## Valérie Vingtdeux

List of Publications by Year in descending order

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50 papers

4,737 citations

172457 29 h-index 50 g-index

60 all docs

60 docs citations

60 times ranked

9089 citing authors

#	Article	IF	CITATIONS
1	AMP-activated Protein Kinase Signaling Activation by Resveratrol Modulates Amyloid- $\hat{l}^2$ Peptide Metabolism. Journal of Biological Chemistry, 2010, 285, 9100-9113.	3.4	560
2	CALHM1 ion channel mediates purinergic neurotransmission of sweet, bitter and umami tastes. Nature, 2013, 495, 223-226.	27.8	405
3	Massive CA1/2 Neuronal Loss with Intraneuronal and N-Terminal Truncated AÎ <sup>2</sup> 42 Accumulation in a Novel Alzheimer Transgenic Model. American Journal of Pathology, 2004, 165, 1289-1300.	3.8	375
4	Resveratrol mitigates lipopolysaccharide―and Aβâ€mediated microglial inflammation by inhibiting the TLR4/NFâ€PB/STAT signaling cascade. Journal of Neurochemistry, 2012, 120, 461-472.	3.9	363
5	A Polymorphism in CALHM1 Influences Ca2+ Homeostasis, Aβ Levels, and Alzheimer's Disease Risk. Cell, 2008, 133, 1149-1161.	28.9	310
6	Calcium signaling in neurodegeneration. Molecular Neurodegeneration, 2009, 4, 20.	10.8	258
7	AMPK is abnormally activated in tangle- and pre-tangle-bearing neurons in Alzheimerâ $\in$ <sup>™</sup> s disease and other tauopathies. Acta Neuropathologica, 2011, 121, 337-349.	7.7	247
8	Novel synthetic smallâ€molecule activators of AMPK as enhancers of autophagy and amyloidâ€Î² peptide degradation. FASEB Journal, 2011, 25, 219-231.	0.5	209
9	Therapeutic potential of resveratrol in Alzheimer's disease. BMC Neuroscience, 2008, 9, S6.	1.9	178
10	Alkalizing Drugs Induce Accumulation of Amyloid Precursor Protein By-products in Luminal Vesicles of Multivesicular Bodies. Journal of Biological Chemistry, 2007, 282, 18197-18205.	3.4	176
11	Inhibition of AMP-Activated Protein Kinase Signaling Alleviates Impairments in Hippocampal Synaptic Plasticity Induced by Amyloid $\hat{l}^2$ . Journal of Neuroscience, 2014, 34, 12230-12238.	3.6	143
12	Calcium homeostasis modulator 1 (CALHM1) is the pore-forming subunit of an ion channel that mediates extracellular Ca <sup>2+</sup> regulation of neuronal excitability. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E1963-71.	7.1	132
13	AMP-activated protein kinase modulates tau phosphorylation and tau pathology in vivo. Scientific Reports, 2016, 6, 26758.	3.3	95
14	Potential Contribution of Exosomes to the Prion-Like Propagation of Lesions in Alzheimer's Disease. Frontiers in Physiology, 2012, 3, 229.	2.8	93
15	Down-Regulation of the Met Receptor Tyrosine Kinase by Presenilin-dependent Regulated Intramembrane Proteolysis. Molecular Biology of the Cell, 2009, 20, 2495-2507.	2.1	92
16	Phosphorylation of amyloid precursor carboxy-terminal fragments enhances their processing by a gamma-secretase-dependent mechanism. Neurobiology of Disease, 2005, 20, 625-637.	4.4	82
17	Intracellular pH regulates amyloid precursor protein intracellular domain accumulation. Neurobiology of Disease, 2007, 25, 686-696.	4.4	78
18	Identification and biology of αâ€secretase. Journal of Neurochemistry, 2012, 120, 34-45.	3.9	77

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19	Small-Molecule Activators of AMP-Activated Protein Kinase (AMPK), RSVA314 and RSVA405, Inhibit Adipogenesis. Molecular Medicine, 2011, 17, 1022-1030.	4.4	75
20	AMP-Activated Protein Kinase Is Essential for the Maintenance of Energy Levels during Synaptic Activation. IScience, 2018, 9, 1-13.	4.1	59
21	CB2 Receptor Deficiency Increases Amyloid Pathology and Alters Tau Processing in a Transgenic Mouse Model of Alzheimer's Disease. Molecular Medicine, 2014, 20, 29-36.	4.4	55
22	Neuronal AMP-activated protein kinase hyper-activation induces synaptic loss by an autophagy-mediated process. Cell Death and Disease, 2019, 10, 221.	6.3	54
23	Overexpression of MBNL1 fetal isoforms and modified splicing of Tau in the DM1 brain: Two individual consequences of CUG trinucleotide repeats. Experimental Neurology, 2008, 210, 467-478.	4.1	47
24	AMPK in Neurodegenerative Diseases. Exs, 2016, 107, 153-177.	1.4	38
25	CALHM1 controls the Ca2+-dependent MEK, ERK, RSK and MSK signaling cascade in neurons. Journal of Cell Science, 2013, 126, 1199-1206.	2.0	35
26	Chloroquine and Chloroquinoline Derivatives as Models for the Design of Modulators of Amyloid Peptide Precursor Metabolism. ACS Chemical Neuroscience, 2015, 6, 559-569.	3.5	35
27	CALHM1 ion channel elicits amyloid- $\hat{l}^2$ clearance by insulin-degrading enzyme in cell lines and <i>in vivo</i> in the mouse brain. Journal of Cell Science, 2015, 128, 2330-2338.	2.0	32
28	Epstein-Barr Virus Protein EB2 Contains an N-Terminal Transferable Nuclear Export Signal That Promotes Nucleocytoplasmic Export by Directly Binding TAP/NXF1. Journal of Virology, 2009, 83, 12759-12768.	3.4	31
29	AMPK in Neurodegenerative Diseases: Implications and Therapeutic Perspectives. Current Drug Targets, 2016, 17, 890-907.	2.1	31
30	Protein Kinase CK2 Phosphorylation of EB2 Regulates Its Function in the Production of Epstein-Barr Virus Infectious Viral Particles. Journal of Virology, 2007, 81, 11850-11860.	3.4	30
31	CALHM1 deficiency impairs cerebral neuron activity and memory flexibility in mice. Scientific Reports, 2016, 6, 24250.	3.3	30
32	Identification of potent smallâ€molecule inhibitors of <scp>STAT</scp> 3 with antiâ€inflammatory properties in <scp>RAW</scp> Â264.7 macrophages. FEBS Journal, 2012, 279, 3791-3799.	4.7	29
33	AMP-activated Protein Kinase Controls Immediate Early Genes Expression Following Synaptic Activation Through the PKA/CREB Pathway. International Journal of Molecular Sciences, 2018, 19, 3716.	4.1	29
34	CALHM1 P86L Polymorphism Modulates CSF Aβ Levels in Cognitively Healthy Individuals at Risk for Alzheimer's Disease. Molecular Medicine, 2011, 17, 974-979.	4.4	26
35	Response: CALHM1 Association with Alzheimer's Disease Risk. Cell, 2008, 135, 994-996.	28.9	25
36	Contribution of the Endosomal-Lysosomal and Proteasomal Systems in Amyloid-Î <sup>2</sup> Precursor Protein Derived Fragments Processing. Frontiers in Cellular Neuroscience, 2018, 12, 435.	3.7	24

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37	CB2 Receptor Deficiency Increases Amyloid Pathology and Alters Tau Processing in a Transgenic Mouse Model of Alzheimer's Disease. Molecular Medicine, 2013, 19, 29-36.	4.4	22
38	New piperazine multi-effect drugs prevent neurofibrillary degeneration and amyloid deposition, and preserve memory in animal models of Alzheimer's disease. Neurobiology of Disease, 2019, 129, 217-233.	4.4	21
39	The neuroprotective activity of heat-treated human platelet lysate biomaterials manufactured from outdated pathogen-reduced (amotosalen/UVA) platelet concentrates. Journal of Biomedical Science, 2019, 26, 89.	7.0	20
40	Recovery of brain biomarkers following peroxisome proliferator-activated receptor agonist neuroprotective treatment before ischemic stroke. Proteome Science, 2014, 12, 24.	1.7	17
41	Tau pathology modulates Pin1 post-translational modifications and may be relevant as biomarker. Neurobiology of Aging, 2013, 34, 757-769.	3.1	16
42	A phenotypic approach to the discovery of compounds that promote non-amyloidogenic processing of the amyloid precursor protein: Toward a new profile of indirect $\hat{l}^2$ -secretase inhibitors. European Journal of Medicinal Chemistry, 2018, 159, 104-125.	5.5	16
43	Growth arrest-specific 1 binds to and controls the maturation and processing of the amyloid- $\hat{l}^2$ precursor protein. Human Molecular Genetics, 2011, 20, 2026-2036.	2.9	15
44	Postnatal neurodevelopmental expression and glutamate-dependent regulation of the ZNF804A rodent homologue. Schizophrenia Research, 2015, 168, 402-410.	2.0	12
45	Effect of the CALHM1 G330D and R154H Human Variants on the Control of Cytosolic Ca2+ and A $\hat{l}^2$ Levels. PLoS ONE, 2014, 9, e112484.	2.5	11
46	A Modification-Specific Peptide-Based immunization Approach Using CRM197 Carrier Protein: Development of a Selective Vaccine Against Pyroglutamate AÎ <sup>2</sup> Peptides. Molecular Medicine, 2016, 22, 841-849.	4.4	7
47	Study of AMPK-Regulated Metabolic Fluxes in Neurons Using the Seahorse XFe Analyzer. Methods in Molecular Biology, 2018, 1732, 289-305.	0.9	7
48	Gas1 Interferes with AÎ <sup>2</sup> PP Trafficking by Facilitating the Accumulation of Immature AÎ <sup>2</sup> PP in Endoplasmic Reticulum-Associated Raft Subdomains. Journal of Alzheimer's Disease, 2012, 28, 127-135.	2.6	2
49	Letter to the Editor on "Involvement of AMP-activated-protein-kinase (AMPK) in neuronal amyloidogenesis― Biochemical and Biophysical Research Communications, 2010, 400, 452.	2.1	1
50	Contribution of Multivesicular Bodies to the Prion-Like Propagation of Lesions in Alzheimer's Disease. , 2011, , .		0