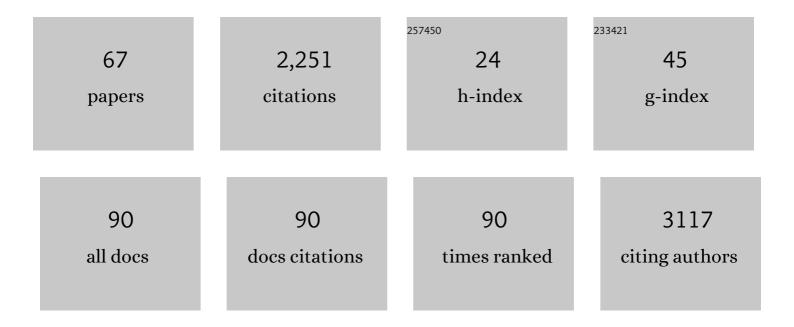
Pascal Kienlen-Campard

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	An evaluation of the self-assembly enhancing properties of cell-derived hexameric amyloid-β. Scientific Reports, 2021, 11, 11570.	3.3	9
2	Overexpression of wild-type human amyloid precursor protein alters GABAergic transmission. Scientific Reports, 2021, 11, 17600.	3.3	11
3	How to Build and to Protect the Neuromuscular Junction: The Role of the Glial Cell Line-Derived Neurotrophic Factor. International Journal of Molecular Sciences, 2021, 22, 136.	4.1	16
4	Mechanism of Cellular Formation and In Vivo Seeding Effects of Hexameric β-Amyloid Assemblies. Molecular Neurobiology, 2021, 58, 6647-6669.	4.0	8
5	Presenilin-Deficient Neurons and Astrocytes Display Normal Mitochondrial Phenotypes. Frontiers in Neuroscience, 2020, 14, 586108.	2.8	6
6	Amyloid Precursor Protein (APP) Controls the Expression of the Transcriptional Activator Neuronal PAS Domain Protein 4 (NPAS4) and Synaptic GABA Release. ENeuro, 2020, 7, ENEURO.0322-19.2020.	1.9	24
7	Dimeric Transmembrane Orientations of APP/C99 Regulate Î ³ -Secretase Processing Line Impacting Signaling and Oligomerization. IScience, 2020, 23, 101887.	4.1	9
8	Influence of the familial Alzheimer's disease–associated T43I mutation on the transmembrane structure and γ-secretase processing of the C99 peptide. Journal of Biological Chemistry, 2019, 294, 5854-5866.	3.4	5
9	Sex-regulated gene dosage effect of PPARα on synaptic plasticity. Life Science Alliance, 2019, 2, e201800262.	2.8	16
10	Specificity of presenilinâ€1―and presenilinâ€2â€dependent γâ€secretases towards substrate processing. Journa of Cellular and Molecular Medicine, 2018, 22, 823-833.	 3.6	23
11	Contribution of the Endosomal-Lysosomal and Proteasomal Systems in Amyloid-Î ² Precursor Protein Derived Fragments Processing. Frontiers in Cellular Neuroscience, 2018, 12, 435.	3.7	24
12	A Role for GDNF and Soluble APP as Biomarkers of Amyotrophic Lateral Sclerosis Pathophysiology. Frontiers in Neurology, 2018, 9, 384.	2.4	33
13	Tau interactome mappingÂbased identification of Otub1 as Tau deubiquitinase involved in accumulation of pathological Tau forms in vitro and in vivo. Acta Neuropathologica, 2017, 133, 731-749.	7.7	74
14	Cortical cells reveal APP as a new player in the regulation of GABAergic neurotransmission. Scientific Reports, 2017, 7, 370.	3.3	31
15	[P4–037]: IDENTIFICATION OF NOVEL TARGETS FOR INHIBITING PRIONâ€LIKE SEEDING AND PROPAGATION OF TAU PATHOLOGY IN VITRO AND IN VIVO. Alzheimer's and Dementia, 2017, 13, P1270.	0.8	0
16	β-Sheet Structure within the Extracellular Domain of C99 Regulates Amyloidogenic Processing. Scientific Reports, 2017, 7, 17159.	3.3	17
17	Presenilin 2-Dependent Maintenance of Mitochondrial Oxidative Capacity and Morphology. Frontiers in Physiology, 2017, 8, 796.	2.8	40
18	Glycines from the APP GXXXG/GXXXA Transmembrane Motifs Promote Formation of Pathogenic AÎ ² Oligomers in Cells. Frontiers in Aging Neuroscience, 2016, 8, 107.	3.4	28

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19	O5-04-01: MOLECULAR MECHANISMS OF ABETA-INDUCED TAU-PATHOLOGY: ANALYSIS OF CROSS-SEEDING OF ABETA AND TAU AND ITS ROLE IN PRION-LIKE PROPAGATION OF TAU-PATHOLOGY IN VITRO AND IN VIVO. , 2016, 12, P385-P385.		0
20	Activation of phagocytic activity in astrocytes by reduced expression of the inflammasome component ASC and its implication in a mouse model of Alzheimer disease. Journal of Neuroinflammation, 2016, 13, 20.	7.2	73
21	APPâ€dependent glial cell lineâ€derived neurotrophic factor gene expression drives neuromuscular junction formation. FASEB Journal, 2016, 30, 1696-1711.	0.5	27
22	Heterotypic seeding of Tau fibrillization by pre-aggregated Abeta provides potent seeds for prion-like seeding and propagation of Tau-pathology in vivo. Acta Neuropathologica, 2016, 131, 549-569.	7.7	129
23	Analysis by a highly sensitive split luciferase assay of the regions involved in APP dimerization and its impact on processing. FEBS Open Bio, 2015, 5, 763-773.	2.3	25
24	Presenilin Transmembrane Domain 8 Conserved AXXXAXXXG Motifs Are Required for the Activity of the Î ³ -Secretase Complex. Journal of Biological Chemistry, 2015, 290, 7169-7184.	3.4	11
25	Characterization of Pterocarpus erinaceus kino extract and its gamma-secretase inhibitory properties. Journal of Ethnopharmacology, 2015, 163, 192-202.	4.1	17
26	Templated misfolding of Tau by prion-like seeding along neuronal connections impairs neuronal network function and associated behavioral outcomes in Tau transgenic mice. Acta Neuropathologica, 2015, 129, 875-894.	7.7	122
27	Epigenetic Regulations of Immediate Early Genes Expression Involved in Memory Formation by the Amyloid Precursor Protein of Alzheimer Disease. PLoS ONE, 2014, 9, e99467.	2.5	60
28	Tauopathy contributes to synaptic and cognitive deficits in a murine model for Alzheimer's disease. FASEB Journal, 2014, 28, 2620-2631.	0.5	37
29	Conformational Changes Induced by the A21G Flemish Mutation in the Amyloid Precursor Protein Lead to Increased Al ² Production. Structure, 2014, 22, 387-396.	3.3	40
30	Gamma-Secretase Inhibitor Activity of a <i>Pterocarpus erinaceus</i> Extract. Neurodegenerative Diseases, 2014, 14, 39-51.	1.4	12
31	P1-033: AMYLOID-INDUCED TAUOPATHY CONTRIBUTES TO SYNAPTIC AND COGNITIVE DEFICITS IN A TRANSGENIC MODEL FOR ALZHEIMER'S DISEASE. , 2014, 10, P315-P315.		0
32	Amyloid precursor protein controls cholesterol turnover needed for neuronal activity. EMBO Molecular Medicine, 2013, 5, 608-625.	6.9	88
33	Epigenetic Induction of EGR-1 Expression by the Amyloid Precursor Protein during Exposure to Novelty. PLoS ONE, 2013, 8, e74305.	2.5	22
34	Contribution of Kunitz Protease Inhibitor and Transmembrane Domains to Amyloid Precursor Protein Homodimerization. Neurodegenerative Diseases, 2012, 10, 92-95.	1.4	12
35	Structural features of the KPI domain control APP dimerization, trafficking, and processing. FASEB Journal, 2012, 26, 855-867.	0.5	40
36	What is the role of amyloid precursor protein dimerization?. Cell Adhesion and Migration, 2010, 4, 268-272.	2.7	36

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37	In vitro screening on β-amyloid peptide production of plants used in traditional medicine for cognitive disorders. Journal of Ethnopharmacology, 2010, 131, 585-591.	4.1	26
38	Epigenetic control of aquaporin 1 expression by the amyloid precursor protein. FASEB Journal, 2009, 23, 4158-4167.	0.5	48
39	A helix-to-coil transition at the Îμ-cut site in the transmembrane dimer of the amyloid precursor protein is required for proteolysis. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 1421-1426.	7.1	115
40	A Helix-to-Coil Transition in the Transmembrane Dimer of the Amyloid Precursor Protein is Required for Proteolysis by Î ³ -Secretase. Biophysical Journal, 2009, 96, 335a.	0.5	0
41	Amyloidogenic Processing but Not Amyloid Precursor Protein (APP) Intracellular C-terminal Domain Production Requires a Precisely Oriented APP Dimer Assembled by Transmembrane GXXXG Motifs. Journal of Biological Chemistry, 2008, 283, 7733-7744.	3.4	125
42	Amyloidogenic processing but not amyloid precursor protein (APP) intracellular C-terminal domain production requires a precisely oriented APP dimer assembled by transmembrane GXXXG motifs. VOLUME 283 (2008) PAGES 7733-7744. Journal of Biological Chemistry, 2008, 283, 12680.	3.4	0
43	Phosphorylation of APP695 at Thr668 decreases γ-cleavage and extracellular Aβ. Biochemical and Biophysical Research Communications, 2007, 357, 1004-1010.	2.1	28
44	Fe65 does not stabilize AICD during activation of transcription in a luciferase assay. Biochemical and Biophysical Research Communications, 2007, 361, 317-322.	2.1	14
45	Inhibitors of Amyloid Toxicity Based on β-sheet Packing of Aβ40 and Aβ42. Biochemistry, 2006, 45, 5503-5516.	2.5	183
46	Lactacystin decreases amyloid-β peptide production by inhibiting β-secretase activity. Journal of Neuroscience Research, 2006, 84, 1311-1322.	2.9	9
47	Lithium Chloride Increases the Production of Amyloid-Î ² Peptide Independently from Its Inhibition of Glycogen Synthase Kinase 3. Journal of Biological Chemistry, 2005, 280, 33220-33227.	3.4	43
48	Adenylosuccinate Lyase Deficiency: Study of Physiopathologic Mechanism(s). Nucleosides, Nucleotides and Nucleic Acids, 2004, 23, 1227-1229.	1.1	2
49	Intracellular Amyloid-β1–42, but Not Extracellular Soluble Amyloid-β Peptides, Induces Neuronal Apoptosis. Journal of Biological Chemistry, 2002, 277, 15666-15670.	3.4	181
50	Correlation between β-amyloid peptide production and human APP-induced neuronal death. Peptides, 2002, 23, 1199-1204.	2.4	15
51	Failure of the interaction between presenilin 1 and the substrate of Î ³ -secretase to produce AÎ ² in insect cells. Journal of Neurochemistry, 2002, 83, 390-399.	3.9	13
52	Continuous Activation of Pituitary Adenylate Cyclase-Activating Polypeptide Receptors Elicits Antipodal Effects on Cyclic AMP and Inositol Phospholipid Signaling Pathways in CATH.a Cells: Role of Protein Synthesis and Protein Kinases. Journal of Neurochemistry, 2002, 70, 1431-1440.	3.9	12
53	The processing and biological function of the human amyloid precursor protein (APP): lessons from different cellular models. Experimental Gerontology, 2000, 35, 843-850.	2.8	12
54	Experimental gerontology in Belgium: from model organisms to age-related pathologies. Experimental Gerontology, 2000, 35, 901-916.	2.8	7

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55	Depolarization regulates cyclin D1 degradation and neuronal apoptosis: a hypothesis about the role of the ubiquitin/proteasome signalling pathway. European Journal of Neuroscience, 1999, 11, 441-448.	2.6	63
56	A mouse model of familial amyotrophic lateral sclerosis expressing a mutant superoxide dismutase 1 shows evidence of disordered transport in the vasopressin hypothalamo-neurohypophysial axis. European Journal of Neuroscience, 1999, 11, 4179-4187.	2.6	21
57	Pharmacological, molecular and functional characterization of vasoactive intestinal polypeptide/pituitary adenylate cyclase-activating polypeptide receptors in the rat pineal gland. Neuroscience, 1998, 85, 887-896.	2.3	46
58	PACAP Type I Receptor Activation Promotes Cerebellar Neuron Survival Through the cAMP/PKA Signaling Pathway. DNA and Cell Biology, 1997, 16, 323-333.	1.9	109
59	GABAB receptors negatively regulate transcription in cerebellar granular neurons through cyclic AMP responsive element binding protein-dependent mechanisms. Neuroscience, 1996, 70, 417-427.	2.3	27
60	Expression of the c-ets 1 gene in the hypothalamus and pituitary during rat development. Developmental Brain Research, 1996, 97, 107-117.	1.7	10
61	Glucocorticoids, but not Dopamine, Negatively Regulate the Melanotrophic Activity of the Rabbit Pituitary Intermediate Lobe. Journal of Neuroendocrinology, 1994, 6, 385-390.	2.6	6
62	APP-deficient neurons show a subtle differential gene expression pattern: impairment in the expression of the activity-dependent transcription factor, NPAS4 Frontiers in Neuroscience, 0, 11, .	2.8	0
63	Cortical cells reveal APP as a regulator of GABAergic neurotransmission. Frontiers in Neuroscience, 0, 11, .	2.8	0
64	Improvement of synaptic plasticity by pharmacological activation of RXR nuclear receptors is PPARα dependent Frontiers in Neuroscience, 0, 12, .	2.8	0
65	Improvement of synaptic plasticity by pharmacological activation of RXR nuclear receptors is PPAR $\hat{I}\pm$ dependent Frontiers in Neuroscience, 0, 12, .	2.8	0
66	Dimeric Transmembrane Orientations of APP/C99 Regulate \hat{I}^3 -Secretase Processing Line Impacting Signaling and Oligomerization. SSRN Electronic Journal, O, , .	0.4	1
67	Dimeric Transmembrane Orientations of AAPP/C99 Regulate γ-Secretase Processing Line Impacting Signaling and Oligomerization. SSRN Electronic Journal, 0, , .	0.4	0