## Raphaëlle Pardossi-Piquard

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	The neuronal sortilin-related receptor SORL1 is genetically associated with Alzheimer disease. Nature Genetics, 2007, 39, 168-177.	21.4	1,045
2	Presenilin-Dependent Transcriptional Control of the Aβ-Degrading Enzyme Neprilysin by Intracellular Domains of βAPP and APLP. Neuron, 2005, 46, 541-554.	8.1	317
3	TMP21 is a presenilin complex component that modulates Î <sup>3</sup> -secretase but not É>-secretase activity. Nature, 2006, 440, 1208-1212.	27.8	286
4	The β-Secretase-Derived C-Terminal Fragment of βAPP, C99, But Not Aβ, Is a Key Contributor to Early Intraneuronal Lesions in Triple-Transgenic Mouse Hippocampus. Journal of Neuroscience, 2012, 32, 16243-16255.	3.6	168
5	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
6	Intraneuronal aggregation of the β-CTF fragment of APP (C99) induces Aβ-independent lysosomal-autophagic pathology. Acta Neuropathologica, 2016, 132, 257-276.	7.7	158
7	The physiology of the βâ€∎myloid precursor protein intracellular domain AICD. Journal of Neurochemistry, 2012, 120, 109-124.	3.9	130
8	Accumulation ofÂamyloid precursor protein C-terminal fragments triggers mitochondrial structure, function, and mitophagy defects in Alzheimer's disease models and human brains. Acta Neuropathologica, 2021, 141, 39-65.	7.7	114
9	Palmitate Is Increased in the Cerebrospinal Fluid of Humans with Obesity and Induces Memory Impairment in Mice via Pro-inflammatory TNF-α. Cell Reports, 2020, 30, 2180-2194.e8.	6.4	80
10	Evidence that the Amyloid-β Protein Precursor Intracellular Domain, AICD, Derives From β-Secretase-Generated C-Terminal Fragment. Journal of Alzheimer's Disease, 2012, 30, 145-153.	2.6	73
11	β-Amyloid Precursor Protein Intracellular Domain Controls Mitochondrial Function by Modulating Phosphatase and Tensin Homolog–Induced Kinase 1 Transcription in Cells and in Alzheimer Mice Models. Biological Psychiatry, 2018, 83, 416-427.	1.3	45
12	Does Intraneuronal Accumulation of Carboxyl-terminal Fragments of the Amyloid Precursor Protein Trigger Early Neurotoxicity in Alzheimer's Disease?. Current Alzheimer Research, 2019, 16, 453-457.	1.4	41
13	Intraneuronal accumulation of C99 contributes to synaptic alterations, apathy-like behavior, and spatial learning deficits in 3×TgAD and 2×TgAD mice. Neurobiology of Aging, 2018, 71, 21-31.	3.1	40
14	Neprilysin activity and expression are controlled by nicastrin. Journal of Neurochemistry, 2006, 97, 1052-1056.	3.9	39
15	The γ /η-Secretase-Derived APP Intracellular Domain Fragments Regulate p53. Current Alzheimer Research, 2007, 4, 423-426.	1.4	38
16	p53 Is Regulated by and Regulates Members of the Î <sup>3</sup> -Secretase Complex. Neurodegenerative Diseases, 2010, 7, 50-55.	1.4	38
17	Is Î <sup>3</sup> -secretase a beneficial inactivating enzyme of the toxic APP C-terminal fragment C99?. Journal of Biological Chemistry, 2021, 296, 100489.	3.4	32
18	γ-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

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19	APH1 Polar Transmembrane Residues Regulate the Assembly and Activity of Presenilin Complexes. Journal of Biological Chemistry, 2009, 284, 16298-16307.	3.4	30
20	Targeting Î <sup>3</sup> -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
21	TMP21 Transmembrane Domain Regulates γ-Secretase Cleavage. Journal of Biological Chemistry, 2009, 284, 28634-28641.	3.4	23
22	Response to Correspondence: Pardossi-Piquard etÂal., "Presenilin-Dependent Transcriptional Control of the Aβ-Degrading Enzyme Neprilysin by Intracellular Domains of βAPP and APLP.―Neuron 46, 541–554. Neuron, 2007, 53, 483-486.	8.1	21
23	p53-dependent control of transactivation of the Pen2 promoter by presenilins. Journal of Cell Science, 2009, 122, 4003-4008.	2.0	21
24	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
25	Influence of Genetic Background on Apathy-Like Behavior in Triple Transgenic AD Mice. Current Alzheimer Research, 2016, 13, 942-949.	1.4	19
26	p53â€Dependent control of cell death by nicastrin: lack of requirement for presenilinâ€dependent γâ€secretase complex. Journal of Neurochemistry, 2009, 109, 225-237.	3.9	17
27	The Transcription Factor EB Reduces the Intraneuronal Accumulation of the Beta-Secretase-Derived APP Fragment C99 in Cellular and Mouse Alzheimer's Disease Models. Cells, 2020, 9, 1204.	4.1	10
28	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
29	p53, a Molecular Bridge Between Alzheimer's Disease Pathology and Cancers?. Research and Perspectives in Alzheimer's Disease, 2011, , 95-101.	0.1	Ο