Jan NÃ¤slund

List of Publications by Year
in descending order

36 papers	6,726 citations	236925 25 h-index	345221 36 g-index
36 all docs	36 docs citations	36 times ranked	7561 citing authors

#	Article	IF	Citations
1	Amyloid neuropathology in the single Arctic APP transgenic model affects interconnected brain regions. Neurobiology of Aging, 2012, 33, 831.e11-831.e19.	3.1	40
2	Progressive neuropathology and cognitive decline in a single Arctic APP transgenic mouse model. Neurobiology of Aging, 2011, 32, 280-292.	3.1	42
3	Mutations in Nicastrin Protein Differentially Affect Amyloid Î²-Peptide Production and Notch Protein Processing. Journal of Biological Chemistry, 2011, 286, 31153-31158.	3.4	15
4	The Large Hydrophilic Loop of Presenilin 1 Is Important for Regulating Î³-Secretase Complex Assembly and Dictating the Amyloid Î² Peptide (AÎ²) Profile without Affecting Notch Processing*. Journal of Biological Chemistry, 2010, 285, 8527-8536.	3.4	13
5	Clinical and Neuropathological Features of the Arctic APP Gene Mutation Causing Early-Onset Alzheimer Disease. Archives of Neurology, 2008, 65, 499.	4.5	91
6	The Alzheimer's disease-associated Î³-secretase complex: Functional domains in the presenilin 1 protein. Physiology and Behavior, 2007, 92, 115-120.	2.1	18
7	CLAC Binds to Aggregated AÎ² and AÎ² Fragments, and Attenuates Fibril Elongation. Biochemistry, 2005, 44, 15602-15609.	2.5	30
8	Aph-1 interacts at the cell surface with proteins in the active Î³-secretase complex and membrane-tethered Notch. Journal of Neurochemistry, 2005, 92, 1010-1020.	3.9	25
9	gamma-Secretase complexes containing N- and C-terminal fragments of different presenilin origin retain normal gamma-secretase activity. Journal of Neurochemistry, 2005, 95, 880-890.	3.9	20
10	Collagenous Alzheimer amyloid plaque component assembles amyloid fibrils into protease resistant aggregates. FEBS Journal, 2005, 272, 2231-2236.	4.7	34
11	Characterization of the Alzheimer's Disease-associated CLAC Protein and Identification of an Amyloid Î²-Peptide-binding Site. Journal of Biological Chemistry, 2005, 280, 1007-1015.	3.4	25
12	A Nine-transmembrane Domain Topology for Presenilin 1. Journal of Biological Chemistry, 2005, 280, 35352-35360.	3.4	162
13	Macroautophagyâ€”a novel Î²-amyloid peptide-generating pathway activated in Alzheimer's disease. Journal of Cell Biology, 2005, 171, 87-98.	5.2	891
14	The Extreme C Terminus of Presenilin 1 Is Essential for Î³-Secretase Complex Assembly and Activity. Journal of Biological Chemistry, 2004, 279, 45564-45572.	3.4	43
15	Functional Domains in Presenilin 1. Journal of Biological Chemistry, 2004, 279, 23925-23932.	3.4	16
16	Pen-2 Is Sequestered in the Endoplasmic Reticulum and Subjected to Ubiquitylation and Proteasome-mediated Degradation in the Absence of Presenilin. Journal of Biological Chemistry, 2004, 279, 16744-16753.	3.4	66
17	Co-expressed presenilin 1 NTF and CTF form functional Î³-secretase complexes in cells devoid of full-length protein. Journal of Neurochemistry, 2004, 89, 44-53.	3.9	47
18	APP intracellular domain formation and unaltered signaling in the presence of familial Alzheimerâ€™s disease mutations. Experimental Cell Research, 2003, 287, 1-9.	2.6	25

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19	Partial Purification and Characterization of Î³-Secretase from Post-mortem Human Brain. Journal of Biological Chemistry, 2003, 278, 24277-24284.	3.4	135
20	Presenilin-1 Mutation L271V Results in Altered Exon 8 Splicing and Alzheimer's Disease with Non-cored Plaques and No Neuritic Dystrophy. Journal of Biological Chemistry, 2003, 278, 6748-6754.	3.4	54
21	Molecular Identification of AMY, an Alzheimer Disease Amyloid-Associated Protein. Journal of Neuropathology and Experimental Neurology, 2003, 62, 1108-1117.	1.7	18
22	The Arctic mutation interferes with processing of the amyloid precursor protein. NeuroReport, 2002, 13, 1857-1860.	1.2	48
23	A Sensitive and Quantitative Assay for Measuring Cleavage of Presenilin Substrates. Journal of Biological Chemistry, 2002, 277, 6763-6766.	3.4	94
24	The 'Arctic' APP mutation (E693G) causes Alzheimer's disease by enhanced AÎ² protofibril formation. Nature Neuroscience, 2001, 4, 887-893.	14.8	1,042
25	Quantification of Alzheimer Amyloid Î² Peptides Ending at Residues 40 and 42 by Novel ELISA Systems. Molecular Medicine, 2000, 6, 291-302.	4.4	66
26	Correlation Between Elevated Levels of Amyloid Î²-Peptide in the Brain and Cognitive Decline. JAMA - Journal of the American Medical Association, 2000, 283, 1571.	7.4	1,110
27	Controlling Polymerization of Î²-Amyloid and Prion-derived Peptides with Synthetic Small Molecule Ligands. Journal of Biological Chemistry, 2000, 275, 1673-1678.	3.4	65
28	Intraneuronal AÎ²42 Accumulation in Human Brain. American Journal of Pathology, 2000, 156, 15-20.	3.8	930
29	Endogenous Proteins Controlling Amyloid Î²-Peptide Polymerization. Journal of Biological Chemistry, 1999, 274, 15990-15995.	3.4	144
30	Amyloid fibril formation by pulmonary surfactant protein C. FEBS Letters, 1999, 464, 138-142.	2.8	100
31	Generation of Alzheimer Amyloid Î² Peptide through Nonspecific Proteolysis. Journal of Biological Chemistry, 1997, 272, 1870-1875.	3.4	30
32	Controlling Amyloid Î²-Peptide Fibril Formation with Protease-stable Ligands. Journal of Biological Chemistry, 1997, 272, 12601-12605.	3.4	207
33	Arrest of -Amyloid Fibril Formation by a Pentapeptide Ligand. Journal of Biological Chemistry, 1996, 271, 8545-8548.	3.4	850
34	Highâ€Resolution Separation of Amyloid Î²â€Peptides: Structural Variants Present in Alzheimer's Disease Amyloid. Journal of Neurochemistry, 1996, 67, 294-301.	3.9	23
35	Characterization of stable complexes involving apolipoprotein E and the amyloid Î² peptide in Alzheimer's disease brain. Neuron, 1995, 15, 219-228.	8.1	195
36	A porcine gut polypeptide identical to the pancreatic hormone PP (pancreatic polypeptide). FEBS Letters, 1994, 341, 239-243.	2.8	12