Harald Steiner

List of Publications by Year
in descending order

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78 papers	8,396 citations	46918 47 h-index	69108 77 g-index
89 all docs	89 docs citations	89 times ranked	6070 citing authors

#	Article	IF	Citations
1	Secretases in Alzheimer's disease: Novel insights into proteolysis of APP and TREM2. Current Opinion in Neurobiology, 2022, 72, 101-110.	2.0	28
2	Active site geometry stabilization of a presenilin homolog by the lipid bilayer promotes intramembrane proteolysis. ELife, 2022, 11, .	2.8	3
3	Microbiota-derived short chain fatty acids modulate microglia and promote AÎ² plaque deposition. ELife, 2021, 10, .	2.8	148
4	Modulation of Î³-Secretase Activity by a Carborane-Based Flurbiprofen Analogue. Molecules, 2021, 26, 2843.	1.7	10
5	Effective sample preparation procedure for the analysis of free neutral steroids, free steroid acids and sterol sulfates in different tissues by GCâ€“MS. Journal of Steroid Biochemistry and Molecular Biology, 2021, 211, 105880.	1.2	1
6	Understanding intramembrane proteolysis by Î³-secretase. Seminars in Cell and Developmental Biology, 2020, 105, 1-2.	2.3	0
7	AÎ²43â€producing <scp>PS</scp>1 <scp>FAD</scp> mutants cause altered substrate interactions and respond to Î³â€secretase modulation. EMBO Reports, 2020, 21, e47996.	2.0	24
8	Pathogenic AÎ² generation in familial Alzheimerâ€™s disease: novel mechanistic insights and therapeutic implications. Current Opinion in Neurobiology, 2020, 61, 73-81.	2.0	22
9	Photo-controlled delivery of very long chain fatty acids to cell membranes and modulation of membrane protein function. Biochimica Et Biophysica Acta - Biomembranes, 2020, 1862, 183200.	1.4	8
10	Substrate recruitment by Î³-secretase. Seminars in Cell and Developmental Biology, 2020, 105, 54-63.	2.3	13
11	Î³â€Secretase cleavage of the Alzheimer risk factor <scp>TREM</scp> 2 is determined by its intrinsic structural dynamics. EMBO Journal, 2020, 39, e104247.	3.5	16
12	Comparison of Strategies for the Determination of Sterol Sulfates via GC-MS Leading to a Novel Deconjugation-Derivatization Protocol. Molecules, 2019, 24, 2353.	1.7	14
13	Identification of a rare presenilin 1 single amino acid deletion mutation (F175del) with unusual amyloid-Î² processing effects. Neurobiology of Aging, 2019, 84, 241.e5-241.e11.	1.5	9
14	Modulating Hinge Flexibility in the APP Transmembrane Domain Alters Î³-Secretase Cleavage. Biophysical Journal, 2019, 116, 2103-2120.	0.2	34
15	Bexarotene Binds to the Amyloid Precursor Protein Transmembrane Domain, Alters Its Î±-Helical Conformation, and Inhibits Î³-Secretase Nonselectively in Liposomes. ACS Chemical Neuroscience, 2018, 9, 1702-1713.	1.7	11
16	Making the final cut: pathogenic amyloid-Î² peptide generation by Î³-secretase. Cell Stress, 2018, 2, 292-310.	1.4	100
17	Substrate processing in intramembrane proteolysis by Î³-secretase â€“ the role of protein dynamics. Biological Chemistry, 2017, 398, 441-453.	1.2	40
18	An Alzheimerâ€associated TREM2 variant occurs at the <scp>ADAM</scp> cleavage site and affects shedding and phagocytic function. EMBO Molecular Medicine, 2017, 9, 1356-1365.	3.3	164

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19	Substrate recruitment of Î³â€secretase and mechanism of clinical presenilin mutations revealed by photoaffinity mapping. EMBO Journal, 2016, 35, 1628-1643.	3.5	104
20	Generation and deposition of AÎ²43 by the virtually inactive presenilinâ€1 L435F mutant contradicts the presenilin lossâ€ofâ€function hypothesis of Alzheimer's disease. EMBO Molecular Medicine, 2016, 8, 458-465.	3.3	60
21	Proteolytic Processing of Neuregulin 1 Type III by Three Intramembrane-cleaving Proteases. Journal of Biological Chemistry, 2016, 291, 318-333.	1.6	42
22	Inhibition of amyloid-Î² plaque formation by Î±-synuclein. Nature Medicine, 2015, 21, 802-807.	15.2	97
23	Intramembrane Proteolysis of Î²-Amyloid Precursor Protein by Î³-Secretase Is an Unusually Slow Process. Biophysical Journal, 2015, 108, 1229-1237.	0.2	77
24	Homodimerization Protects the Amyloid Precursor Protein C99 Fragment from Cleavage by Î³-Secretase. Biochemistry, 2015, 54, 6149-6152.	1.2	43
25	Shedding of glycanâ€modifying enzymes by signal peptide peptidaseâ€like 3 (<scp>SPPL</scp>3) regulates cellular Nâ€glycosylation. EMBO Journal, 2014, 33, 2890-2905.	3.5	81
26	Important functional role of residue x of the presenilin Gx<scp>GD</scp> protease active site motif for <scp>APP</scp> substrate cleavage specificity and substrate selectivity of Î³â€secretase. Journal of Neurochemistry, 2013, 125, 144-156.	2.1	18
27	Generation of Alzheimer Disease-associated Amyloid Î²42/43 Peptide by Î³-Secretase Can Be Inhibited Directly by Modulation of Membrane Thickness. Journal of Biological Chemistry, 2012, 287, 21326-21334.	1.6	89
28	Loss of PAFAH1B2 Reduces Amyloid-Î² Generation by Promoting the Degradation of Amyloid Precursor Protein C-Terminal Fragments. Journal of Neuroscience, 2012, 32, 18204-18214.	1.7	23
29	Foamy Virus Envelope Protein Is a Substrate for Signal Peptide Peptidase-like 3 (SPPL3). Journal of Biological Chemistry, 2012, 287, 43401-43409.	1.6	38
30	The Nicastrin ectodomain adopts a highly thermostable structure. Biological Chemistry, 2011, 392, 995-1001.	1.2	4
31	Regulated intramembrane proteolysis - lessons from amyloid precursor protein processing. Journal of Neurochemistry, 2011, 117, 779-796.	2.1	213
32	Attenuated AÎ²42 Responses to Low Potency Î³-Secretase Modulators Can Be Overcome for Many Pathogenic Presenilin Mutants by Second-generation Compounds. Journal of Biological Chemistry, 2011, 286, 15240-15251.	1.6	42
33	Novel Î³-Secretase Enzyme Modulators Directly Target Presenilin Protein. Journal of Biological Chemistry, 2011, 286, 37181-37186.	1.6	82
34	Requirement for small side chain residues within the GxGDâ€motif of presenilin for Î³â€secretase substrate cleavage. Journal of Neurochemistry, 2010, 112, 940-950.	2.1	18
35	Bepridil and Amiodarone Simultaneously Target the Alzheimer's Disease Â- and Â-Secretase via Distinct Mechanisms. Journal of Neuroscience, 2010, 30, 8974-8983.	1.7	51
36	Three-Amino Acid Spacing of Presenilin Endoproteolysis Suggests a General Stepwise Cleavage of Â-Secretase-Mediated Intramembrane Proteolysis. Journal of Neuroscience, 2010, 30, 7853-7862.	1.7	93

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37	Î²-Amyloid Precursor Protein Mutants Respond to Î³-Secretase Modulators. Journal of Biological Chemistry, 2010, 285, 17798-17810.	1.6	64
38	Intramembrane Proteolysis by Signal Peptide Peptidases: A Comparative Discussion of GXGD-type Aspartyl Proteases. Journal of Biological Chemistry, 2009, 284, 13975-13979.	1.6	56
39	Purification, Pharmacological Modulation, and Biochemical Characterization of Interactors of Endogenous Human Î³-Secretase. Biochemistry, 2009, 48, 1183-1197.	1.2	65
40	Chemical Cross-linking Provides a Model of the Î³-Secretase Complex Subunit Architecture and Evidence for Close Proximity of the C-terminal Fragment of Presenilin with APH-1. Journal of Biological Chemistry, 2008, 283, 34677-34686.	1.6	37
41	The Catalytic Core of γ-Secretase: Presenilin Revisited. Current Alzheimer Research, 2008, 5, 147-157.	0.7	33
42	Intramembrane Proteolysis by Î³-Secretase. Journal of Biological Chemistry, 2008, 283, 29627-29631.	1.6	186
43	Generation of AÎ²38 and AÎ²42 Is Independently and Differentially Affected by Familial Alzheimer Disease-associated Presenilin Mutations and Î³-Secretase Modulation. Journal of Biological Chemistry, 2008, 283, 677-683.	1.6	152
44	Active Î³-Secretase Complexes Contain Only One of Each Component. Journal of Biological Chemistry, 2007, 282, 33985-33993.	1.6	155
45	Endoplasmic reticulum retention of the Î³â€secretase complex component Pen2 by Rer1. EMBO Reports, 2007, 8, 743-748.	2.0	74
46	Pathological activity of familial Alzheimerâ€™s disease-associated mutant presenilin can be executed by six different Î³-secretase complexes. Neurobiology of Disease, 2007, 27, 102-107.	2.1	74
47	Assembly, Trafficking and Function of Î³-Secretase. Neurodegenerative Diseases, 2006, 3, 275-283.	0.8	133
48	Pore-forming scissors? A first structural glimpse of Î³-secretase. Trends in Biochemical Sciences, 2006, 31, 491-493.	3.7	5
49	The GxGD Motif of Presenilin Contributes to Catalytic Function and Substrate Identification of Â-Secretase. Journal of Neuroscience, 2006, 26, 3821-3828.	1.7	79
50	Differential Localization and Identification of a Critical Aspartate Suggest Non-redundant Proteolytic Functions of the Presenilin Homologues SPPL2b and SPPL3. Journal of Biological Chemistry, 2005, 280, 39515-39523.	1.6	78
51	Î³-Secretase Complex Assembly within the Early Secretory Pathway. Journal of Biological Chemistry, 2005, 280, 6471-6478.	1.6	77
52	Uncovering γ-Secretase. Current Alzheimer Research, 2004, 1, 175-181.	0.7	40
53	Co-expression of Nicastrin and Presenilin Rescues a Loss of Function Mutant of APH-1. Journal of Biological Chemistry, 2004, 279, 37311-37315.	1.6	25
54	Requirement of PEN-2 for Stabilization of the Presenilin N-/C-terminal Fragment Heterodimer within the Î³-Secretase Complex. Journal of Biological Chemistry, 2004, 279, 23255-23261.	1.6	107

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55	Identification of Distinct Î³-Secretase Complexes with Different APH-1 Variants. Journal of Biological Chemistry, 2004, 279, 41340-41345.	1.6	149
56	Immature nicastrin stabilizes APHâ€1 independent of PENâ€2 and presenilin: identification of nicastrin mutants that selectively interact with APHâ€1. Journal of Neurochemistry, 2004, 89, 1520-1527.	2.1	60
57	The presenilin C-terminus is required for ER-retention, nicastrin-binding and Î³-secretase activity. EMBO Journal, 2004, 23, 4738-4748.	3.5	91
58	Reconstitution of Î³-secretase activity. Nature Cell Biology, 2003, 5, 486-488.	4.6	850
59	Nicastrin Interacts with Î³-Secretase Complex Components via the N-terminal Part of Its Transmembrane Domain. Journal of Biological Chemistry, 2003, 278, 52519-52523.	1.6	54
60	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
61	Insulin-degrading Enzyme Rapidly Removes the Î²-Amyloid Precursor Protein Intracellular Domain (AICD). Journal of Biological Chemistry, 2002, 277, 13389-13393.	1.6	185
62	Presenilin-1 mutations of leucine 166 equally affect the generation of the Notch and APP intracellular domains independent of their effect on AÂ42 production. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 8025-8030.	3.3	265
63	Presenilin and nicastrin regulate each other and determine amyloid Â-peptide production via complex formation. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 8666-8671.	3.3	229
64	Presenilin-1 affects trafficking and processing of Î²APP and is targeted in a complex with nicastrin to the plasma membrane. Journal of Cell Biology, 2002, 158, 551-561.	2.3	179
65	PEN-2 Is an Integral Component of the Î³-Secretase Complex Required for Coordinated Expression of Presenilin and Nicastrin. Journal of Biological Chemistry, 2002, 277, 39062-39065.	1.6	244
66	Alzheimer disease Î³-secretase: a complex story of GxGD-type presenilin proteases. Trends in Cell Biology, 2002, 12, 556-562.	3.6	165
67	A Î³â€secretase inhibitor blocks Notch signalingin vivoand causes a severe neurogenic phenotype in zebrafish. EMBO Reports, 2002, 3, 688-694.	2.0	459
68	Presenilins mediate a dual intramembranous gamma-secretase cleavage of Notch-1. EMBO Journal, 2002, 21, 5408-5416.	3.5	214
69	Nuclear Signaling: A Common Function of Presenilin Substrates?. Journal of Molecular Neuroscience, 2001, 17, 193-198.	1.1	18
70	Presenilinâ€dependent Î³â€secretase processing of Î²â€amyloid precursor protein at a site corresponding to the S3 cleavage of Notch. EMBO Reports, 2001, 2, 835-841.	2.0	457
71	Presenilin-1 differentially facilitates endoproteolysis of the Î²-amyloid precursor protein and Notch. Nature Cell Biology, 2000, 2, 205-211.	4.6	146
72	Glycine 384 is required for presenilin-1 function and is conserved in bacterial polytopic aspartyl proteases. Nature Cell Biology, 2000, 2, 848-851.	4.6	263

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73	Intramembrane proteolysis by presenilins. Nature Reviews Molecular Cell Biology, 2000, 1, 217-224.	16.1	151
74	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
75	The Biological and Pathological Function of the Presenilin-1 Î”Exon 9 Mutation Is Independent of Its Defect to Undergo Proteolytic Processing. Journal of Biological Chemistry, 1999, 274, 7615-7618.	1.6	121
76	An in vivo assay for the identification of target proteases which cleave membrane-associated substrates. FEBS Letters, 1999, 463, 245-249.	1.3	17
77	Amyloidogenic Function of the Alzheimer's Disease-Associated Presenilin 1 in the Absence of Endoproteolysis. Biochemistry, 1999, 38, 14600-14605.	1.2	99
78	Expression of Alzheimerâ€™s Disease-associated Presenilin-1 Is Controlled by Proteolytic Degradation and Complex Formation. Journal of Biological Chemistry, 1998, 273, 32322-32331.	1.6	182