RaphaÃ«lle Pardossi-Piquard

List of Publications by Year
in descending order

29 papers	3,034 citations	331670 21 h-index	501196 28 g-index
30 all docs	30 docs citations	30 times ranked	4320 citing authors

#	Article	IF	Citations
1	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.	7.7	114
2	Is Î³-secretase a beneficial inactivating enzyme of the toxic APP C-terminal fragment C99?. Journal of Biological Chemistry, 2021, 296, 100489.	3.4	32
3	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.	4.1	10
4	Palmitate Is Increased in the Cerebrospinal Fluid of Humans with Obesity and Induces Memory Impairment in Mice via Pro-inflammatory TNF-Î±. Cell Reports, 2020, 30, 2180-2194.e8.	6.4	80
5	Does Intraneuronal Accumulation of Carboxyl-terminal Fragments of the Amyloid Precursor Protein Trigger Early Neurotoxicity in Alzheimerâ€™s Disease?. Current Alzheimer Research, 2019, 16, 453-457.	1.4	41
6	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.	8.0	28
7	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.	1.3	45
8	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.	3.1	40
9	Intraneuronal aggregation of the Î²-CTF fragment of APP (C99) induces AÎ²-independent lysosomal-autophagic pathology. Acta Neuropathologica, 2016, 132, 257-276.	7.7	158
10	Influence of Genetic Background on Apathy-Like Behavior in Triple Transgenic AD Mice. Current Alzheimer Research, 2016, 13, 942-949.	1.4	19
11	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.	3.6	168
12	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.	2.6	73
13	The physiology of the Î²â€amyloid precursor protein intracellular domain AICD. Journal of Neurochemistry, 2012, 120, 109-124.	3.9	130
14	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.	2.6	31
15	p53, a Molecular Bridge Between Alzheimerâ€™s Disease Pathology and Cancers?. Research and Perspectives in Alzheimer's Disease, 2011, , 95-101.	0.1	0
16	p53 Is Regulated by and Regulates Members of the Î³-Secretase Complex. Neurodegenerative Diseases, 2010, 7, 50-55.	1.4	38
17	TMP21 Transmembrane Domain Regulates Î³-Secretase Cleavage. Journal of Biological Chemistry, 2009, 284, 28634-28641.	3.4	23
18	APH1 Polar Transmembrane Residues Regulate the Assembly and Activity of Presenilin Complexes. Journal of Biological Chemistry, 2009, 284, 16298-16307.	3.4	30

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19	p53-dependent control of transactivation of the Pen2 promoter by presenilins. Journal of Cell Science, 2009, 122, 4003-4008.	2.0	21
20	p53â€Dependent control of cell death by nicastrin: lack of requirement for presenilinâ€dependent Î³â€secretase complex. Journal of Neurochemistry, 2009, 109, 225-237.	3.9	17
21	A novel presenilin 2 mutation (V393M) in earlyâ€onset dementia with profound language impairment. European Journal of Neurology, 2008, 15, 1135-1139.	3.3	19
22	The γ /η-Secretase-Derived APP Intracellular Domain Fragments Regulate p53. Current Alzheimer Research, 2007, 4, 423-426.	1.4	38
23	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.	8.1	21
24	Overexpression of Human CRB1 or Related Isoforms, CRB2 and CRB3, Does Not Regulate the Human Presenilin Complex in Culture Cells. Biochemistry, 2007, 46, 13704-13710.	2.5	7
25	The neuronal sortilin-related receptor SORL1 is genetically associated with Alzheimer disease. Nature Genetics, 2007, 39, 168-177.	21.4	1,045
26	Neprilysin activity and expression are controlled by nicastrin. Journal of Neurochemistry, 2006, 97, 1052-1056.	3.9	39
27	TMP21 is a presenilin complex component that modulates Î³-secretase but not É›-secretase activity. Nature, 2006, 440, 1208-1212.	27.8	286
28	Presenilin-Dependent Â-Secretase-Mediated Control of p53-Associated Cell Death in Alzheimer's Disease. Journal of Neuroscience, 2006, 26, 6377-6385.	3.6	164
29	Presenilin-Dependent Transcriptional Control of the AÎ²-Degrading Enzyme Neprilysin by Intracellular Domains of Î²APP and APLP. Neuron, 2005, 46, 541-554.	8.1	317