Pascal Kienlen-Campard

List of Publications by Year
in descending order

67	2,251	257357	233338
papers	citations	24 h-index	45 g-index

90	90	90	3117
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Inhibitors of Amyloid Toxicity Based on Î²-sheet Packing of AÎ²40 and AÎ²42. Biochemistry, 2006, 45, 5503-5516.	1.2	183
2	Intracellular Amyloid-Î²1â€“42, but Not Extracellular Soluble Amyloid-Î² Peptides, Induces Neuronal Apoptosis. Journal of Biological Chemistry, 2002, 277, 15666-15670.	1.6	181
3	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.	3.9	129
4	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.	1.6	125
5	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.	3.9	122
6	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.	3.3	115
7	PACAP Type I Receptor Activation Promotes Cerebellar Neuron Survival Through the cAMP/PKA Signaling Pathway. DNA and Cell Biology, 1997, 16, 323-333.	0.9	109
8	Amyloid precursor protein controls cholesterol turnover needed for neuronal activity. EMBO Molecular Medicine, 2013, 5, 608-625.	3.3	88
9	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.	3.9	74
10	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.	3.1	73
11	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.	1.2	63
12	Epigenetic Regulations of Immediate Early Genes Expression Involved in Memory Formation by the Amyloid Precursor Protein of Alzheimer Disease. PLoS ONE, 2014, 9, e99467.	1.1	60
13	Epigenetic control of aquaporin 1 expression by the amyloid precursor protein. FASEB Journal, 2009, 23, 4158-4167.	0.2	48
14	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.	1.1	46
15	Lithium Chloride Increases the Production of Amyloid-Î² Peptide Independently from Its Inhibition of Glycogen Synthase Kinase 3. Journal of Biological Chemistry, 2005, 280, 33220-33227.	1.6	43
16	Structural features of the KPI domain control APP dimerization, trafficking, and processing. FASEB Journal, 2012, 26, 855-867.	0.2	40
17	Conformational Changes Induced by the A21G Flemish Mutation in the Amyloid Precursor Protein Lead to Increased AÎ² Production. Structure, 2014, 22, 387-396.	1.6	40
18	Presenilin 2-Dependent Maintenance of Mitochondrial Oxidative Capacity and Morphology. Frontiers in Physiology, 2017, 8, 796.	1.3	40

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19	Tauopathy contributes to synaptic and cognitive deficits in a murine model for Alzheimer's disease. FASEB Journal, 2014, 28, 2620-2631.	0.2	37
20	What is the role of amyloid precursor protein dimerization?. Cell Adhesion and Migration, 2010, 4, 268-272.	1.1	36
21	A Role for GDNF and Soluble APP as Biomarkers of Amyotrophic Lateral Sclerosis Pathophysiology. Frontiers in Neurology, 2018, 9, 384.	1.1	33
22	Cortical cells reveal APP as a new player in the regulation of GABAergic neurotransmission. Scientific Reports, 2017, 7, 370.	1.6	31
23	Phosphorylation of APP695 at Thr668 decreases Î³-cleavage and extracellular AÎ². Biochemical and Biophysical Research Communications, 2007, 357, 1004-1010.	1.0	28
24	Glycines from the APP GXXXG/GXXXA Transmembrane Motifs Promote Formation of Pathogenic AÎ² Oligomers in Cells. Frontiers in Aging Neuroscience, 2016, 8, 107.	1.7	28
25	GABAB receptors negatively regulate transcription in cerebellar granular neurons through cyclic AMP responsive element binding protein-dependent mechanisms. Neuroscience, 1996, 70, 417-427.	1.1	27
26	APPâ€dependent glial cell lineâ€derived neurotrophic factor gene expression drives neuromuscular junction formation. FASEB Journal, 2016, 30, 1696-1711.	0.2	27
27	In vitro screening on Î²-amyloid peptide production of plants used in traditional medicine for cognitive disorders. Journal of Ethnopharmacology, 2010, 131, 585-591.	2.0	26
28	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.	1.0	25
29	Contribution of the Endosomal-Lysosomal and Proteasomal Systems in Amyloid-Î² Precursor Protein Derived Fragments Processing. Frontiers in Cellular Neuroscience, 2018, 12, 435.	1.8	24
30	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.	0.9	24
31	Specificity of presenilinâ€1â€and presenilinâ€2â€dependent Î³â€secretases towards substrate processing. Journal of Cellular and Molecular Medicine, 2018, 22, 823-833.	1.6	23
32	Epigenetic Induction of EGR-1 Expression by the Amyloid Precursor Protein during Exposure to Novelty. PLoS ONE, 2013, 8, e74305.	1.1	22
33	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.	1.2	21
34	Characterization of Pterocarpus erinaceus kino extract and its gamma-secretase inhibitory properties. Journal of Ethnopharmacology, 2015, 163, 192-202.	2.0	17
35	Î²-Sheet Structure within the Extracellular Domain of C99 Regulates Amyloidogenic Processing. Scientific Reports, 2017, 7, 17159.	1.6	17
36	Sex-regulated gene dosage effect of PPARÎ± on synaptic plasticity. Life Science Alliance, 2019, 2, e201800262.	1.3	16

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37	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.	1.8	16
38	Correlation between Î²-amyloid peptide production and human APP-induced neuronal death. Peptides, 2002, 23, 1199-1204.	1.2	15
39	Fe65 does not stabilize AICD during activation of transcription in a luciferase assay. Biochemical and Biophysical Research Communications, 2007, 361, 317-322.	1.0	14
40	Failure of the interaction between presenilin 1 and the substrate of Î³-secretase to produce AÎ² in insect cells. Journal of Neurochemistry, 2002, 83, 390-399.	2.1	13
41	The processing and biological function of the human amyloid precursor protein (APP): lessons from different cellular models. Experimental Gerontology, 2000, 35, 843-850.	1.2	12
42	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.	2.1	12
43	Contribution of Kunitz Protease Inhibitor and Transmembrane Domains to Amyloid Precursor Protein Homodimerization. Neurodegenerative Diseases, 2012, 10, 92-95.	0.8	12
44	Gamma-Secretase Inhibitor Activity of a <i>Pterocarpus erinaceus</i> Extract. Neurodegenerative Diseases, 2014, 14, 39-51.	0.8	12
45	Presenilin Transmembrane Domain 8 Conserved AXXXAXXXG Motifs Are Required for the Activity of the Î³-Secretase Complex. Journal of Biological Chemistry, 2015, 290, 7169-7184.	1.6	11
46	Overexpression of wild-type human amyloid precursor protein alters GABAergic transmission. Scientific Reports, 2021, 11, 17600.	1.6	11
47	Expression of the c-ets 1 gene in the hypothalamus and pituitary during rat development. Developmental Brain Research, 1996, 97, 107-117.	2.1	10
48	Lactacystin decreases amyloid-Î² peptide production by inhibiting Î²-secretase activity. Journal of Neuroscience Research, 2006, 84, 1311-1322.	1.3	9
49	An evaluation of the self-assembly enhancing properties of cell-derived hexameric amyloid-Î². Scientific Reports, 2021, 11, 11570.	1.6	9
50	Dimeric Transmembrane Orientations of APP/C99 Regulate Î³-Secretase Processing Line Impacting Signaling and Oligomerization. IScience, 2020, 23, 101887.	1.9	9
51	Mechanism of Cellular Formation and In Vivo Seeding Effects of Hexameric Î²-Amyloid Assemblies. Molecular Neurobiology, 2021, 58, 6647-6669.	1.9	8
52	Experimental gerontology in Belgium: from model organisms to age-related pathologies. Experimental Gerontology, 2000, 35, 901-916.	1.2	7
53	Glucocorticoids, but not Dopamine, Negatively Regulate the Melanotrophic Activity of the Rabbit Pituitary Intermediate Lobe. Journal of Neuroendocrinology, 1994, 6, 385-390.	1.2	6
54	Presenilin-Deficient Neurons and Astrocytes Display Normal Mitochondrial Phenotypes. Frontiers in Neuroscience, 2020, 14, 586108.	1.4	6

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55	Influence of the familial Alzheimer's diseaseâ€“associated T43I mutation on the transmembrane structure and Î³-secretase processing of the C99 peptide. Journal of Biological Chemistry, 2019, 294, 5854-5866.	1.6	5
56	Adenylosuccinate Lyase Deficiency: Study of Physiopathologic Mechanism(s). Nucleosides, Nucleotides and Nucleic Acids, 2004, 23, 1227-1229.	0.4	2
57	Dimeric Transmembrane Orientations of APP/C99 Regulate Î³-Secretase Processing Line Impacting Signaling and Oligomerization. SSRN Electronic Journal, 0, , .	0.4	1
58	A Helix-to-Coil Transition in the Transmembrane Dimer of the Amyloid Precursor Protein is Required for Proteolysis by Î³-Secretase. Biophysical Journal, 2009, 96, 335a.	0.2	0
59	P1-033: AMYLOID-INDUCED TAUOPATHY CONTRIBUTES TO SYNAPTIC AND COGNITIVE DEFICITS IN A TRANSGENIC MODEL FOR ALZHEIMER'S DISEASE. , 2014, 10, P315-P315.		0
60	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
61	[P4â€“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.	0.4	0
62	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.	1.6	0
63	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, .	1.4	0
64	Cortical cells reveal APP as a regulator of GABAergic neurotransmission. Frontiers in Neuroscience, 0, 11, .	1.4	0
65	Improvement of synaptic plasticity by pharmacological activation of RXR nuclear receptors is PPARÎ± dependent.. Frontiers in Neuroscience, 0, 12, .	1.4	0
66	Improvement of synaptic plasticity by pharmacological activation of RXR nuclear receptors is PPARÎ± dependent.. Frontiers in Neuroscience, 0, 12, .	1.4	0
67	Dimeric Transmembrane Orientations of AAPP/C99 Regulate Î³-Secretase Processing Line Impacting Signaling and Oligomerization. SSRN Electronic Journal, 0, , .	0.4	0