

Sylvain E LesnÃ©

List of Publications by Year in descending order

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41
papers

6,974
citations

136950

32
h-index

315739

38
g-index

46
all docs

46
docs citations

46
times ranked

8538
citing authors

#	ARTICLE	IF	CITATIONS
1	A specific amyloid- β protein assembly in the brain impairs memory. <i>Nature</i> , 2006, 440, 352-357.	27.8	2,662
2	Amyloid Oligomers: A Joint Experimental/Computational Perspective on Alzheimer's Disease, Parkinson's Disease, Type II Diabetes, and Amyotrophic Lateral Sclerosis. <i>Chemical Reviews</i> , 2021, 121, 2545-2647.	47.7	406
3	Brain amyloid- β oligomers in ageing and Alzheimer's disease. <i>Brain</i> , 2013, 136, 1383-1398.	7.6	384
4	Accelerating Amyloid- β Fibrillization Reduces Oligomer Levels and Functional Deficits in Alzheimer Disease Mouse Models*. <i>Journal of Biological Chemistry</i> , 2007, 282, 23818-23828.	3.4	375
5	Soluble A β oligomer production and toxicity. <i>Journal of Neurochemistry</i> , 2012, 120, 125-139.	3.9	312
6	The Complex PrP ^{Sc} -Fyn Couples Human Oligomeric A β with Pathological Tau Changes in Alzheimer's Disease. <i>Journal of Neuroscience</i> , 2012, 32, 16857-16871.	3.6	254
7	Cyclooxygenase-2 inhibition improves amyloid- β -mediated suppression of memory and synaptic plasticity. <i>Brain</i> , 2008, 131, 651-664.	7.6	208
8	NMDA Receptor Activation Inhibits β -Secretase and Promotes Neuronal Amyloid- β Production. <i>Journal of Neuroscience</i> , 2005, 25, 9367-9377.	3.6	178
9	Ischemia-Induced Interleukin-6 as a Potential Endogenous Neuroprotective Cytokine against NMDA Receptor-Mediated Excitotoxicity in the Brain. <i>Journal of Cerebral Blood Flow and Metabolism</i> , 2000, 20, 956-966.	4.3	176
10	Orally available compound prevents deficits in memory caused by the Alzheimer amyloid- β oligomers. <i>Annals of Neurology</i> , 2006, 60, 668-676.	5.3	167
11	Plaque-bearing mice with reduced levels of oligomeric amyloid- β assemblies have intact memory function. <i>Neuroscience</i> , 2008, 151, 745-749.	2.3	163
12	Transforming Growth Factor- β 1 Potentiates Amyloid- β Generation in Astrocytes and in Transgenic Mice. <i>Journal of Biological Chemistry</i> , 2003, 278, 18408-18418.	3.4	127
13	Cognitive effects of cell-derived and synthetically derived A β oligomers. <i>Neurobiology of Aging</i> , 2011, 32, 1784-1794.	3.1	124
14	Involvement of -site APP cleaving enzyme 1 (BACE1) in amyloid precursor protein-mediated enhancement of memory and activity-dependent synaptic plasticity. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007, 104, 8167-8172.	7.1	107
15	β -Amyloid Modulation of Synaptic Transmission and Plasticity. <i>Journal of Neuroscience</i> , 2007, 27, 11832-11837.	3.6	107
16	Soluble A-Synuclein Is a Novel Modulator of Alzheimer's Disease Pathophysiology. <i>Journal of Neuroscience</i> , 2012, 32, 10253-10266.	3.6	107
17	Transforming growth factor-beta and ischemic brain injury. <i>Cellular and Molecular Neurobiology</i> , 2003, 23, 539-550.	3.3	90
18	The amyloid- β oligomer A β *56 induces specific alterations in neuronal signaling that lead to tau phosphorylation and aggregation. <i>Science Signaling</i> , 2017, 10, .	3.6	90

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19	Gain-of-function mutations in protein kinase C δ (PKC δ) may promote synaptic defects in Alzheimer's disease. <i>Science Signaling</i> , 2016, 9, ra47.	3.6	84
20	Smad3-Dependent Induction of Plasminogen Activator Inhibitor-1 in Astrocytes Mediates Neuroprotective Activity of Transforming Growth Factor- β 1 against NMDA-Induced Necrosis. <i>Molecular and Cellular Neurosciences</i> , 2002, 21, 634-644.	2.2	77
21	Oligomers of the amyloid- β 2 protein disrupt working memory: Confirmation with two behavioral procedures. <i>Behavioural Brain Research</i> , 2008, 193, 230-234.	2.2	75
22	Genetic Modulation of Soluble A β Rescues Cognitive and Synaptic Impairment in a Mouse Model of Alzheimer's Disease. <i>Journal of Neuroscience</i> , 2014, 34, 7871-7885.	3.6	74
23	Sp1 and Smad transcription factors co-operate to mediate TGF- β 2-dependent activation of amyloid- β 2 precursor protein gene transcription. <i>Biochemical Journal</i> , 2004, 383, 393-399.	3.7	73
24	Tau is required for progressive synaptic and memory deficits in a transgenic mouse model of β -synucleinopathy. <i>Acta Neuropathologica</i> , 2019, 138, 551-574.	7.7	58
25	Akt-dependent Expression of NAIP-1 Protects Neurons against Amyloid- β 2 Toxicity. <i>Journal of Biological Chemistry</i> , 2005, 280, 24941-24947.	3.4	51
26	Transforming growth factor β -induced expression of type α 1 plasminogen activator inhibitor in astrocytes rescues neurons from excitotoxicity. <i>FASEB Journal</i> , 2003, 17, 277-279.	0.5	48
27	Reduced brain tissue perfusion in TGF- β 1 transgenic mice showing Alzheimer's disease-like cerebrovascular abnormalities. <i>Neurobiology of Disease</i> , 2005, 19, 38-46.	4.4	47
28	Soluble Conformers of A β 2 and Tau Alter Selective Proteins Governing Axonal Transport. <i>Journal of Neuroscience</i> , 2016, 36, 9647-9658.	3.6	47
29	Selective lowering of synapsins induced by oligomeric β -synuclein exacerbates memory deficits. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, E4648-E4657.	7.1	45
30	Soluble endogenous oligomeric β -synuclein species in neurodegenerative diseases: Expression, spreading, and cross-talk. <i>Journal of Parkinson's Disease</i> , 2020, 10, 1-28.	2.8	40
31	Increased Expression of Transforming Growth Factor- β 2 after Cerebral Ischemia in the Baboon: An Endogenous Marker of Neuronal Stress?. <i>Journal of Cerebral Blood Flow and Metabolism</i> , 2001, 21, 820-827.	4.3	37
32	Detecting A β *56 Oligomers in Brain Tissues. <i>Methods in Molecular Biology</i> , 2010, 670, 45-56.	0.9	35
33	Bidirectional modulation of Alzheimer phenotype by alpha-synuclein in mice and primary neurons. <i>Acta Neuropathologica</i> , 2018, 136, 589-605.	7.7	29
34	Toxic oligomer species of amyloid- β in Alzheimer's disease, a timing issue. <i>Swiss Medical Weekly</i> , 2014, 144, w14021.	1.6	27
35	Breaking the Code of Amyloid- β Oligomers. <i>International Journal of Cell Biology</i> , 2013, 2013, 1-6.	2.5	25
36	Transforming Growth Factor- β 2 Modulated Cerebral Gene Expression. <i>Journal of Cerebral Blood Flow and Metabolism</i> , 2002, 22, 1114-1123.	4.3	24

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37	Amyloid Plaques and Amyloid- \hat{A} Oligomers: An Ongoing Debate. <i>Journal of Neuroscience</i> , 2005, 25, 9319-9320.	3.6	23
38	Discrete Pools of Oligomeric Amyloid- \hat{I}^2 Track with Spatial Learning Deficits in a Mouse Model of Alzheimer Amyloidosis. <i>American Journal of Pathology</i> , 2018, 188, 739-756.	3.8	16
39	P2-055: Cellular prion protein and class-specific $\hat{A}I^2$ oligomers mediate plaque-associated cytopathology in a mouse model of Alzheimer's disease. , 2015, 11, P504-P504.		1
40	Genetic modulation of soluble $\hat{A}I^2$ rescues cognitive and synaptic impairment in a mouse model of AD. <i>Molecular Neurodegeneration</i> , 2013, 8, .	10.8	0
41	Specific alterations of tau phosphorylation and neuronal signaling induced by the amyloid- \hat{I}^2 oligomer $\hat{A}I^2*56$. <i>Neurobiology of Aging</i> , 2016, 39, S27.	3.1	0