of membrane lipids and Alzheimer-related proteins. Molecular Neurodegeneration, 2013, 8, 34.	4.4	64
52	Mutations in phospholipase DDHD2 cause autosomal recessive hereditary spastic paraplegia (SPG54). European Journal of Human Genetics, 2013, 21, 1214-1218.	1.4	63
53	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
54	Loss of Î³-Secretase Function Impairs Endocytosis of Lipoprotein Particles and Membrane Cholesterol Homeostasis. Journal of Neuroscience, 2008, 28, 12097-12106.	1.7	62

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55	Down-regulation of Endogenous Amyloid Precursor Protein Processing due to Cellular Aging. Journal of Biological Chemistry, 2006, 281, 2405-2413.	1.6	61
56	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
57	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
58	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
59	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
60	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
61	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
62	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
63	Sphingolipids in Alzheimer's disease, how can we target them?. Advanced Drug Delivery Reviews, 2020, 159, 214-231.	6.6	53
64	RNA aptamers selectively modulate protein recruitment to the cytoplasmic domain of Â-secretase BACE1 in vitro. Rna, 2006, 12, 1650-1660.	1.6	51
65	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
66	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
67	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
68	Sphingosine-1-Phosphate: Boon and Bane for the Brain. Cellular Physiology and Biochemistry, 2014, 34, 148-157.	1.1	47
69	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
70	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
71	Presenilins and Î³-Secretase in Membrane Proteostasis. Cells, 2019, 8, 209.	1.8	45
72	The coarse-grained plaque: a divergent AÎ² plaque-type in early-onset Alzheimerâ€™s disease. Acta Neuropathologica, 2020, 140, 811-830.	3.9	45

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73	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
74	Sphingosine 1-phosphate lyase ablation disrupts presynaptic architecture and function via an ubiquitin- proteasome mediated mechanism. Scientific Reports, 2016, 6, 37064.	1.6	43
75	Turn Plasticity Distinguishes Different Modes of Amyloid-Î² Aggregation. Journal of the American Chemical Society, 2014, 136, 4913-4919.	6.6	39
76	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
77	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
78	Brain Expression of Presenilins in Sporadic and Early-onset, Familial Alzheimerâ€™s Disease. Molecular Medicine, 2000, 6, 878-891.	1.9	35
79	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
80	Sphingolipid storage impairs autophagic clearance of Alzheimer-associated proteins. Autophagy, 2011, 7, 645-646.	4.3	31
81	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
82	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
83	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
84	Altered Sphingolipid Balance in Capillary Cerebral Amyloid Angiopathy. Journal of Alzheimer's Disease, 2017, 60, 795-807.	1.2	26
85	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
86	Statins in Unconventional Secretion of Insulin-Degrading Enzyme and Degradation of the Amyloid-Î² Peptide. Neurodegenerative Diseases, 2012, 10, 309-312.	0.8	22
87	Phosphorylation Interferes with Maturation of Amyloid-Î² Fibrillar Structure in the N Terminus. Journal of Biological Chemistry, 2016, 291, 16059-16067.	1.6	22
88	Casein Kinase 2 Dependent Phosphorylation of Neprilysin Regulates Receptor Tyrosine Kinase Signaling to Akt. PLoS ONE, 2010, 5, e13134.	1.1	22
89	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
90	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

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91	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
92	Lithium Decreases Glial Fibrillary Acidic Protein in a Mouse Model of Alexander Disease. PLoS ONE, 2015, 10, e0138132.	1.1	16
93	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
94	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
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	Î³â€Secretase in microglia â€“ implications for neurodegeneration and neuroinflammation. Journal of Neurochemistry, 2017, 143, 445-454.	2.1	15
97	Phosphorylated AÎ² peptides in human Down syndrome brain and different Alzheimerâ€™s-like mouse models. Acta Neuropathologica Communications, 2020, 8, 118.	2.4	14
98	Impact of amyloid Î² aggregate maturation on antibody treatment in APP23 mice. Acta Neuropathologica Communications, 2015, 3, 41.	2.4	13
99	Intramembranous processing by Î³â€secretase regulates reverse signaling of ephrinâ€B2 in migration of microglia. Glia, 2017, 65, 1103-1118.	2.5	13
100	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
101	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
102	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
103	Secretases as targets for beta-amyloid lowering drugs. Drug Development Research, 2002, 56, 201-210.	1.4	10
104	The intact Kunitz domain protects the amyloid precursor protein from being processed by matriptase-2. Biological Chemistry, 2016, 397, 777-790.	1.2	10
105	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
106	Differential interaction with <scp>TREM2</scp> modulates microglial uptake of modified AÎ² species. Glia, 2021, 69, 2917-2932.	2.5	9
107	Importance of Î³-secretase in the regulation of liver X receptor and cellular lipid metabolism. Life Science Alliance, 2020, 3, e201900521.	1.3	9
108	γ-Secretase, Apolipoprotein E and Cellular Cholesterol Metabolism. Current Alzheimer Research, 2012, 9, 189-199.	0.7	8

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109	Deposition of phosphorylated amyloidâ€î² in brains of aged nonhuman primates and canines. Brain Pathology, 2018, 28, 427-430.	2.1	8
110	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
111	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
112	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
113	Twenty Years of Presenilinsâ€"Important Proteins in Health and Disease. Molecular Medicine, 2015, 21, S41-S48.	1.9	5
114	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
115	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
116	Fishing for function â€" distinct roles of Bace1 and Bace2 in Zebrafish development. Journal of Neurochemistry, 2013, 127, 435-437.	2.1	4
117	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
118	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
119	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
120	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
121	The Phosphorylation of Presenilin Proteins. , 2000, 32, 317-332.		2
122	P2-049: Functional characterization of a novel TREM2 coding variant linked to familial Alzheimer's disease. , 2015, 11, P500-P500.		2
123	3 .Neuropathologie und molekulare Mechanismen. , 2018, , 35-122.		1
124	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
125	Modulation of Proteolytic Processing by Glycosphingolipids Generates Amyloid î²-Peptide. , 2006, , 319-328.		1
126	[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

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127	P3â€151: GAMMAâ€SECRETASE INHIBITION INDUCES LIPID DROPLET ACCUMULATION VIA APPâ€CTF ACCUMULATION. Alzheimer's and Dementia, 2018, 14, P1126.	0.4	0
128	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
129	A novel type of amyloidâ€beta plaques identified in earlyâ€onset AD. Alzheimer's and Dementia, 2020, 16, e040626.	0.4	0
130	Implication of protein glycosylation impairment in cellular cholesterol accumulation caused by Presenilin deficiency. FASEB Journal, 2020, 34, 1-1.	0.2	0