Martin Citron

List of Publications by Year in descending order

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69 papers	20,310 citations	44069 48 h-index	110387 64 g-index
71 all docs	71 docs citations	71 times ranked	13836 citing authors

#	Article	IF	Citations
1	Current directions in tau research: Highlights from Tau 2020. Alzheimer's and Dementia, 2022, 18, 988-1007.	0.8	42
2	Monitoring of a progressive functional dopaminergic deficit in the A53T-AAV synuclein rats by combining 6-[18F]fluoro-L-m-tyrosine imaging and motor performances analysis. Neurobiology of Aging, 2021, 107, 142-152.	3.1	4
3	Prevention of tau seeding and propagation by immunotherapy with a central tau epitope antibody. Brain, 2019, 142, 1736-1750.	7.6	113
4	The tau positronâ€emission tomography tracer AVâ€1451 binds with similar affinities to tau fibrils and monoamine oxidases. Movement Disorders, 2018, 33, 273-281.	3.9	119
5	Epitope determines efficacy of therapeutic anti-Tau antibodies in a functional assay with human Alzheimer Tau. Acta Neuropathologica, 2018, 136, 729-745.	7.7	84
6	O4-07-01: T807, a reported selective tau tracer, binds with nanomolar affinity to monoamine oxidase a. , 2015, 11, P283-P283.		29
7	Hydroxyethylamine-based inhibitors of BACE1: P1â€“P3 macrocyclization can improve potency, selectivity, and cell activity. Bioorganic and Medicinal Chemistry Letters, 2013, 23, 4459-4464.	2.2	17
8	Design and Preparation of a Potent Series of Hydroxyethylamine Containing Î²-Secretase Inhibitors That Demonstrate Robust Reduction of Central Î²-Amyloid. Journal of Medicinal Chemistry, 2012, 55, 9009-9024.	6.4	60
9	Constitutive secretion of tau protein by an unconventional mechanism. Neurobiology of Disease, 2012, 48, 356-366.	4.4	198
10	Establishing the Relationship between In Vitro Potency, Pharmacokinetic, and Pharmacodynamic Parameters in a Series of Orally Available, Hydroxyethylamine-Derived Î²-Secretase Inhibitors. Journal of Pharmacology and Experimental Therapeutics, 2012, 343, 460-467.	2.5	11
11	A Potent and Orally Efficacious, Hydroxyethylamine-Based Inhibitor of Î²-Secretase. ACS Medicinal Chemistry Letters, 2012, 3, 886-891.	2.8	28
12	Design and Synthesis of Potent, Orally Efficacious Hydroxyethylamine Derived Î²-Site Amyloid Precursor Protein Cleaving Enzyme (BACE1) Inhibitors. Journal of Medicinal Chemistry, 2012, 55, 9025-9044.	6.4	43
13	Robust Central Reduction of Amyloid-Î² in Humans with an Orally Available, Non-Peptidic Î²-Secretase Inhibitor. Journal of Neuroscience, 2011, 31, 16507-16516.	3.6	340
14	Passive Immunization with Anti-Tau Antibodies in Two Transgenic Models. Journal of Biological Chemistry, 2011, 286, 34457-34467.	3.4	303
15	Î²â€Secretase cleavage is not required for generation of the intracellular Câ€terminal domain of the amyloid precursor family of proteins. FEBS Journal, 2010, 277, 1503-1518.	4.7	22
16	Alzheimer's disease: strategies for disease modification. Nature Reviews Drug Discovery, 2010, 9, 387-398.	46.4	928
17	Â-Site Amyloid Precursor Protein Cleaving Enzyme 1 Levels Become Elevated in Neurons around Amyloid Plaques: Implications for Alzheimer's Disease Pathogenesis. Journal of Neuroscience, 2007, 27, 3639-3649.	3.6	333
18	Involvement of -site APP cleaving enzyme 1 (BACE1) in amyloid precursor protein-mediated enhancement of memory and activity-dependent synaptic plasticity. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 8167-8172.	7.1	107

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19	BACE1 gene deletion prevents neuron loss and memory deficits in 5XFAD APP/PS1 transgenic mice. Neurobiology of Disease, 2007, 26, 134-145.	4.4	272
20	Control of Peripheral Nerve Myelination by the ÃŸ-Secretase BACE1. Science, 2006, 314, 664-666.	12.6	652
21	Temporal memory deficits in Alzheimer's mouse models: rescue by genetic deletion of BACE1. European Journal of Neuroscience, 2006, 23, 251-260.	2.6	256
22	In Vivo Cleavage of Î±2,6-Sialyltransferase by Alzheimer Î²-Secretase. Journal of Biological Chemistry, 2005, 280, 8589-8595.	3.4	88
23	Strategies for disease modification in Alzheimer's disease. Nature Reviews Neuroscience, 2004, 5, 677-685.	10.2	409
24	Amyloid Î² peptide load is correlated with increased Î²-secretase activity in sporadic Alzheimer's disease patients. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 3632-3637.	7.1	485
25	Î²-Secretase inhibition for the treatment of Alzheimer's disease â€“ promise and challenge. Trends in Pharmacological Sciences, 2004, 25, 92-97.	8.7	197
26	BACE1 Deficiency Rescues Memory Deficits and Cholinergic Dysfunction in a Mouse Model of Alzheimer's Disease. Neuron, 2004, 41, 27-33.	8.1	506
27	Elevated Î²-secretase expression and enzymatic activity detected in sporadic Alzheimer disease. Nature Medicine, 2003, 9, 3-4.	30.7	686
28	BACE1 (Î²-secretase) knockout mice do not acquire compensatory gene expression changes or develop neural lesions over time. Neurobiology of Disease, 2003, 14, 81-88.	4.4	160
29	Anti-Inflammatory Drug Therapy Alters Î²-Amyloid Processing and Deposition in an Animal Model of Alzheimer's Disease. Journal of Neuroscience, 2003, 23, 7504-7509.	3.6	473
30	Emerging Alzheimerâ€™s disease therapies: inhibition of Î²-secretase. Neurobiology of Aging, 2002, 23, 1017-1022.	3.1	60
31	Î²â€secretase as a target for the treatment of Alzheimer's disease. Journal of Neuroscience Research, 2002, 70, 373-379.	2.9	69
32	Alzheimer's disease: treatments in discovery and development. Nature Neuroscience, 2002, 5, 1055-1057.	14.8	154
33	Î²-Secretase â€” A Target for Alzheimerâ€™s Disease. Advances in Behavioral Biology, 2002, , 79-83.	0.2	0
34	Human Î²-secretase and Alzheimerâ€™s disease. Expert Opinion on Therapeutic Targets, 2001, 5, 341-348.	3.4	10
35	Mice deficient in BACE1, the Alzheimer's Î²-secretase, have normal phenotype and abolished Î²-amyloid generation. Nature Neuroscience, 2001, 4, 231-232.	14.8	978
36	Î±-Synuclein fibrillogenesis as target for drug development. , 2001, , 143-149.		0

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37	Î²-Secretase: Tissue Culture Studies of Sequence Specificity, Inhibitors, and Candidate Enzymes. , 2000, 32, 229-238.		0
38	Secretases as targets for the treatment of Alzheimerâ€™s disease. Trends in Molecular Medicine, 2000, 6, 392-397.	2.6	38
39	Characterization of Alzheimer's Î²-Secretase Protein BACE. Journal of Biological Chemistry, 2000, 275, 21099-21106.	3.4	208
40	Parkinson's Disease-associated Î±-Synuclein Is More Fibrillogenic than Î²- and Î³-Synuclein and Cannot Cross-seed Its Homologs. Journal of Biological Chemistry, 2000, 275, 34574-34579.	3.4	158
41	Expression Analysis of BACE2 in Brain and Peripheral Tissues. Journal of Biological Chemistry, 2000, 275, 20647-20651.	3.4	264
42	AÎ²-Generating Enzymes. Neuron, 2000, 27, 419-422.	8.1	311
43	Fenchylamine Sulfonamide Inhibitors of Amyloid Î² Peptide Production by the Î³-Secretase Proteolytic Pathway:â€‰ Potential Small-Molecule Therapeutic Agents for the Treatment of Alzheimer's Disease. Journal of Medicinal Chemistry, 2000, 43, 2297-2299.	6.4	67
44	A Furin-like Convertase Mediates Propeptide Cleavage of BACE, the Alzheimer's Î²-Secretase. Journal of Biological Chemistry, 2000, 275, 37712-37717.	3.4	234
45	Identifying Proteases That Cleave APP. Annals of the New York Academy of Sciences, 2000, 920, 192-196.	3.8	5
46	Both Familial Parkinson's Disease Mutations Accelerate Î±-Synuclein Aggregation. Journal of Biological Chemistry, 1999, 274, 9843-9846.	3.4	659
47	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.	3.4	279
48	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.	3.4	121
49	Î²-Secretase Cleavage of Alzheimer's Amyloid Precursor Protein by the Transmembrane Aspartic Protease BACE. Science, 1999, 286, 735-741.	12.6	3,619
50	Î±-Synuclein Fibrillogenesis Is Nucleation-dependent. Journal of Biological Chemistry, 1999, 274, 19509-19512.	3.4	608
51	Amyloidogenic Function of the Alzheimer's Disease-Associated Presenilin 1 in the Absence of Endoproteolysis. Biochemistry, 1999, 38, 14600-14605.	2.5	99
52	A Substrate-Based Difluoro Ketone Selectively Inhibits Alzheimer's Î³-Secretase Activity. Journal of Medicinal Chemistry, 1998, 41, 6-9.	6.4	219
53	Additive Effects of PS1 and APP Mutations on Secretion of the 42-Residue Amyloid Î²-Protein. Neurobiology of Disease, 1998, 5, 107-116.	4.4	94
54	Expression of Alzheimerâ€™s Disease-associated Presenilin-1 Is Controlled by Proteolytic Degradation and Complex Formation. Journal of Biological Chemistry, 1998, 273, 32322-32331.	3.4	182

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55	Amyloid Precursor Protein Processing in Sterol Regulatory Element-binding Protein Site 2 Protease-deficient Chinese Hamster Ovary Cells. Journal of Biological Chemistry, 1998, 273, 15309-15312.	3.4	16
56	The Proteolytic Fragments of the Alzheimer's Disease-associated Presenilin-1 Form Heterodimers and Occur as a 100â€“150-kDa Molecular Mass Complex. Journal of Biological Chemistry, 1998, 273, 3205-3211.	3.4	306
57	Enhanced Production and Oligomerization of the 42-residue Amyloid Î²-Protein by Chinese Hamster Ovary Cells Stably Expressing Mutant Presenilins. Journal of Biological Chemistry, 1997, 272, 7977-7982.	3.4	269
58	Presenilin Proteins Undergo Heterogeneous Endoproteolysis between Thr291and Ala299and Occur as Stable N- and C-Terminal Fragments in Normal and Alzheimer Brain Tissue. Neurobiology of Disease, 1997, 3, 325-337.	4.4	304
59	Mutant presenilins of Alzheimer's disease increase production of 42-residue amyloid Î²-protein in both transfected cells and transgenic mice. Nature Medicine, 1997, 3, 67-72.	30.7	1,271
60	Substitution of phenylalanine by proline at position 19 of amyloid Î² peptide results in an increased production of amyloid Î² peptides with alternative N-termini after protein kinase C stimulation. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 1996, 3, 150-155.	3.0	4
61	The Swedish mutation causes early-onset Alzheimer's disease by Î²-secretase cleavage within the secretory pathway. Nature Medicine, 1995, 1, 1291-1296.	30.7	529
62	The Vacuolar H+-ATPase Inhibitor Bafilomycin A1 Differentially Affects Proteolytic Processing of Mutant and Wild-type Î²-Amyloid Precursor Protein. Journal of Biological Chemistry, 1995, 270, 6186-6192.	3.4	85
63	Generation of amyloid Î² protein from its precursor is sequence specific. Neuron, 1995, 14, 661-670.	8.1	282
64	Normal Production of the Amyloid Î²-Protein and the Pathogenesis of Alzheimerâ€™s Disease. Advances in Behavioral Biology, 1995, , 95-97.	0.2	0
65	Production of amyloid-Î²-peptide by cultured cells: No evidence for internal initiation of translation at Met596. Neurobiology of Aging, 1993, 14, 571-573.	3.1	31
66	Thec4 repressor of bacteriophage P1 is a processed 77 base antisense RNA. Nucleic Acids Research, 1992, 20, 3085-3090.	14.5	30
67	Mutation of the Î²-amyloid precursor protein in familial Alzheimer's disease increases Î²-protein production. Nature, 1992, 360, 672-674.	27.8	1,732
68	The c4 repressors of bacteriophages P1 and P7 are antisense RNAs. Cell, 1990, 62, 591-598.	28.9	48
69	Characterization of Alzheimer's Î²-Secretase Protein BACE: Processing and Other Post-translational Modifications. , 0, , 739-745.		0