FrÃ©dÃ©ric Checler

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/8185480/publications.pdf

Version: 2024-02-01

257 papers	17,026 citations	15001 68 h-index	21239 119 g-index
274 all docs	274 docs citations	274 times ranked	15405 citing authors

#	Article	IF	Citations
1	Mitophagy in Alzheimerâ€™s disease: Molecular defects and therapeutic approaches. Molecular Psychiatry, 2023, 28, 202-216.	4.1	48
2	Neurotrophic fragments as therapeutic alternatives to ameliorate brain aging. Neural Regeneration Research, 2022, Publish Ahead of Print, 2215-2217.	1.6	3
3	Parkin as a Molecular Bridge Linking Alzheimerâ€™s and Parkinsonâ€™s Diseases?. Biomolecules, 2022, 12, 559.	1.8	3
4	Alzheimerâ€™s genetic risk factor FERMT2 (Kindlin-2) controls axonal growth and synaptic plasticity in an APP-dependent manner. Molecular Psychiatry, 2021, 26, 5592-5607.	4.1	28
5	Accumulation ofÂamyloid precursor protein C-terminal fragments triggers mitochondrial structure, function, and mitophagy defects in Alzheimerâ€™s disease models and human brains. Acta Neuropathologica, 2021, 141, 39-65.	3.9	114
6	Is Î³-secretase a beneficial inactivating enzyme of the toxic APP C-terminal fragment C99?. Journal of Biological Chemistry, 2021, 296, 100489.	1.6	32
7	Aminopeptidase A contributes to biochemical, anatomical and cognitive defects in Alzheimerâ€™s disease (AD) mouse model and is increased at early stage in sporadic AD brain. Acta Neuropathologica, 2021, 141, 823-839.	3.9	16
8	Transcription- and phosphorylation-dependent control of a functional interplay between XBP1s and PINK1 governs mitophagy and potentially impacts Parkinson disease pathophysiology. Autophagy, 2021, 17, 4363-4385.	4.3	26
9	MT5â€MMP controls APP and Î²â€CTF/C99 metabolism through proteolyticâ€dependent and â€independent mechanisms relevant for Alzheimer's disease. FASEB Journal, 2021, 35, e21727.	0.2	6
10	Dipeptidyl peptidase 4 contributes to Alzheimerâ€™s diseaseâ€“like defects in a mouse model and is increased in sporadic Alzheimerâ€™s disease brains. Journal of Biological Chemistry, 2021, 297, 100963.	1.6	16
11	Therapeutic potential of parkin as a tumor suppressor via transcriptional control of cyclins in glioblastoma cell and animal models. Theranostics, 2021, 11, 10047-10063.	4.6	7
12	The Endoplasmic Reticulum Stress/Unfolded Protein Response and Their Contributions to Parkinsonâ€™s Disease Physiopathology. Cells, 2020, 9, 2495.	1.8	54
13	Alterations of the Endoplasmic Reticulum (ER) Calcium Signaling Molecular Components in Alzheimerâ€™s Disease. Cells, 2020, 9, 2577.	1.8	32
14	Pyk2 overexpression in postsynaptic neurons blocks amyloid Î²1â€“42-induced synaptotoxicity in microfluidic co-cultures. Brain Communications, 2020, 2, fcaa139.	1.5	13
15	Molecular Dysfunctions of Mitochondria-Associated Membranes (MAMs) in Alzheimerâ€™s Disease. International Journal of Molecular Sciences, 2020, 21, 9521.	1.8	34
16	The Transcription Factor EB Reduces the Intraneuronal Accumulation of the Beta-Secretase-Derived APP Fragment C99 in Cellular and Mouse Alzheimerâ€™s Disease Models. Cells, 2020, 9, 1204.	1.8	10
17	Targeting Post-Translational Remodeling of Ryanodine Receptor: A New Track for Alzheimer's Disease Therapy?. Current Alzheimer Research, 2020, 17, 313-323.	0.7	5
18	Targeting Î³-secretase triggers the selective enrichment of oligomeric APP-CTFs in brain extracellular vesicles from Alzheimer cell and mouse models. Translational Neurodegeneration, 2019, 8, 35.	3.6	28

#	Article	IF	Citations
19	Upregulation of the Sarco-Endoplasmic Reticulum Calcium ATPase 1 Truncated Isoform Plays a Pathogenic Role in Alzheimerâ€™s Disease. Cells, 2019, 8, 1539.	1.8	9
20	Chronic fornix deep brain stimulation in a transgenic Alzheimerâ€™s rat model reduces amyloid burden, inflammation, and neuronal loss. Brain Structure and Function, 2019, 224, 363-372.	1.2	43
21	Proamyloidogenic effects of membrane type 1 matrix metalloproteinase involve MMPâ€2 and BACEâ€1 activities, and the modulation of APP trafficking. FASEB Journal, 2019, 33, 2910-2927.	0.2	25
22	Nuclear p53-mediated repression of autophagy involves PINK1 transcriptional down-regulation. Cell Death and Differentiation, 2018, 25, 873-884.	5.0	87
23	Î²-Amyloid Precursor Protein Intracellular Domain Controls Mitochondrial Function by Modulating Phosphatase and Tensin Homologâ€“Induced Kinase 1 Transcription in Cells and in Alzheimer Mice Models. Biological Psychiatry, 2018, 83, 416-427.	0.7	45
24	Neurolysin: From Initial Detection to Latest Advances. Neurochemical Research, 2018, 43, 2017-2024.	1.6	17
25	Intraneuronal accumulation of C99 contributes to synaptic alterations, apathy-like behavior, and spatial learning deficits in 3Ã—TgAD and 2Ã—TgAD mice. Neurobiology of Aging, 2018, 71, 21-31.	1.5	40
26	Nuclear TP53: An unraveled function as transcriptional repressor of PINK1. Autophagy, 2018, 14, 1-3.	4.3	11
27	Are N- and C-terminally truncated AÎ² species key pathological triggers in Alzheimer's disease?. Journal of Biological Chemistry, 2018, 293, 15419-15428.	1.6	84
28	The Transcription Factor Function of Parkin: Breaking the Dogma. Frontiers in Neuroscience, 2018, 12, 965.	1.4	27
29	Amyloid Î² production is regulated by Î²2-adrenergic signaling-mediated post-translational modifications of the ryanodine receptor. Journal of Biological Chemistry, 2017, 292, 10153-10168.	1.6	50
30	Post-translational remodeling of ryanodine receptor induces calcium leak leading to Alzheimerâ€™s disease-like pathologies and cognitive deficits. Acta Neuropathologica, 2017, 134, 749-767.	3.9	130
31	Genome-wide, high-content siRNA screening identifies the Alzheimerâ€™s genetic risk factor FERMT2 as a major modulator of APP metabolism. Acta Neuropathologica, 2017, 133, 955-966.	3.9	60
32	Presenilins at the crossroad of a functional interplay between PARK2/PARKIN and PINK1 to control mitophagy: Implication for neurodegenerative diseases. Autophagy, 2017, 13, 2004-2005.	4.3	30
33	The transcription factor XBP1s restores hippocampal synaptic plasticity and memory by control of the Kalirin-7 pathway in Alzheimer model. Molecular Psychiatry, 2017, 22, 1562-1575.	4.1	79
34	Î±-synuclein and p53 functional interplay in physiopathological contexts. Oncotarget, 2017, 8, 9001-9002.	0.8	8
35	The Polyherbal Wattana Formula Displays Anti-Amyloidogenic Properties by Increasing Î±-Secretase Activities. PLoS ONE, 2017, 12, e0170360.	1.1	2
36	The Transcription Factor XBP1 in Memory and Cognition: implications in Alzheimerâ€™s Disease. Molecular Medicine, 2016, 22, 905-917.	1.9	27

#	Article	IF	Citations
37	Localization and Processing ofÂtheÂAmyloid-Î² Protein Precursor inÂMitochondria-Associated Membranes. Journal of Alzheimer's Disease, 2016, 55, 1549-1570.	1.2	107
38	Presenilin 1 and Presenilin 2 Target Î³-Secretase Complexes to Distinct Cellular Compartments. Journal of Biological Chemistry, 2016, 291, 12821-12837.	1.6	52
39	AÎ²42 oligomers modulate Î²-secretase through an XBP-1s-dependent pathway involving HRD1. Scientific Reports, 2016, 6, 37436.	1.6	19
40	Intraneuronal aggregation of the Î²-CTF fragment of APP (C99) induces AÎ²-independent lysosomal-autophagic pathology. Acta Neuropathologica, 2016, 132, 257-276.	3.9	158
41	Direct Î±-synuclein promoter transactivation by the tumor suppressor p53. Molecular Neurodegeneration, 2016, 11, 13.	4.4	33
42	ADAM30 Downregulates APP-Linked Defects Through Cathepsin D Activation in Alzheimer's Disease. EBioMedicine, 2016, 9, 278-292.	2.7	40
43	Translational research on cognitive and behavioural disorders in neurological and psychiatric diseases. Therapie, 2016, 71, 15-26.	0.6	3
44	Sox2 functionally interacts with Î²APP, the Î²APP intracellular domain and ADAM10 at a transcriptional level in human cells. Neuroscience, 2016, 312, 153-164.	1.1	21
45	MT5-MMP is a new pro-amyloidogenic proteinase that promotes amyloid pathology and cognitive decline in a transgenic mouse model of Alzheimerâ€™s disease. Cellular and Molecular Life Sciences, 2016, 73, 217-236.	2.4	96
46	MT5-MMP Promotes Alzheimerâ€™s Pathogenesis in the Frontal Cortex of 5xFAD Mice and APP Trafficking in vitro. Frontiers in Molecular Neuroscience, 2016, 9, 163.	1.4	34
47	Influence of Genetic Background on Apathy-Like Behavior in Triple Transgenic AD Mice. Current Alzheimer Research, 2016, 13, 942-949.	0.7	19
48	Melatonin stimulates the nonamyloidogenic processing of Î²<scp>APP</scp> through the positive transcriptional regulation of ADAM10 and ADAM17. Journal of Pineal Research, 2015, 58, 151-165.	3.4	68
49	Eph receptors: New players in Alzheimer's disease pathogenesis. Neurobiology of Disease, 2015, 73, 137-149.	2.1	34
50	Visualization of Specific Î³-Secretase Complexes using Bimolecular Fluorescence Complementation. Journal of Alzheimer's Disease, 2014, 40, 161-176.	1.2	9
51	Differential spatio-temporal regulation of MMPs in the 5xFAD mouse model of AlzheimerÃ¢â‚¬â„¢s disease: evidence for a pro-amyloidogenic role of MT1-MMP. Frontiers in Aging Neuroscience, 2014, 6, 247.	1.7	60
52	The transcription factor X-box binding protein-1 in neurodegenerative diseases. Molecular Neurodegeneration, 2014, 9, 35.	4.4	28
53	Interplay between Parkin and p53 Governs a Physiological Homeostasis That Is Disrupted in Parkinson's Disease and Cerebral Cancer. Neurodegenerative Diseases, 2014, 13, 118-121.	0.8	14
54	Ryanodine receptors. Channels, 2014, 8, 168-168.	1.5	7

#	Article	IF	Citations
55	Experimental stroke: neurolysin back on stage. Journal of Neurochemistry, 2014, 129, 1-3.	2.1	10
56	Study on AÎ²34 biology and detection in transgenic mice brains. Neurobiology of Aging, 2014, 35, 1570-1581.	1.5	17
57	Ryanodine receptors: physiological function and deregulation in Alzheimer disease. Molecular Neurodegeneration, 2014, 9, 21.	4.4	135
58	Glioma tumor grade correlates with parkin depletion in mutant p53-linked tumors and results from loss of function of p53 transcriptional activity. Oncogene, 2014, 33, 1764-1775.	2.6	49
59	p53 in neurodegenerative diseases and brain cancers. , 2014, 142, 99-113.		77
60	Alzheimer's and prion diseases: PDK1 at the crossroads. Nature Medicine, 2013, 19, 1088-1090.	15.2	4
61	Parkin acts as a transcription factor modulating presenilin-1 and presenilin-2 promoter transactivations. Molecular Neurodegeneration, 2013, 8, P56.	4.4	0
62	Leaky Ryanodine receptors increases Amyloid-beta load and induces memory impairments in Tg2576 mouse model of Alzheimer disease. Molecular Neurodegeneration, 2013, 8, P54.	4.4	3
63	The transcription factor XBP-1 in neurodegenerative diseases. Molecular Neurodegeneration, 2013, 8, .	4.4	0
64	N-truncated AÎ² peptides in complex fluids unraveled by new specific immunoassays. Neurobiology of Aging, 2013, 34, 523-539.	1.5	6
65	Further characterization of a putative serine protease contributing to the Î³-secretase cleavage of Î²-amyloid precursor protein. Bioorganic and Medicinal Chemistry, 2013, 21, 1018-1029.	1.4	3
66	ER-stress-associated functional link between Parkin and DJ-1 via a transcriptional cascade involving the tumor suppressor p53 and the spliced X-box binding protein XBP-1. Journal of Cell Science, 2013, 126, 2124-33.	1.2	65
67	Parkin differently regulates presenilin-1 and presenilin-2 functions by direct control of their promoter transcription. Journal of Molecular Cell Biology, 2013, 5, 132-142.	1.5	31
68	6-Hydroxydopamine but not 1-methyl-4-phenylpyridinium abolishes Î±-synuclein anti-apoptotic phenotype by inhibiting its proteasomal degradation and by promoting its aggregation.. Journal of Biological Chemistry, 2013, 288, 21208.	1.6	0
69	Î±-Secretase-derived fragment of cellular prion, N1, protects against monomeric and oligomeric amyloid Î² (AÎ²)-associated cell death.. Journal of Biological Chemistry, 2013, 288, 21210.	1.6	0
70	The disintegrin ADAM9 indirectly contributes to the physiological processing of cellular prion by modulating ADAM10 activity.. Journal of Biological Chemistry, 2013, 288, 23433.	1.6	0
71	Cerebrospinal AÎ²11-x and 17-x levels as indicators of mild cognitive impairment and patientsâ€™ stratification in Alzheimerâ€™s disease. Translational Psychiatry, 2013, 3, e281-e281.	2.4	13
72	α-Secretase in Alzheimers Disease and Beyond: Mechanistic, Regulation and Function in the Shedding of Membrane Proteins. Current Alzheimer Research, 2012, 9, 140-156.	0.7	35

#	Article	IF	Citations
73	Lysosomal Dysfunction in a Mouse Model of Sandhoff Disease Leads to Accumulation of Ganglioside-Bound Amyloid-Î² Peptide. Journal of Neuroscience, 2012, 32, 5223-5236.	1.7	84
74	The Î²-Secretase-Derived C-Terminal Fragment of Î²APP, C99, But Not AÎ², Is a Key Contributor to Early Intraneuronal Lesions in Triple-Transgenic Mouse Hippocampus. Journal of Neuroscience, 2012, 32, 16243-16255.	1.7	168
75	Two-steps control of cellular prion physiology by the Extracellular Regulated Kinase-1 (ERK1). Prion, 2012, 6, 23-25.	0.9	1
76	Î±-Secretase-derived Fragment of Cellular Prion, N1, Protects against Monomeric and Oligomeric Amyloid Î² (AÎ²)-associated Cell Death. Journal of Biological Chemistry, 2012, 287, 5021-5032.	1.6	84
77	p53, a Pivotal Effector of a Functional Cross-Talk Linking Presenilins and Pen-2. Neurodegenerative Diseases, 2012, 10, 52-55.	0.8	7
78	Î±-Secretase-Derived Cleavage of Cellular Prion Yields Biologically Active Catabolites with Distinct Functions. Neurodegenerative Diseases, 2012, 10, 294-297.	0.8	9
79	Evidence that the Amyloid-Î² Protein Precursor Intracellular Domain, AICD, Derives From Î²-Secretase-Generated C-Terminal Fragment. Journal of Alzheimer's Disease, 2012, 30, 145-153.	1.2	73
80	Ryanodine Receptor Blockade Reduces Amyloid-Î² Load and Memory Impairments in Tg2576 Mouse Model of Alzheimer Disease. Journal of Neuroscience, 2012, 32, 11820-11834.	1.7	197
81	Parkin: Much More than a Simple Ubiquitin Ligase. Neurodegenerative Diseases, 2012, 10, 49-51.	0.8	9
82	BACE1 is at the crossroad of a toxic vicious cycle involving cellular stress and Î²-amyloid production in Alzheimerâ€™s disease. Molecular Neurodegeneration, 2012, 7, 52.	4.4	131
83	The caspase 6 derived N-terminal fragment of DJ-1 promotes apoptosis via increased ROS production. Cell Death and Differentiation, 2012, 19, 1769-1778.	5.0	19
84	Nuclear Factor-ÎºB Regulates Î²APP and Î²- and Î³-Secretases Differently at Physiological and Supraphysiological AÎ² Concentrations. Journal of Biological Chemistry, 2012, 287, 24573-24584.	1.6	102
85	The physiology of the Î²â€amyloid precursor protein intracellular domain AICD. Journal of Neurochemistry, 2012, 120, 109-124.	2.1	130
86	Journal of Neurochemistry special issue on Alzheimerâ€™s disease: â€˜amyloid cascade hypothesis â€“ 20â€ƒyears onâ€™. Journal of Neurochemistry, 2012, 120, iii-iv.	2.1	18
87	ERK1-independent Î±-secretase cut of Î²-amyloid precursor protein via M1 muscarinic receptors and PKCÎ±/Îµ. Molecular and Cellular Neurosciences, 2011, 47, 223-232.	1.0	32
88	Î³-Secretase-Mediated Regulation of Neprilysin: Influence of Cell Density and Aging and Modulation by Imatinib. Journal of Alzheimer's Disease, 2011, 27, 511-520.	1.2	31
89	Apoptosis in Parkinson's disease: Is p53 the missing link between genetic and sporadic Parkinsonism?. Cellular Signalling, 2011, 23, 963-968.	1.7	60
90	The Extracellular Regulated Kinase-1 (ERK1) Controls Regulated Î±-Secretase-mediated Processing, Promoter Transactivation, and mRNA Levels of the Cellular Prion Protein. Journal of Biological Chemistry, 2011, 286, 29192-29206.	1.6	22

#	Article	IF	Citations
91	The extracellular regulated kinase-1 (ERK1) controls regulated Î±-secretase-mediated processing, promoter transactivation, and mRNA levels of the cellular prion protein.. Journal of Biological Chemistry, 2011, 286, 33708.	1.6	0
92	p53, a Molecular Bridge Between Alzheimerâ€™s Disease Pathology and Cancers?. Research and Perspectives in Alzheimer's Disease, 2011, , 95-101.	0.1	0
93	Days to criterion as an indicator of toxicity associated with human Alzheimer amyloidâ€Î² oligomers. Annals of Neurology, 2010, 68, 220-230.	2.8	123
94	Loss of function of DJ-1 triggered by Parkinson's disease-associated mutation is due to proteolytic resistance to caspase-6. Cell Death and Differentiation, 2010, 17, 158-169.	5.0	68
95	A novel parkin-mediated transcriptional function links p53 to familial Parkinsonâ€™s disease. Cell Cycle, 2010, 9, 16-17.	1.3	13
96	p53 Is Regulated by and Regulates Members of the Î³-Secretase Complex. Neurodegenerative Diseases, 2010, 7, 50-55.	0.8	38
97	The Î±-Secretase-derived N-terminal Product of Cellular Prion, N1, Displays Neuroprotective Function in Vitro and in Vivo. Journal of Biological Chemistry, 2009, 284, 35973-35986.	1.6	129
98	TMP21 Transmembrane Domain Regulates Î³-Secretase Cleavage. Journal of Biological Chemistry, 2009, 284, 28634-28641.	1.6	23
99	p53-Dependent Transcriptional Control of Cellular Prion by Presenilins. Journal of Neuroscience, 2009, 29, 6752-6760.	1.7	54
100	APH1 Polar Transmembrane Residues Regulate the Assembly and Activity of Presenilin Complexes. Journal of Biological Chemistry, 2009, 284, 16298-16307.	1.6	30
101	p53-dependent control of transactivation of the Pen2 promoter by presenilins. Journal of Cell Science, 2009, 122, 4003-4008.	1.2	21
102	Amyloid-Î²42 is preferentially accumulated in muscle fibers of patients with sporadic inclusion-body myositis. Acta Neuropathologica, 2009, 117, 569-574.	3.9	56
103	Transcriptional repression of p53 by parkin and impairment by mutations associated with autosomal recessive juvenile Parkinson's disease. Nature Cell Biology, 2009, 11, 1370-1375.	4.6	173
104	Aminopeptidase A contributes to the Nâ€terminal truncation of amyloid Î²â€peptide. Journal of Neurochemistry, 2009, 109, 248-256.	2.1	98
105	p53â€Dependent control of cell death by nicastrin: lack of requirement for presenilinâ€dependent Î³â€secretase complex. Journal of Neurochemistry, 2009, 109, 225-237.	2.1	17
106	Pharmacological evidences for DFK167â€sensitive presenilinâ€independent Î³â€secretaseâ€like activity. Journal of Neurochemistry, 2009, 110, 275-283.	2.1	15
107	Mutant Presenilin 1 Increases the Levels of Alzheimer Amyloid Î²-Peptide AÎ²42 in Late Compartments of the Constitutive Secretory Pathway. Journal of Neurochemistry, 2008, 74, 1878-1884.	2.1	38
108	Isoform-specific contribution of protein kinase C to prion processing. Molecular and Cellular Neurosciences, 2008, 39, 400-410.	1.0	20

#	Article	IF	Citations
109	TMP21 regulates AÎ² production but does not affect caspase-3, p53, and neprilysin. Biochemical and Biophysical Research Communications, 2008, 371, 69-74.	1.0	14
110	NFÎºB-dependent Control of BACE1 Promoter Transactivation by AÎ²42. Journal of Biological Chemistry, 2008, 283, 10037-10047.	1.6	117
111	Editorial [Production and Fate of Amyloid Peptides: Recent Advances and Perspectives]. Current Alzheimer Research, 2008, 5, 90-91.	0.7	4
112	Regulation of βAPP and PrPc Cleavage by α-Secretase: Mechanistic and Therapeutic Perspectives. Current Alzheimer Research, 2008, 5, 202-211.	0.7	40
113	Physiological Processing of the Cellular Prion Protein and Î²APP: Enzymes and Regulation. , 2008, , 305-316.		0
114	The C-terminal Products of Cellular Prion Protein Processing, C1 and C2, Exert Distinct Influence on p53-dependent Staurosporine-induced Caspase-3 Activation. Journal of Biological Chemistry, 2007, 282, 1956-1963.	1.6	65
115	p53-dependent Aph-1 and Pen-2 Anti-apoptotic Phenotype Requires the Integrity of the Î³-Secretase Complex but Is Independent of Its Activity. Journal of Biological Chemistry, 2007, 282, 10516-10525.	1.6	24
116	The γ /η-Secretase-Derived APP Intracellular Domain Fragments Regulate p53. Current Alzheimer Research, 2007, 4, 423-426.	0.7	38
117	Study on the Putative Contribution of Caspases and the Proteasome to the Degradation of Aph-1a and Pen-2. Neurodegenerative Diseases, 2007, 4, 156-163.	0.8	4
118	M1 and M3 Muscarinic Receptors Control Physiological Processing of Cellular Prion by Modulating ADAM17 Phosphorylation and Activity. Journal of Neuroscience, 2007, 27, 4083-4092.	1.7	51
119	Response to Correspondence: Pardossi-Piquard etÂal., â€œPresenilin-Dependent Transcriptional Control of the AÎ²-Degrading Enzyme Neprilysin by Intracellular Domains of Î²APP and APLP.â€•Neuron 46, 541â€“554. Neuron, 2007, 53, 483-486.	3.8	21
120	2.109 A novel function of parkin as a transcriptional repressor of the oncogene p53 and its impairment by familial associated Parkinson's disease mutations. Parkinsonism and Related Disorders, 2007, 13, S94.	1.1	0
121	2.112 DJ-1 regulation of p53 pathway and its impairment by Parkinson's disease-associated mutations. Parkinsonism and Related Disorders, 2007, 13, S95.	1.1	0
122	Design and characterization of a novel cellular prion-derived quenched fluorimetric substrate of Î±-secretase. Biochemical and Biophysical Research Communications, 2006, 347, 254-260.	1.0	23
123	Catabolism of endogenous and overexpressed APH1a and PEN2: evidence for artifactual involvement of the proteasome in the degradation of overexpressed proteins. Biochemical Journal, 2006, 394, 501-509.	1.7	25
124	APPÎµ, the Îµ-secretase-derived N-terminal product of the Î²-amyloid precursor protein, behaves as a type I protein and undergoes Î±-, Î²-, and Î³-secretase cleavages. Journal of Neurochemistry, 2006, 97, 807-817.	2.1	21
125	Neprilysin activity and expression are controlled by nicastrin. Journal of Neurochemistry, 2006, 97, 1052-1056.	2.1	39
126	TMP21 is a presenilin complex component that modulates Î³-secretase but not Ê›-secretase activity. Nature, 2006, 440, 1208-1212.	13.7	286

#	Article	IF	Citations
127	Phenotype associated with APP duplication in five families. Brain, 2006, 129, 2966-2976.	3.7	230
128	Presenilin-Dependent Â-Secretase-Mediated Control of p53-Associated Cell Death in Alzheimer's Disease. Journal of Neuroscience, 2006, 26, 6377-6385.	1.7	164
129	6-Hydroxydopamine but Not 1-Methyl-4-phenylpyridinium Abolishes Î±-Synuclein Anti-apoptotic Phenotype by Inhibiting Its Proteasomal Degradation and by Promoting Its Aggregation. Journal of Biological Chemistry, 2006, 281, 9824-9831.	1.6	48
130	Caspase-3-derived C-terminal Product of Synphilin-1 Displays Antiapoptotic Function via Modulation of the p53-dependent Cell Death Pathway. Journal of Biological Chemistry, 2006, 281, 11515-11522.	1.6	34
131	Combined pharmacological, mutational and cell biology approaches indicate that p53-dependent caspaseâ€ƒ3 activation triggered by cellular prion is dependent on its endocytosis. Journal of Neurochemistry, 2005, 92, 1399-1407.	2.1	23
132	Design and characterization of a new cell-permeant inhibitor of the Î² -secretase BACE1. British Journal of Pharmacology, 2005, 145, 228-235.	2.7	33
133	JLK Inhibitors: Isocoumarin Compounds as Putative Probes to Selectively Target the γ-Secretase Pathway. Current Alzheimer Research, 2005, 2, 327-334.	0.7	10
134	Intracellular AÎ²42 activates p53 promoter: a pathway to neurodegeneration in Alzheimer's disease. FASEB Journal, 2005, 19, 1-29.	0.2	244
135	The Disintegrin ADAM9 Indirectly Contributes to the Physiological Processing of Cellular Prion by Modulating ADAM10 Activity. Journal of Biological Chemistry, 2005, 280, 40624-40631.	1.6	101
136	Presenilin-Dependent Transcriptional Control of the AÎ²-Degrading Enzyme Neprilysin by Intracellular Domains of Î²APP and APLP. Neuron, 2005, 46, 541-554.	3.8	317
137	Primary Cultured Neurons Devoid of Cellular Prion Display Lower Responsiveness to Staurosporine through the Control of p53 at Both Transcriptional and Post-transcriptional Levels. Journal of Biological Chemistry, 2004, 279, 612-618.	1.6	62
138	Presenilin-directed inhibitors of gamma-secretase trigger caspase3 activation in presenilin-expressing and presenilin-deficient cells. Journal of Neurochemistry, 2004, 90, 800-806.	2.1	14
139	Increased expression of neuronal cyclooxygenase-2 in the hippocampus in amyotrophic lateral sclerosis both with and without dementia. Acta Neuropathologica, 2004, 107, 399-405.	3.9	17
140	P1-209 APH-1 and PEN-2: a study on their proteolysis. Neurobiology of Aging, 2004, 25, S155.	1.5	0
141	C-terminal fragments of amyloid-beta peptide cause cholinergic axonal degeneration by a toxic effect rather than by physical injury in the nondemented human brain. Neurochemical Research, 2003, 28, 493-498.	1.6	3
142	Variability and heterogeneity in Alzheimer's disease with cotton wool plaques: a clinicopathological study of four autopsy cases. Acta Neuropathologica, 2003, 106, 348-356.	3.9	29
143	JLK isocoumarin inhibitors: Selective ?-secretase inhibitors that do not interfere with notch pathway in vitro or in vivo. Journal of Neuroscience Research, 2003, 74, 370-377.	1.3	43
144	Synthesis of new 3-alkoxy-7-amino-4-chloro-isocoumarin derivatives as new Î²-amyloid peptide production inhibitors and their activities on various classes of protease. Bioorganic and Medicinal Chemistry, 2003, 11, 3141-3152.	1.4	44

#	Article	IF	Citations
145	Cyclooxygenase-2 in the hippocampus is up-regulated in Alzheimer's disease but not in variant Alzheimer's disease with cotton wool plaques in humans. Neuroscience Letters, 2003, 343, 175-179.	1.0	19
146	Cellular Prion Protein Sensitizes Neurons to Apoptotic Stimuli through Mdm2-regulated and p53-dependent Caspase 3-like Activation. Journal of Biological Chemistry, 2003, 278, 10061-10066.	1.6	93
147	Î²-Synuclein Displays an Antiapoptotic p53-dependent Phenotype and Protects Neurons from 6-Hydroxydopamine-induced Caspase 3 Activation. Journal of Biological Chemistry, 2003, 278, 37330-37335.	1.6	70
148	The C-terminal Fragment of Presenilin 2 Triggers p53-mediated Staurosporine-induced Apoptosis, a Function Independent of the Presenilinase-derived N-terminal Counterpart. Journal of Biological Chemistry, 2003, 278, 12064-12069.	1.6	50
149	BACE1- and BACE2-expressing Human Cells. Journal of Biological Chemistry, 2003, 278, 25859-25866.	1.6	68
150	Î±-Synuclein Lowers p53-dependent Apoptotic Response of Neuronal Cells. Journal of Biological Chemistry, 2002, 277, 50980-50984.	1.6	119
151	Wild-type and mutated presenilins 2 trigger p53-dependent apoptosis and down-regulate presenilin 1 expression in HEK293 human cells and in murine neurons. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 4043-4048.	3.3	129
152	Î³-Secretase-like Cleavages of Notch and Î²APP Are Mutually Exclusive in Human Cells. Biochemical and Biophysical Research Communications, 2002, 290, 1408-1410.	1.0	14
153	Murine T cells expressing high activity of prolyl endopeptidase are susceptible to activation-induced cell death. FEBS Letters, 2002, 512, 163-167.	1.3	15
154	Human amyloid-Î² causes changes in the levels of endothelial protein kinase C and its Î± isoform in vitro. Neurochemistry International, 2002, 41, 409-414.	1.9	27
155	Alzheimer's and prion diseases: distinct pathologies, common proteolytic denominators. Trends in Neurosciences, 2002, 25, 616-620.	4.2	92
156	MÃ©tabolisme du prÃ©curseur du peptide amyloÃ¯de et prÃ©sÃ©nilines. Medecine/Sciences, 2002, 18, 717-724	0.0	7
157	Amyloid Precursor Protein, Presenilins, and alpha -Synuclein: Molecular Pathogenesis and Pharmacological Applications in Alzheimer's Disease. Pharmacological Reviews, 2002, 54, 469-525.	7.1	421
158	NACP/Î±-Synuclein, NAC, and Î²-amyloid pathology of familial Alzheimer's disease with the E184D presenilin-1 mutation: a clinicopathological study of two autopsy cases. Acta Neuropathologica, 2002, 104, 637-648.	3.9	37
159	Overexpression of PrPc triggers caspaseâ€ƒ3 activation: potentiation by proteasome inhibitors and blockade by anti-PrP antibodies. Journal of Neurochemistry, 2002, 83, 1208-1214.	2.1	65
160	Influence of Region-Specific Alterations of Neuropeptidase Content on the Catabolic Fates of Neuropeptides in Alzheimer's Disease. Journal of Neurochemistry, 2002, 62, 645-655.	2.1	46
161	Amyloid-lowering isocoumarins are not direct inhibitors of Î³-secretase - Reponse. Nature Cell Biology, 2002, 4, E111-E112.	4.6	5
162	Reply: Potential external source of AÎ² in biological samples. Nature Cell Biology, 2002, 4, E165-E166.	4.6	1

#	Article	IF	Citations
163	Wild-Type and Mutated Nicastrins Do Not Display Aminopeptidase M- and B-like Activities. Biochemical and Biophysical Research Communications, 2001, 289, 678-680.	1.0	11
164	Constitutive Î±-secretase cleavage of the Î²-amyloid precursor protein in the furin-deficient LoVo cell line: involvement of the pro-hormone convertase 7 and the disintegrin metalloprotease ADAM10. Journal of Neurochemistry, 2001, 76, 1532-1539.	2.1	113
165	The multiple paradoxes of presenilins. Journal of Neurochemistry, 2001, 76, 1621-1627.	2.1	45
166	The caspase-derived C-terminal fragment of Î²APP induces caspase-independent toxicity and triggers selective increase of AÎ²42 in mammalian cells. Journal of Neurochemistry, 2001, 78, 1153-1161.	2.1	33
167	The C-terminal fragment of the Alzheimer's disease amyloid protein precursor is degraded by a proteasome-dependent mechanism distinct from Î³-secretase. FEBS Journal, 2001, 268, 5329-5336.	0.2	116
168	Endogenous Î²-amyloid production in presenilin-deficient embryonic mouse fibroblasts. Nature Cell Biology, 2001, 3, 1030-1033.	4.6	94
169	New protease inhibitors prevent Î³-secretase-mediated production of AÎ²40/42 without affecting Notch cleavage. Nature Cell Biology, 2001, 3, 507-511.	4.6	181
170	The Disintegrins ADAM10 and TACE Contribute to the Constitutive and Phorbol Ester-regulated Normal Cleavage of the Cellular Prion Protein. Journal of Biological Chemistry, 2001, 276, 37743-37746.	1.6	222
171	Pathogenic APP mutations near the gamma-secretase cleavage site differentially affect Abeta secretion and APP C-terminal fragment stability. Human Molecular Genetics, 2001, 10, 1665-1671.	1.4	178
172	Post-translational Processing of Î²-Secretase (Î²-Amyloid-converting Enzyme) and Its Ectodomain Shedding. Journal of Biological Chemistry, 2001, 276, 10879-10887.	1.6	273
173	Novel proline endopeptidase inhibitors do not modify AÎ²40/42 formation and degradation by human cells expressing wild-type and Swedish mutated Î²-amyloid precursor protein. British Journal of Pharmacology, 2000, 130, 1613-1617.	2.7	18
174	Aging Increased Amyloid Peptide and Caused Amyloid Plaques in Brain of Old APP/V717I Transgenic Mice by a Different Mechanism than Mutant Presenilin1. Journal of Neuroscience, 2000, 20, 6452-6458.	1.7	107
175	Behavioral alterations associated with apoptosis and down-regulation of presenilin 1 in the brains of p53-deficient mice. Proceedings of the National Academy of Sciences of the United States of America, 2000, 97, 5346-5350.	3.3	58
176	Wild-type but Not Parkinson's Disease-related Ala-53 â†' Thr Mutant Î±-Synuclein Protects Neuronal Cells from Apoptotic Stimuli. Journal of Biological Chemistry, 2000, 275, 24065-24069.	1.6	198
177	Phorbol Ester-regulated Cleavage of Normal Prion Protein in HEK293 Human Cells and Murine Neurons. Journal of Biological Chemistry, 2000, 275, 35612-35616.	1.6	98
178	Overexpression of Rab11 or Constitutively Active Rab11 Does Not Affect sAPPÎ± and AÎ² Secretions by Wild-Type and Swedish Mutated Î²APP-Expressing HEK293 Cells. Biochemical and Biophysical Research Communications, 2000, 275, 910-915.	1.0	8
179	Immunohistochemical Analysis of Cerebral Cortical and Vascular Lesions in the Primate Microcebus murinus Reveal Distinct Amyloid Î²1â€“42 and Î²1â€“40 Immunoreactivity Profiles. Neurobiology of Disease, 2000, 7, 1-8.	2.1	58
180	Behavioral Disturbances without Amyloid Deposits in Mice Overexpressing Human Amyloid Precursor Protein with Flemish (A692G) or Dutch (E693Q) Mutation. Neurobiology of Disease, 2000, 7, 9-22.	2.1	100

#	Article	IF	Citations
181	Prominent Cerebral Amyloid Angiopathy in Transgenic Mice Overexpressing the London Mutant of Human APP in Neurons. American Journal of Pathology, 2000, 157, 1283-1298.	1.9	213
182	Intraneuronal AÎ²42 Accumulation in Human Brain. American Journal of Pathology, 2000, 156, 15-20.	1.9	930
183	Î±-Synuclein and the Parkinson's disease-related mutant Ala53Thr-Î±-synuclein do not undergo proteasomal degradation in HEK293 and neuronal cells. Neuroscience Letters, 2000, 285, 79-82.	1.0	121
184	Vascular and parenchymal AÃŽÂ² deposition in the aging dog: correlation with behavior. Neurobiology of Aging, 2000, 21, 695-704.	1.5	133
185	Role of the proteasome in Alzheimerâ€™s disease. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2000, 1502, 133-138.	1.8	89
186	Laminar specific loss of isocortical presenilin 1 immunoreactivity in Alzheimer's disease. Correlations with the amyloid load and the density of tau-positive neurofibrillary tangles. Neuropathology and Applied Neurobiology, 2000, 26, 117-123.	1.8	10
187	C-Terminal Maturation Fragments of Presenilin 1 and 2 Control Secretion of APPÎ± and AÎ² by Human Cells and Are Degraded by Proteasome. Molecular Medicine, 1999, 5, 160-168.	1.9	26
188	Unusual phenotypic alteration of Â amyloid precursor protein (ÂAPP) maturation by a new Val-715 -> Met ÂAPP-770 mutation responsible for probable early-onset Alzheimer's disease. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 4119-4124.	3.3	183
189	Presenilins: Multifunctional Proteins Involved in Alzheimer's Disease Pathology. IUBMB Life, 1999, 48, 33-39.	1.5	35
190	Presenilin 2 deficiency causes a mild pulmonary phenotype and no changes in amyloid precursor protein processing but enhances the embryonic lethal phenotype of presenilin 1 deficiency. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 11872-11877.	3.3	481
191	Early Phenotypic Changes in Transgenic Mice That Overexpress Different Mutants of Amyloid Precursor Protein in Brain. Journal of Biological Chemistry, 1999, 274, 6483-6492.	1.6	611
192	Transgenic mice with Alzheimer presenilin 1 mutations show accelerated neurodegeneration without amyloid plaque formation. Nature Medicine, 1999, 5, 560-564.	15.2	355
193	Effect of protein kinase A inhibitors on the production of AÎ²40 and AÎ²42 by human cells expressing normal and Alzheimer's disease-linked mutated Î²APP and presenilin 1. British Journal of Pharmacology, 1999, 126, 1186-1190.	2.7	24
194	Presenilins: Multifunctional Proteins Involved in Alzheimer's Disease Pathology. IUBMB Life, 1999, 48, 33-39.	1.5	41
195	Presenilins. Molecular Neurobiology, 1999, 19, 255-265.	1.9	26
196	Endoplasmic reticulum and trans-Golgi network generate distinct populations of Alzheimer beta -amyloid peptides. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 742-747.	3.3	354
197	Confocal Microscopy Reveals Thimet Oligopeptidase (EC 3.4.24.15) and Neurolysin (EC 3.4.24.16) in the Classical Secretory Pathway. DNA and Cell Biology, 1999, 18, 323-331.	0.9	33
198	S 17092-1, a Highly Potent, Specific and Cell Permeant Inhibitor of Human Proline Endopeptidase. Biochemical and Biophysical Research Communications, 1999, 257, 657-661.	1.0	57

#	Article	IF	Citations
199	Estrogen reduces neuronal generation of Alzheimer Î²-amyloid peptides. Nature Medicine, 1998, 4, 447-451.	15.2	545
200	Neuropathology of preclinical and clinical lateonset Alzheimer's disease. Annals of Neurology, 1998, 43, 673-676.	2.8	83
201	Neuropeptide Specificity and Inhibition of Recombinant Isoforms of the Endopeptidase 3.4.24.16 Family: Comparison with the Related Recombinant Endopeptidase 3.4.24.15. Biochemical and Biophysical Research Communications, 1998, 250, 5-11.	1.0	80
202	Alzheimer's Disease-Linked Mutation of Presenilin 2 (N141I-PS2) Drastically Lowers APPÎ± Secretion: Control by the Proteasome. Biochemical and Biophysical Research Communications, 1998, 252, 134-138.	1.0	42
203	Proteasome Inhibitors Prevent the Degradation of Familial Alzheimerâ€™s Disease-Linked Presenilin 1 and Potentiate AÎ²42 Recovery from Human Cells. Molecular Medicine, 1998, 4, 147-157.	1.9	67
204	Contribution of endopeptidase 3.4.24.15 to central neurotensin inactivation. European Journal of Pharmacology, 1997, 334, 49-53.	1.7	35
205	Lack of effect of Presenilin 1, Î²APP and their Alzheimer's disease-related mutated forms on Xenopus oocytes membrane currents. Neuroscience Letters, 1997, 221, 85-88.	1.0	7
206	Characterization of New Polyclonal Antibodies Specific for 40 and 42 Amino Acid-Long Amyloid Î² Peptides: Their Use to Examine the Cell Biology of Presenilins and the Immunohistochemistry of Sporadic Alzheimerâ€™s Disease and Cerebral Amyloid Angiopathy Cases. Molecular Medicine, 1997, 3, 695-707.	1.9	142
207	Examination of the role of endopeptidase 3.4.24.15 in AÎ² secretion by human transfected cells. British Journal of Pharmacology, 1997, 121, 556-562.	2.7	37
208	Effect of a novel selective and potent phosphinic peptide inhibitor of endopeptidase 3.4.24.16 on neurotensin-induced analgesia and neuronal inactivation. British Journal of Pharmacology, 1997, 121, 705-710.	2.7	34
209	Cathepsin D displays in vitro Î²-secretase-like specificity. Brain Research, 1997, 750, 11-19.	1.1	94
210	Proteasome Contributes to the Î±â€Secretase Pathway of Amyloid Precursor Protein in Human Cells. Journal of Neurochemistry, 1997, 68, 698-703.	2.1	48
211	Stably Transfected Human Cells Overexpressing Rat Brain Endopeptidase 3.4.24.16: Biochemical Characterization of the Activity and Expression of Soluble and Membraneâ€Associated Counterparts. Journal of Neurochemistry, 1997, 68, 837-845.	2.1	18
212	Î±â€Secretaseâ€Derived Product of Î²â€Amyloid Precursor Protein Is Decreased by Presenilin 1 Mutations Linked to Familial Alzheimer's Disease. Journal of Neurochemistry, 1997, 69, 2494-2499.	2.1	63
213	Constitutive and Protein Kinase Câ€Regulated Secretory Cleavage of Alzheimer's Î²â€Amyloid Precursor Protein: Different Control of Early and Late Events by the Proteasome. Journal of Neurochemistry, 1997, 69, 2500-2505.	2.1	41
214	Contribution of the Proteasome to the Î±-Secretase Pathway in Alzheimerâ€™s Disease. Advances in Experimental Medicine and Biology, 1997, 421, 267-272.	0.8	5
215	Distinct Properties of Neuronal and Astrocytic Endopeptidase 3.4.24.16: A Study on Differentiation, Subcellular Distribution, and Secretion Processes. Journal of Neuroscience, 1996, 16, 5049-5059.	1.7	68
216	Purification and characterization of human endopeptidase 3.4.24.16. Comparison with the porcine counterpart indicates a unique cleavage site on neurotensin. Brain Research, 1996, 709, 51-58.	1.1	19

#	Article	IF	Citations
217	Development of the First Potent and Selective Inhibitor of the Zinc Endopeptidase Neurolysin Using a Systematic Approach Based on Combinatorial Chemistry of Phosphinic Peptides. Journal of Biological Chemistry, 1996, 271, 19606-19611.	1.6	86
218	Protein Kinase A Phosphorylation of the Proteasome: A Contribution to the Î±â€Secretase Pathway in Human Cells. Journal of Neurochemistry, 1996, 67, 2616-2619.	2.1	36
219	Molecular Cloning and Expression of Rat Brain Endopeptidase 3.4.24.16. Journal of Biological Chemistry, 1995, 270, 27266-27271.	1.6	64
220	Development of Highly Potent and Selective Phosphinic Peptide Inhibitors of Zinc Endopeptidase 24-15 Using Combinatorial Chemistry. Journal of Biological Chemistry, 1995, 270, 21701-21706.	1.6	104
221	Phosphorusâ€containing peptides as mixed inhibitors of endopeptidase 3.4.24.15 and 3.4.24.16: effect on neurotensin degradation in vitro and in vivo. British Journal of Pharmacology, 1995, 115, 1053-1063.	2.7	31
222	Processing of the Î²â€Amyloid Precursor Protein and Its Regulation in Alzheimer's Disease. Journal of Neurochemistry, 1995, 65, 1431-1444.	2.1	402
223	Neurotensin and neuromedin N undergo distinct catabolic processes in murine astrocytes and primary cultured neurons. FEBS Journal, 1994, 221, 297-306.	0.2	16
224	Cholinesterases Display Genuine Arylacylamidase Activity but Are Totally Devoid of Intrinsic Peptidase Activities. Journal of Neurochemistry, 1994, 62, 756-763.	2.1	28
225	Rat kidney endopeptidase 24.16. Purification, physico-chemical characteristics and differential specificity towards opiates, tachykinins and neurotensin-related peptides. FEBS Journal, 1993, 211, 79-90.	0.2	33
226	Synthesis and analgesic effects of N-[3-[(hydroxyamino)carbonyl]-1-oxo-2(R)-benzylpropyl]-L-isoleucyl-L-leucine, a new potent inhibitor of multiple neurotensin/neuromedin N degrading enzymes. Journal of Medicinal Chemistry, 1993, 36, 1369-1379.	2.9	35
227	Differential catabolic fate of neuromedin N and neurotensin in the canine intestinal mucosa. Peptides, 1993, 14, 457-463.	1.2	3
228	A survey of the cerebral regionalization and ontogeny of eight exo- and endopeptidases in murines. Peptides, 1993, 14, 593-599.	1.2	34
229	Neurotensin Receptors in Primary Culture of Neurons. Methods in Neurosciences, 1993, 11, 334-351.	0.5	4
230	Neurotensin Receptor Localization on Neurons Bearing the Neurotensin-Degrading Enzyme Endopeptidase 24-16. Annals of the New York Academy of Sciences, 1992, 668, 326-328.	1.8	1
231	Neurotensin and Neuromedin N Undergo Distinct Proteolytic Inactivation in Ventral Tegmental Area and Nucleus Accumbens. Annals of the New York Academy of Sciences, 1992, 668, 329-332.	1.8	0
232	Light and Electron Microscopic Localization of the Neutral Metalloendopeptidase EC 3.4.24.16 in the Mesencephalon of the Rat. European Journal of Neuroscience, 1992, 4, 1309-1319.	1.2	33
233	Endopeptidase 24-16 in Murines: Tissue Distribution, Cerebral Regionalization, and Ontogeny. Journal of Neurochemistry, 1992, 59, 1862-1867.	2.1	27
234	Neurotensin and Neuromedin N Are Differently Metabolized in Ventral Tegmental Area and Nucleus Accumbens. Journal of Neurochemistry, 1991, 56, 1320-1328.	2.1	22

#	Article	IF	Citations
235	Specific inhibition of endopeptidase 24.16 by dipeptides. FEBS Journal, 1991, 202, 269-276.	0.2	57
236	Monoclonal Antibodies Allow Precipitation of Esterasic but Not Peptidasic Activities Associated with Butyrylcholinesterase. Journal of Neurochemistry, 1990, 55, 750-755.	2.1	22
237	Non-cholinergic actions of acetylcholinesterases: a genuine peptidase function or contaminating proteases?. Trends in Biochemical Sciences, 1990, 15, 337-338.	3.7	9
238	Peptidasic Activities Associated with Acetylcholinesterase Are Due to Contaminating Enzymes. Journal of Neurochemistry, 1989, 53, 924-928.	2.1	40
239	Neuropeptide-hydrolysing activities in synaptosomal fractions from dog ileum myenteric, deep muscular and submucous plexi. Their participation in neurotensin inactivation. Peptides, 1989, 10, 1055-1061.	1.2	12
240	Peripheral inactivation of neurotensin. Isolation and characterization of a metallopeptidase from rat ileum. FEBS Journal, 1988, 175, 481-489.	0.2	35
241	Neurotensin metabolism in various tissues of central and peripheral origins: ubiquitous involvement of a novel neurotensin degrading metalloendopeptidase. Biochimie, 1988, 70, 75-82.	1.3	53
242	Further characterization of a neurotensin-degrading neutral metalloendopeptidase from rat brain. Neurochemistry International, 1988, 12, 351-359.	1.9	29
243	Neurotensin-Metabolizing Peptidases in Rat Fundus Plasma Membranes. Journal of Neurochemistry, 1987, 49, 507-512.	2.1	10
244	Peptidases in dog-ileum circular and longitudinal smooth-muscle plasma membranes. Their relative contribution to the metabolism of neurotensin. FEBS Journal, 1987, 166, 461-468.	0.2	16
245	Neuromedin N: High affinity interaction with brain neurotensin receptors and rapid inactivation by brain synaptic peptidases. European Journal of Pharmacology, 1986, 126, 239-244.	1.7	75
246	Catabolism of neurotensin by neural (neuroblastoma clone N1E115) and extraneural (HT29) cell lines. Peptides, 1986, 7, 1071-1077.	1.2	33
247	High-Affinity Receptor Sites and Rapid Proteolytic Inactivation of Neurotensin in Primary Cultured Neurons. Journal of Neurochemistry, 1986, 47, 1742-1748.	2.1	45
248	Inactivation of Neurotensin by Rat Brain Synaptic Membranes Partly Occurs Through Cleavage at the Arg8-Arg9Peptide Bond by a Metalloendopeptidase. Journal of Neurochemistry, 1985, 45, 1509-1513.	2.1	81
249	Regulation of cyclic GMP levels by neurotensin in neuroblastoma clone N1E115. Biochemical and Biophysical Research Communications, 1985, 129, 117-125.	1.0	53
250	Inactivation of Neurotensin by Rat Brain Synaptic Membranes. Cleavage at the Pro10-Tyr11Bond by Endopeptidase 24.11 (Enkephalinase) and a Peptidase Different from Proline-Endopeptidase. Journal of Neurochemistry, 1984, 43, 1295-1301.	2.1	80
251	Loss of high affinity neurotensin receptors in substantia nigra from parkinsonian subjects. Biochemical and Biophysical Research Communications, 1984, 125, 395-404.	1.0	71
252	Comparison of some biological properties of neurotensin and its natural analogue LANT-6. European Journal of Pharmacology, 1984, 99, 357-360.	1.7	10

#	Article	IF	Citations
253	Degradation of Neurotensin by Rat Brain Synaptic Membranes: Involvement of a Thermolysin-Like Metalloendopeptidase (Enkephalinase), Angiotensin-Converting Enzyme, and Other Unidentified Peptidases. Journal of Neurochemistry, 1983, 41, 375-384.	2.1	147
254	[TRP11]-Neurotensin and xenopsin discriminate between rat and guinea-pig neurotensin receptors. Life Sciences, 1982, 31, 1145-1150.	2.0	21
255	DEGRADATION OF NEUROTENSIN BY BRAIN SYNAPTIC MEMBRANES. Annals of the New York Academy of Sciences, 1982, 400, 413-414.	1.8	8
256	MONOIODO-Trp11-NEUROTENSIN, A NEW LIGAND TO STUDY THE INTERACTION OF NEUROTENSIN WITH ITS RECEPTOR. Annals of the New York Academy of Sciences, 1982, 400, 436-437.	1.8	1
257	Insulin stimulation of amino acid transport in primary cultured rat hepatocytes varies in direct proportion to insulin binding. FEBS Letters, 1981, 128, 321-324.	1.3	8