Taisuke Tomita

List of Publications by Year in descending order

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180 papers	14,624 citations	24978 57 h-index	20307 116 g-index
217 all docs	217 docs citations	217 times ranked	13813 citing authors

#	Article	IF	Citations
1	APP Processing and Synaptic Function. Neuron, 2003, 37, 925-937.	3.8	1,423
2	High performance plasma amyloid-Î² biomarkers for Alzheimerâ€™s disease. Nature, 2018, 554, 249-254.	13.7	1,180
3	AMPAR Removal Underlies AÎ²-Induced Synaptic Depression and Dendritic Spine Loss. Neuron, 2006, 52, 831-843.	3.8	920
4	The role of presenilin cofactors in the Î³-secretase complex. Nature, 2003, 422, 438-441.	13.7	839
5	Glial cytoplasmic inclusions in white matter oligodendrocytes of multiple system atrophy brains contain insoluble ?-synuclein. Annals of Neurology, 1998, 44, 415-422.	2.8	633
6	Structures of Î±-synuclein filaments from multiple system atrophy. Nature, 2020, 585, 464-469.	13.7	446
7	Mechanism of Ca2+ Disruption in Alzheimer's Disease by Presenilin Regulation of InsP3 Receptor Channel Gating. Neuron, 2008, 58, 871-883.	3.8	426
8	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
9	Effects of PS1 Deficiency on Membrane Protein Trafficking in Neurons. Neuron, 1998, 21, 1213-1221.	3.8	359
10	Neprilysin Degrades Both Amyloid Î² Peptides 1â€“40 and 1â€“42 Most Rapidly and Efficiently among Thiorphan- and Phosphoramidon-sensitive Endopeptidases. Journal of Biological Chemistry, 2001, 276, 21895-21901.	1.6	282
11	A Loss of Function Mutation of Presenilin-2 Interferes with Amyloid Î²-Peptide Production and Notch Signaling. Journal of Biological Chemistry, 1999, 274, 28669-28673.	1.6	279
12	Presenilins mediate a dual intramembranous gamma-secretase cleavage of Notch-1. EMBO Journal, 2002, 21, 5408-5416.	3.5	214
13	A presenilin dimer at the core of the Â-secretase enzyme: Insights from parallel analysis of Notch 1 and APP proteolysis. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 13075-13080.	3.3	203
14	Activity-Dependent Proteolytic Cleavage of Neuroligin-1. Neuron, 2012, 76, 410-422.	3.8	179
15	C-terminal Fragment of Presenilin Is the Molecular Target of a Dipeptidic Î³-Secretase-specific Inhibitor DAPT (N-[N-(3,5-Difluorophenacetyl)-L-alanyl]-S-phenylglycine t-Butyl Ester). Journal of Biological Chemistry, 2006, 281, 14670-14676.	1.6	174
16	Sulindac Sulfide Is a Noncompetitive Î³-Secretase Inhibitor That Preferentially Reduces AÎ²42 Generation. Journal of Biological Chemistry, 2003, 278, 18664-18670.	1.6	172
17	Amyloid Precursor Proteins Inhibit Heme Oxygenase Activity and Augment Neurotoxicity in Alzheimer's Disease. Neuron, 2000, 28, 461-473.	3.8	168
18	BACE1 Activity Is Modulated by Cell-Associated Sphingosine-1-Phosphate. Journal of Neuroscience, 2011, 31, 6850-6857.	1.7	157

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19	Structure of the Catalytic Pore of Â-Secretase Probed by the Accessibility of Substituted Cysteines. Journal of Neuroscience, 2006, 26, 12081-12088.	1.7	147
20	Molecular Cloning and Characterization of CALP/KChIP4, a Novel EF-hand Protein Interacting with Presenilin 2 and Voltage-gated Potassium Channel Subunit Kv4. Journal of Biological Chemistry, 2002, 277, 14965-14975.	1.6	140
21	The C-Terminal PAL Motif and Transmembrane Domain 9 of Presenilin 1 Are Involved in the Formation of the Catalytic Pore of the Â-Secretase. Journal of Neuroscience, 2008, 28, 6264-6271.	1.7	140
22	Presenilin-dependent Î³-secretase activity mediates the intramembranous cleavage of CD44. Oncogene, 2003, 22, 1511-1516.	2.6	139
23	Variant Alzheimer's disease with spastic paraparesis and cotton wool plaques is caused by PS-1 mutations that lead to exceptionally high amyloid-? concentrations. Annals of Neurology, 2000, 48, 806-808.	2.8	135
24	Enhancement of amyloid Î² 42 secretion by 28 different presenilin 1 mutations of familial Alzheimer's disease. Neuroscience Letters, 1999, 265, 61-63.	1.0	129
25	Association of active Î³-secretase complex with lipid rafts. Journal of Lipid Research, 2005, 46, 904-912.	2.0	127
26	A Noncompetitive BACE1 Inhibitor TAK-070 Ameliorates AÎ² Pathology and Behavioral Deficits in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2010, 30, 11157-11166.	1.7	126
27	Secretase inhibitors and modulators for Alzheimerâ€™s disease treatment. Expert Review of Neurotherapeutics, 2009, 9, 661-679.	1.4	122
28	Attenuation of the Aggregation and Neurotoxicity of Amyloidâ€Î² Peptides by Catalytic Photooxygenation. Angewandte Chemie - International Edition, 2014, 53, 1382-1385.	7.2	111
29	Phenylpiperidine-type Î³-secretase modulators target the transmembrane domain 1 of presenilin 1. EMBO Journal, 2011, 30, 4815-4824.	3.5	105
30	C Terminus of Presenilin Is Required for Overproduction of Amyloidogenic AÎ²42 through Stabilization and Endoproteolysis of Presenilin. Journal of Neuroscience, 1999, 19, 10627-10634.	1.7	104
31	Pen-2 Is Incorporated into the Î³-Secretase Complex through Binding to Transmembrane Domain 4 of Presenilin 1*. Journal of Biological Chemistry, 2005, 280, 41967-41975.	1.6	101
32	Seeded assembly inÂvitro does not replicate the structures of Î±â€synuclein filaments from multiple system atrophy. FEBS Open Bio, 2021, 11, 999-1013.	1.0	95
33	Three-dimensional structure of the Î³-secretase complex. Biochemical and Biophysical Research Communications, 2006, 343, 525-534.	1.0	92
34	Application of boronated anti-CEA immunoliposome to tumour cell growth inhibition in in vitro boron neutron capture therapy model. British Journal of Cancer, 1991, 63, 522-526.	2.9	90
35	Divergent Synthesis of Multifunctional Molecular Probes To Elucidate the Enzyme Specificity of Dipeptidic Î³-Secretase Inhibitors. ACS Chemical Biology, 2007, 2, 408-418.	1.6	87
36	The First Proline of PALP Motif at the C Terminus of Presenilins Is Obligatory for Stabilization, Complex Formation, and Î³-Secretase Activities of Presenilins. Journal of Biological Chemistry, 2001, 276, 33273-33281.	1.6	81

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37	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
38	Aph-1 Contributes to the Stabilization and Trafficking of the Î³-Secretase Complex through Mechanisms Involving Intermolecular and Intramolecular Interactions. Journal of Biological Chemistry, 2005, 280, 12967-12975.	1.6	79
39	Inhibition of Î³-Secretase Activity by Helical Î²-Peptide Foldamers. Journal of the American Chemical Society, 2009, 131, 7353-7359.	6.6	78
40	Decreased CALM expression reduces AÎ²42 to total AÎ² ratio through clathrin-mediated endocytosis of Î³-secretase. Nature Communications, 2014, 5, 3386.	5.8	78
41	Caveolin-3 Upregulation Activates Î²-Secretaseâ€“Mediated Cleavage of the Amyloid Precursor Protein in Alzheimerâ€™s Disease. Journal of Neuroscience, 1999, 19, 6538-6548.	1.7	77
42	Selective Reconstitution and Recovery of Functional Î³-Secretase Complex on Budded Baculovirus Particles. Journal of Biological Chemistry, 2004, 279, 38040-38046.	1.6	77
43	FTY720/Fingolimod, a Sphingosine Analogue, Reduces Amyloid-Î² Production in Neurons. PLoS ONE, 2013, 8, e64050.	1.1	75
44	Molecular Dissection of Domains in Mutant Presenilin 2 That Mediate Overproduction of Amyloidogenic Forms of Amyloid Î² Peptides. Journal of Biological Chemistry, 1998, 273, 21153-21160.	1.6	74
45	The intracellular domain of the amyloid precursor protein (AICD) enhances the p53-mediated apoptosis. Biochemical and Biophysical Research Communications, 2006, 351, 57-63.	1.0	72
46	Both Notch1 and Notch2 contribute to the regulation of melanocyte homeostasis. Pigment Cell and Melanoma Research, 2008, 21, 70-78.	1.5	72
47	Allosteric regulation of Î³-secretase activity by a phenylimidazole-type Î³-secretase modulator. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 10544-10549.	3.3	72
48	Presenilin-dependent intramembrane cleavage of ephrin-B1. Molecular Neurodegeneration, 2006, 1, 2.	4.4	70
49	Evidence That Intramolecular Associations between Presenilin Domains Are Obligatory for Endoproteolytic Processing. Journal of Biological Chemistry, 1999, 274, 13818-13823.	1.6	69
50	BIN1 regulates BACE1 intracellular trafficking and amyloid-Î² production. Human Molecular Genetics, 2016, 25, ddw146.	1.4	67
51	A Role for Presenilin 1 in Regulating the Delivery of Amyloid Precursor Protein to the Cell Surface. Neurobiology of Disease, 2002, 11, 64-82.	2.1	65
52	AÎ²42 Overproduction Associated with Structural Changes in the Catalytic Pore of Î³-Secretase. Journal of Biological Chemistry, 2007, 282, 12388-12396.	1.6	65
53	Neutralization of the Î³-secretase activity by monoclonal antibody against extracellular domain of nicastrin. Oncogene, 2012, 31, 787-798.	2.6	65
54	Familial Alzheimer's Disease-Linked Mutations at Val717of Amyloid Precursor Protein Are Specific for the Increased Secretion of AÎ²42(43). Biochemical and Biophysical Research Communications, 1996, 227, 730-735.	1.0	64

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55	Imbalance of Clara cell-mediated homeostatic inflammation is involved in lung metastasis. Oncogene, 2011, 30, 3429-3439.	2.6	63
56	The Nonconserved Hydrophilic Loop Domain of Presenilin (PS) Is Not Required for PS Endoproteolysis or Enhanced AÎ²42 Production Mediated by Familial Early Onset Alzheimer's Disease-linked PS Variants. Journal of Biological Chemistry, 2000, 275, 17136-17142.	1.6	61
57	S-Palmitoylation of Î³-Secretase Subunits Nicastrin and APH-1. Journal of Biological Chemistry, 2009, 284, 1373-1384.	1.6	61
58	Notch1 Signaling Influences V2 Interneuron and Motor Neuron Development in the Spinal Cord. Developmental Neuroscience, 2006, 28, 102-117.	1.0	60
59	Naphthyl and Coumarinyl Biarylpiperazine Derivatives as Highly Potent Human Î²-Secretase Inhibitors. Design, Synthesis, and Enzymatic BACE-1 and Cell Assays. Journal of Medicinal Chemistry, 2006, 49, 4275-4285.	2.9	60
60	Functional Analysis of the Transmembrane Domains of Presenilin 1. Journal of Biological Chemistry, 2010, 285, 19738-19746.	1.6	60
61	ComplexN-glycosylated form of nicastrin is stabilized and selectively bound to presenilin fragments. FEBS Letters, 2002, 520, 117-121.	1.3	59
62	Near-Infrared Photoactivatable Oxygenation Catalysts of Amyloid Peptide. CheM, 2018, 4, 807-820.	5.8	59
63	Targeted introduction of V642I mutation in amyloid precursor protein gene causes functional abnormality resembling early stage of Alzheimer's disease in aged mice. European Journal of Neuroscience, 2004, 19, 2826-2838.	1.2	58
64	Participation of Transmembrane Domain 1 of Presenilin 1 in the Catalytic Pore Structure of the Â-Secretase. Journal of Neuroscience, 2010, 30, 15943-15950.	1.7	56
65	Molecular mechanism of intramembrane proteolysis by Î³-secretase. Journal of Biochemistry, 2014, 156, 195-201.	0.9	56
66	Structural Interactions between Inhibitor and Substrate Docking Sites Give Insight into Mechanisms of Human PS1 Complexes. Structure, 2014, 22, 125-135.	1.6	56
67	Cell-free methods to produce structurally intact mammalian membrane proteins. Scientific Reports, 2016, 6, 30442.	1.6	56
68	Primary cultures of neuronal and non-neuronal rat brain cells secrete similar proportions of amyloid Î² peptides ending at AÎ²40 and AÎ²42. NeuroReport, 1999, 10, 2965-2969.	0.6	55
69	Subcellular Compartment and Molecular Subdomain of Î²-Amyloid Precursor Protein Relevant to the AÎ²42-promoting Effects of Alzheimer Mutant Presenilin 2. Journal of Biological Chemistry, 2001, 276, 21678-21685.	1.6	52
70	Biochemical Characterization of the Drosophila Wingless Signaling Pathway Based on RNA Interference. Molecular and Cellular Biology, 2004, 24, 2012-2024.	1.1	52
71	Structural Biology of Presenilins and Signal Peptide Peptidases. Journal of Biological Chemistry, 2013, 288, 14673-14680.	1.6	50
72	Mouse Models of Alzheimerâ€™s Disease. Frontiers in Molecular Neuroscience, 0, 15, .	1.4	50

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73	Cooperative Roles of Hydrophilic Loop 1 and the C-Terminus of Presenilin 1 in the Substrate-Gating Mechanism of Î³-Secretase. Journal of Neuroscience, 2015, 35, 2646-2656.	1.7	48
74	Molecular Cloning and Expression of the Novel Splice Variants of K+ Channel-Interacting Protein 2. Biochemical and Biophysical Research Communications, 2001, 282, 96-102.	1.0	46
75	Highly efficient synthesis of medium-sized lactams via intramolecular Staudingerâ€“aza-Wittig reaction of Î‰-azido pentafluorophenyl ester: synthesis and biological evaluation of LY411575 analogues. Tetrahedron Letters, 2004, 45, 2323-2326.	0.7	46
76	Presenilin 2 regulates the systolic function of heart by modulating Ca 2+ signaling. FASEB Journal, 2005, 19, 2069-2071.	0.2	44
77	Molecular Cloning of a Novel Basic Helix-Loop-Helix Protein from the Rat Brain. Biochemical and Biophysical Research Communications, 1996, 219, 526-530.	1.0	42
78	Catalytic photooxygenation degrades brain AÎ² in vivo. Science Advances, 2021, 7, .	4.7	42
79	The Mechanism of Î³-Secretase Activities through High Molecular Weight Complex Formation of Presenilins Is Conserved inDrosophila melanogaster and Mammals. Journal of Biological Chemistry, 2002, 277, 50198-50205.	1.6	40
80	Loss of kallikreinâ€related peptidase 7 exacerbates amyloid pathology in Alzheimer's disease model mice. EMBO Molecular Medicine, 2018, 10, .	3.3	39
81	Nr <scp>CAM</scp> is a marker for substrateâ€selective activation of <scp>ADAM</scp> 10 in Alzheimer's disease. EMBO Molecular Medicine, 2019, 11, .	3.3	38
82	The presenilin 2 loop domain interacts with the mu-calpain C-terminal region.. International Journal of Molecular Medicine, 1998, 1, 797-9.	1.8	36
83	Cerebrospinal fluid levels of amyloid Î²-peptide1â€“42, but not tau have positive correlation with brain glucose metabolism in humans. Neuroscience Letters, 1999, 273, 203-207.	1.0	35
84	γ-Secretase as a Therapeutic Target for Treatment of Alzheimers Disease. Current Pharmaceutical Design, 2006, 12, 661-670.	0.9	34
85	Dysregulated Metabolism of the Amyloidâ€Î² Protein and Therapeutic Approaches in Alzheimer Disease. Journal of Cellular Biochemistry, 2017, 118, 4183-4190.	1.2	34
86	Genetic Risk Factors for Alzheimer Disease: Emerging Roles of Microglia in Disease Pathomechanisms. Advances in Experimental Medicine and Biology, 2019, 1118, 83-116.	0.8	34
87	Solid-phase synthesis of photoaffinity probes: highly efficient incorporation of biotin-tag and cross-linking groups. Chemical Communications, 2003, , 2244.	2.2	33
88	Synthesis of biotinylated photoaffinity probes based on arylsulfonamide Î³-secretase inhibitors. Bioorganic and Medicinal Chemistry Letters, 2006, 16, 4184-4189.	1.0	31
89	The crystal structure of mouse LC3B in complex with the FYCO1 LIR reveals the importance of the flanking region of the LIR motif. Acta Crystallographica Section F, Structural Biology Communications, 2017, 73, 130-137.	0.4	31
90	Memantine reduces the production of amyloid-Î² peptides through modulation of amyloid precursor protein trafficking. European Journal of Pharmacology, 2017, 798, 16-25.	1.7	31

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91	Aberrant proteolytic processing and therapeutic strategies in Alzheimer disease. Advances in Biological Regulation, 2017, 64, 33-38.	1.4	30
92	Comparison of Presenilin 1 and Presenilin 2 Î³-Secretase Activities Using a Yeast Reconstitution System. Journal of Biological Chemistry, 2011, 286, 44569-44575.	1.6	29
93	The inhibition of gamma-secretase as a therapeutic approach to Alzheimer's disease. Drug News and Perspectives, 2004, 17, 321.	1.9	29
94	Parallel synthesis of DAPT derivatives and their Î³-secretase-inhibitory activity. Bioorganic and Medicinal Chemistry Letters, 2004, 14, 1983-1985.	1.0	28
95	Contribution of the Î³-Secretase Subunits to the Formation of Catalytic Pore of Presenilin 1 Protein. Journal of Biological Chemistry, 2012, 287, 25834-25843.	1.6	28
96	CD2-associated protein (CD2AP) overexpression accelerates amyloid precursor protein (APP) transfer from early endosomes to the lysosomal degradation pathway. Journal of Biological Chemistry, 2019, 294, 10886-10899.	1.6	28
97	Photo-oxygenation by a biocompatible catalyst reduces amyloid-Î² levels in Alzheimerâ€™s disease mice. Brain, 2021, 144, 1884-1897.	3.7	28
98	The Presenilin 1 Mutation (M146V) Linked to Familial Alzheimer's Disease Attenuates the Neuronal Differentiation of NTera 2 Cells. Biochemical and Biophysical Research Communications, 1998, 244, 751-755.	1.0	27
99	Dual antitumor mechanisms of Notch signaling inhibitor in a Tâ€cell acute lymphoblastic leukemia xenograft model. Cancer Science, 2009, 100, 2444-2450.	1.7	27
100	Activation of Î³-Secretase Trimming Activity by Topological Changes of Transmembrane Domain 1 of Presenilin 1. Journal of Neuroscience, 2017, 37, 12272-12280.	1.7	27
101	Photo-oxygenation inhibits tau amyloid formation. Chemical Communications, 2019, 55, 6165-6168.	2.2	26
102	Convenient Synthesis of Photoaffinity Probes and Evaluation of Their Labeling Abilities. Organic Letters, 2007, 9, 2055-2058.	2.4	25
103	Differentiation of CD11c ⁺ CX ₃ CR1 ⁺ cells in the small intestine requires Notch signaling. Proceedings of the National Academy of Sciences of the United States of America, 2014, 111, 5986-5991.	3.3	25
104	Conformational Changes in Transmembrane Domain 4 of Presenilin 1 Are Associated with Altered Amyloid-Î² 42 Production. Journal of Neuroscience, 2016, 36, 1362-1372.	1.7	25
105	Analysis of Neurons Created from Wild-Type and Alzheimer's Mutation Knock-In Embryonic Stem Cells by a Highly Efficient Differentiation Protocol. Journal of Neuroscience, 2003, 23, 8513-8525.	1.7	24
106	Effect of Helical Conformation and Side Chain Structure on Î³-Secretase Inhibition by Î²-Peptide Foldamers: Insight into Substrate Recognition. Journal of Medicinal Chemistry, 2013, 56, 1443-1454.	2.9	24
107	Synthetic ceramide analogues increase amyloid-Î² 42 production by modulating Î³-secretase activity. Biochemical and Biophysical Research Communications, 2015, 457, 194-199.	1.0	24
108	Partial loss of CALM function reduces AÎ²42 production and amyloid depositionin vivo. Human Molecular Genetics, 2016, 25, 3988-3997.	1.4	24

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109	Protein trafficking and maturation regulate intramembrane proteolysis. Biochimica Et Biophysica Acta - Biomembranes, 2013, 1828, 2855-2861.	1.4	22
110	Memantine inhibits Î²-amyloid aggregation and disassembles preformed Î²-amyloid aggregates. Biochemical and Biophysical Research Communications, 2017, 493, 158-163.	1.0	22
111	Three-dimensional Structure of the Signal Peptide Peptidase. Journal of Biological Chemistry, 2011, 286, 26188-26197.	1.6	21
112	Novel Î³-secretase inhibitors discovered by library screening of in-house synthetic natural product intermediates. Bioorganic and Medicinal Chemistry Letters, 2006, 16, 3813-3816.	1.0	20
113	Binding of longer AÎ² to transmembrane domain 1 of presenilin 1 impacts on AÎ²42 generation. Molecular Neurodegeneration, 2014, 9, 7.	4.4	20
114	Rescue of Hypovitaminosis A Induces Non-Amyloidogenic Amyloid Precursor Protein (APP) Processing. Current Alzheimer Research, 2016, 13, 1277-1289.	0.7	20
115	Single Chain Variable Fragment against Nicastrin Inhibits the Î³-Secretase Activity. Journal of Biological Chemistry, 2009, 284, 27838-27847.	1.6	19
116	Molecular interactions between presenilin and calpain: inhibition of m-calpain protease activity by presenilin-1, 2 and cleavage of presenilin-1 by m-, mu-calpain.. International Journal of Molecular Medicine, 2000, 5, 269-73.	1.8	17
117	Human tauopathy-derived tau strains determine the substrates recruited for templated amplification. Brain, 2021, 144, 2333-2348.	3.7	17
118	Concise and Short Synthesis of Functionalized 5,6-Dihydropyridin-2-ones by Means of Palladium(0)-Catalyzed Cross-Coupling of Ketene Aminal Phosphates. Heterocycles, 2006, 70, 101.	0.4	16
119	Suppressor Mutations for Presenilin 1 Familial Alzheimer Disease Mutants Modulate Î³-Secretase Activities. Journal of Biological Chemistry, 2016, 291, 435-446.	1.6	16
120	Distinct Neurotoxic Effects of Extracellular Tau Species in Primary Neuronal-Glial Cultures. Molecular Neurobiology, 2021, 58, 658-667.	1.9	16
121	At the frontline of Alzheimerâ€™s disease treatment: Î³-secretase inhibitor/modulator mechanism. Naunyn-Schmiedeberg's Archives of Pharmacology, 2008, 377, 295-300.	1.4	15
122	Two Novel Mutations in the First Transmembrane Domain of Presenilin1 Cause Young-Onset Alzheimerâ€™s Disease. Journal of Alzheimer's Disease, 2017, 58, 1035-1041.	1.2	15
123	Physiological and pathological functions of LRRK2: implications from substrate proteins. Neuronal Signaling, 2018, 2, NS20180005.	1.7	15
124	The Regulation of Rab GTPases by Phosphorylation. Biomolecules, 2021, 11, 1340.	1.8	15
125	Inhibition of Î³-Secretase Activity by a Monoclonal Antibody against the Extracellular Hydrophilic Loop of Presenilin 1. Biochemistry, 2013, 52, 61-69.	1.2	14
126	Molecular mechanisms of the genetic risk factors in pathogenesis of Alzheimer disease. Frontiers in Bioscience - Landmark, 2017, 22, 180-192.	3.0	14

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127	Identification of calcium and integrinâ€binding protein 1 as a novel regulator of production of amyloid Î² peptide using CRISPR/Cas9â€based screening system. FASEB Journal, 2020, 34, 7661-7674.	0.2	14
128	Efficient fourâ€drug cocktail therapy targeting amyloidâ€Î² peptide for Alzheimer's disease. Journal of Neuroscience Research, 2010, 88, 3588-3597.	1.3	13
129	A novel non-canonical Notch signaling regulates expression of synaptic vesicle proteins in excitatory neurons. Scientific Reports, 2016, 6, 23969.	1.6	13
130	Retromer and Rab2â€dependent trafficking mediate <scp>PS</scp>1 degradation by proteasomes in endocytic disturbance. Journal of Neurochemistry, 2016, 137, 647-658.	2.1	13
131	Specific mutations in presenilin 1 cause conformational changes in Î³-secretase to modulate amyloid Î² trimming. Journal of Biochemistry, 2019, 165, 37-46.	0.9	11
132	Histidine 131 in presenilin 1 is the pH-sensitive residue that causes the increase in AÎ²42 level in acidic pH. Journal of Biochemistry, 2020, 167, 463-471.	0.9	11
133	Probing the Structure and Function Relationships of Presenilin by Substituted-Cysteine Accessibility Method. Methods in Enzymology, 2017, 584, 185-205.	0.4	10
134	Retrograde transport of Î³â€secretase from endosomes to the transâ€Golgi network regulates AÎ²42 production. Journal of Neurochemistry, 2018, 147, 110-123.	2.1	10
135	Flexible and Accurate Substrate Processing with Distinct Presenilin/Î³-Secretases in Human Cortical Neurons. ENeuro, 2021, 8, ENEURO.0500-20.2021.	0.9	10
136	Development of photoaffinity probes for Î³-secretase equipped with a nitrobenzenesulfonamide-type cleavable linker. Bioorganic and Medicinal Chemistry Letters, 2009, 19, 6869-6871.	1.0	9
137	Conformational Dynamics of Transmembrane Domain 3 of Presenilin 1 Is Associated with the Trimming Activity of Î³-Secretase. Journal of Neuroscience, 2019, 39, 8600-8610.	1.7	9
138	Peptide-based short single Î²-strand mimics without hydrogen bonding or aggregation. Chemical Communications, 2020, 56, 1573-1576.	2.2	9
139	Structure-activity relationship of presenilin in Î³-secretase-mediated intramembrane cleavage. Seminars in Cell and Developmental Biology, 2020, 105, 102-109.	2.3	9
140	Dietary cis-9, trans-11-conjugated linoleic acid reduces amyloid Î²-protein accumulation and upregulates anti-inflammatory cytokines in an Alzheimerâ€™s disease mouse model. Scientific Reports, 2021, 11, 9749.	1.6	9
141	Î²APP Î³-secretase and SREBP site 2 protease are two different enzymes. NeuroReport, 1998, 9, 911-913.	0.6	8
142	Synthesis and biological activity of N-substituted spiro[benzoxazepine-piperidine] AÎ²-peptide production inhibitors. Journal of Enzyme Inhibition and Medicinal Chemistry, 2008, 23, 996-1001.	2.5	8
143	EphA4 regulates AÎ² production via BACE1 expression in neurons. FASEB Journal, 2020, 34, 16383-16396.	0.2	8
144	BORCS6 is involved in the enlargement of lung lamellar bodies in Lrrk2 knockout mice. Human Molecular Genetics, 2021, 30, 1618-1631.	1.4	8

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145	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
146	Novel Notch-sparing Î³-secretase inhibitors derived from a peroxisome proliferator-activated receptor agonist library. Bioorganic and Medicinal Chemistry Letters, 2010, 20, 5282-5285.	1.0	7
147	Experimental detection of proteolytic activity in a signal peptide peptidase of Arabidopsis thaliana. BMC Biochemistry, 2013, 14, 16.	4.4	7
148	New photocleavable linker: Î±-Thioacetophenone-type linker. Bioorganic and Medicinal Chemistry Letters, 2014, 24, 2831-2833.	1.0	7
149	GPR120 Signaling Controls Amyloid-Î² Degrading Activity of Matrix Metalloproteinases. Journal of Neuroscience, 2021, 41, 6173-6185.	1.7	7
150	Lipid flippase dysfunction as a therapeutic target for endosomal anomalies in Alzheimerâ€™s disease. IScience, 2022, 25, 103869.	1.9	7
151	Membrane trafficking and proteolytic activity of Î³-secretase in Alzheimerâ€™s disease. Biological Chemistry, 2016, 397, 827-835.	1.2	6
152	AP180 N-Terminal Homology (ANTH) and Epsin N-Terminal Homology (ENTH) Domains: Physiological Functions and Involvement in Disease. Advances in Experimental Medicine and Biology, 2018, 1111, 55-76.	0.8	6
153	Specific Mutations in Aph1 Cause Î³-Secretase Activation. International Journal of Molecular Sciences, 2022, 23, 507.	1.8	5
154	Rab10 Phosphorylation Detection by LRRK2 Activity Using SDS-PAGE with a Phosphate-binding Tag. Journal of Visualized Experiments, 2017, , .	0.2	4
155	Photo-Oxygenation as a New Therapeutic Strategy for Neurodegenerative Proteinopathies by Enhancing the Clearance of Amyloid Proteins. Frontiers in Aging Neuroscience, 0, 14, .	1.7	4
156	Autism-associated variants of neuroligin 4X impair synaptogenic activity by various molecular mechanisms. Molecular Autism, 2020, 11, 68.	2.6	3
157	Suppression of amyloidâ€Î² secretion from neurons by cisâ€9, transâ€11â€octadecadienoic acid, an isomer of conjugated linoleic acid. Journal of Neurochemistry, 2021, 159, 603-617.	2.1	3
158	Variant Alzheimer's disease with spastic paraparesis and cotton wool plaques is caused by PSâ€1 mutations that lead to exceptionally high amyloidâ€Î² concentrations. Annals of Neurology, 2000, 48, 806-808.	2.8	3
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