Harald Steiner

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/9064977/publications.pdf Version: 2024-02-01



HADALD STEINED

#	Article	IF	CITATIONS
1	Reconstitution of Î ³ -secretase activity. Nature Cell Biology, 2003, 5, 486-488.	4.6	850
2	A γâ€secretase inhibitor blocks Notch signalingin vivoand causes a severe neurogenic phenotype in zebrafish. EMBO Reports, 2002, 3, 688-694.	2.0	459
3	Presenilinâ€dependent γâ€secretase processing of βâ€amyloid precursor protein at a site corresponding to the S3 cleavage of Notch. EMBO Reports, 2001, 2, 835-841.	2.0	457
4	A Loss of Function Mutation of Presenilin-2 Interferes with Amyloid β-Peptide Production and Notch Signaling. Journal of Biological Chemistry, 1999, 274, 28669-28673.	1.6	279
5	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
6	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
7	Presenilin-dependent Intramembrane Proteolysis of CD44 Leads to the Liberation of Its Intracellular Domain and the Secretion of an Aβ-like Peptide. Journal of Biological Chemistry, 2002, 277, 44754-44759.	1.6	253
8	PEN-2 Is an Integral Component of the Î ³ -Secretase Complex Required for Coordinated Expression of Presenilin and Nicastrin. Journal of Biological Chemistry, 2002, 277, 39062-39065.	1.6	244
9	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
10	Presenilins mediate a dual intramembranous gamma-secretase cleavage of Notch-1. EMBO Journal, 2002, 21, 5408-5416.	3.5	214
11	Regulated intramembrane proteolysis - lessons from amyloid precursor protein processing. Journal of Neurochemistry, 2011, 117, 779-796.	2.1	213
12	Intramembrane Proteolysis by \hat{I}^3 -Secretase. Journal of Biological Chemistry, 2008, 283, 29627-29631.	1.6	186
13	Insulin-degrading Enzyme Rapidly Removes the β-Amyloid Precursor Protein Intracellular Domain (AICD). Journal of Biological Chemistry, 2002, 277, 13389-13393.	1.6	185
14	Expression of Alzheimer's Disease-associated Presenilin-1 Is Controlled by Proteolytic Degradation and Complex Formation. Journal of Biological Chemistry, 1998, 273, 32322-32331.	1.6	182
15	Presenilin-1 affects trafficking and processing of βAPP and is targeted in a complex with nicastrin to the plasma membrane. Journal of Cell Biology, 2002, 158, 551-561.	2.3	179
16	Alzheimer disease Î ³ -secretase: a complex story of GxGD-type presenilin proteases. Trends in Cell Biology, 2002, 12, 556-562.	3.6	165
17	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
18	Active Î ³ -Secretase Complexes Contain Only One of Each Component. Journal of Biological Chemistry, 2007, 282, 33985-33993.	1.6	155

#	Article	IF	CITATIONS
19	Generation of AÎ ² 38 and AÎ ² 42 Is Independently and Differentially Affected by Familial Alzheimer Disease-associated Presenilin Mutations and Î ³ -Secretase Modulation. Journal of Biological Chemistry, 2008, 283, 677-683.	1.6	152
20	Intramembrane proteolysis by presenilins. Nature Reviews Molecular Cell Biology, 2000, 1, 217-224.	16.1	151
21	ldentification of Distinct γ-Secretase Complexes with Different APH-1 Variants. Journal of Biological Chemistry, 2004, 279, 41340-41345.	1.6	149
22	Microbiota-derived short chain fatty acids modulate microglia and promote Aβ plaque deposition. ELife, 2021, 10, .	2.8	148
23	Presenilin-1 differentially facilitates endoproteolysis of the Î ² -amyloid precursor protein and Notch. Nature Cell Biology, 2000, 2, 205-211.	4.6	146
24	Assembly, Trafficking and Function of \hat{I}^3 -Secretase. Neurodegenerative Diseases, 2006, 3, 275-283.	0.8	133
25	The Biological and Pathological Function of the Presenilin-1 ΔExon 9 Mutation Is Independent of Its Defect to Undergo Proteolytic Processing. Journal of Biological Chemistry, 1999, 274, 7615-7618.	1.6	121
26	Requirement of PEN-2 for Stabilization of the Presenilin N-/C-terminal Fragment Heterodimer within the Î ³ -Secretase Complex. Journal of Biological Chemistry, 2004, 279, 23255-23261.	1.6	107
27	Substrate recruitment of γâ€secretase and mechanism of clinical presenilin mutations revealed by photoaffinity mapping. EMBO Journal, 2016, 35, 1628-1643.	3.5	104
28	Making the final cut: pathogenic amyloid-β peptide generation by γ-secretase. Cell Stress, 2018, 2, 292-310.	1.4	100
29	Amyloidogenic Function of the Alzheimer's Disease-Associated Presenilin 1 in the Absence of Endoproteolysis. Biochemistry, 1999, 38, 14600-14605.	1.2	99
30	Inhibition of amyloid- \hat{l}^2 plaque formation by $\hat{l}\pm$ -synuclein. Nature Medicine, 2015, 21, 802-807.	15.2	97
31	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
32	The presenilin C-terminus is required for ER-retention, nicastrin-binding and Î ³ -secretase activity. EMBO Journal, 2004, 23, 4738-4748.	3.5	91
33	Generation of Alzheimer Disease-associated Amyloid β42/43 Peptide by γ-Secretase Can Be Inhibited Directly by Modulation of Membrane Thickness. Journal of Biological Chemistry, 2012, 287, 21326-21334.	1.6	89
34	Novel γ-Secretase Enzyme Modulators Directly Target Presenilin Protein. Journal of Biological Chemistry, 2011, 286, 37181-37186.	1.6	82
35	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
36	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

#	Article	IF	CITATIONS
37	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
38	Î ³ -Secretase Complex Assembly within the Early Secretory Pathway. Journal of Biological Chemistry, 2005, 280, 6471-6478.	1.6	77
39	Intramembrane Proteolysis of β-Amyloid Precursor Protein by γ-Secretase Is an Unusually Slow Process. Biophysical Journal, 2015, 108, 1229-1237.	0.2	77
40	Endoplasmic reticulum retention of the γâ€secretase complex component Pen2 by Rer1. EMBO Reports, 2007, 8, 743-748.	2.0	74
41	Pathological activity of familial Alzheimer's disease-associated mutant presenilin can be executed by six different γ-secretase complexes. Neurobiology of Disease, 2007, 27, 102-107.	2.1	74
42	Purification, Pharmacological Modulation, and Biochemical Characterization of Interactors of Endogenous Human Î ³ -Secretase. Biochemistry, 2009, 48, 1183-1197.	1.2	65
43	β-Amyloid Precursor Protein Mutants Respond to γ-Secretase Modulators. Journal of Biological Chemistry, 2010, 285, 17798-17810.	1.6	64
44	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
45	Generation and deposition of Aβ43 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
46	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
47	Nicastrin Interacts with Î ³ -Secretase Complex Components via the N-terminal Part of Its Transmembrane Domain. Journal of Biological Chemistry, 2003, 278, 52519-52523.	1.6	54
48	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
49	Homodimerization Protects the Amyloid Precursor Protein C99 Fragment from Cleavage by Î ³ -Secretase. Biochemistry, 2015, 54, 6149-6152.	1.2	43
50	Attenuated Al̂²42 Responses to Low Potency γ-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
51	Proteolytic Processing of Neuregulin 1 Type III by Three Intramembrane-cleaving Proteases. Journal of Biological Chemistry, 2016, 291, 318-333.	1.6	42
52	Uncovering γ-Secretase. Current Alzheimer Research, 2004, 1, 175-181.	0.7	40
53	Substrate processing in intramembrane proteolysis by γ-secretase – the role of protein dynamics. Biological Chemistry, 2017, 398, 441-453.	1.2	40
54	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

#	Article	IF	CITATIONS
55	Chemical Cross-linking Provides a Model of the γ-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
56	Modulating Hinge Flexibility in the APP Transmembrane Domain Alters Î ³ -Secretase Cleavage. Biophysical Journal, 2019, 116, 2103-2120.	0.2	34
57	The Catalytic Core of γ-Secretase: Presenilin Revisited. Current Alzheimer Research, 2008, 5, 147-157.	0.7	33
58	Secretases in Alzheimer's disease: Novel insights into proteolysis of APP and TREM2. Current Opinion in Neurobiology, 2022, 72, 101-110.	2.0	28
59	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
60	Aβ43â€producing <scp>PS</scp> 1 <scp>FAD</scp> mutants cause altered substrate interactions and respond to γâ€secretase modulation. EMBO Reports, 2020, 21, e47996.	2.0	24
61	Loss of PAFAH1B2 Reduces Amyloid-β Generation by Promoting the Degradation of Amyloid Precursor Protein C-Terminal Fragments. Journal of Neuroscience, 2012, 32, 18204-18214.	1.7	23
62	Pathogenic Aβ generation in familial Alzheimer's disease: novel mechanistic insights and therapeutic implications. Current Opinion in Neurobiology, 2020, 61, 73-81.	2.0	22
63	Nuclear Signaling: A Common Function of Presenilin Substrates?. Journal of Molecular Neuroscience, 2001, 17, 193-198.	1.1	18
64	Requirement for small side chain residues within the GxGDâ€motif of presenilin for γâ€secretase substrate cleavage. Journal of Neurochemistry, 2010, 112, 940-950.	2.1	18
65	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 γâ€secretase. Journal of Neurochemistry, 2013, 125, 144-156.	2.1	18
66	An in vivo assay for the identification of target proteases which cleave membrane-associated substrates. FEBS Letters, 1999, 463, 245-249.	1.3	17
67	γâ€5ecretase 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
68	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
69	Substrate recruitment by Î ³ -secretase. Seminars in Cell and Developmental Biology, 2020, 105, 54-63.	2.3	13
70	Bexarotene Binds to the Amyloid Precursor Protein Transmembrane Domain, Alters Its α-Helical Conformation, and Inhibits γ-Secretase Nonselectively in Liposomes. ACS Chemical Neuroscience, 2018, 9, 1702-1713.	1.7	11
71	Modulation of Î ³ -Secretase Activity by a Carborane-Based Flurbiprofen Analogue. Molecules, 2021, 26, 2843.	1.7	10
72	Identification of a rare presenilin 1 single amino acid deletion mutation (F175del) with unusual amyloid-β processing effects. Neurobiology of Aging, 2019, 84, 241.e5-241.e11.	1.5	9

#	Article	IF	CITATIONS
73	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
74	Pore-forming scissors? A first structural glimpse of Î ³ -secretase. Trends in Biochemical Sciences, 2006, 31, 491-493.	3.7	5
75	The Nicastrin ectodomain adopts a highly thermostable structure. Biological Chemistry, 2011, 392, 995-1001.	1.2	4
76	Active site geometry stabilization of a presenilin homolog by the lipid bilayer promotes intramembrane proteolysis. ELife, 2022, 11, .	2.8	3
77	Effective sample preparation procedure for the analysis of free neutral steroids, free steroid acids and sterol sulfates in different tissues by GC–MS. Journal of Steroid Biochemistry and Molecular Biology, 2021, 211, 105880.	1.2	1
78	Understanding intramembrane proteolysis by γ-secretase. Seminars in Cell and Developmental Biology, 2020, 105, 1-2.	2.3	0