

Sylvain E LesnÃ©

List of Publications by Year in descending order

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41
papers

6,974
citations

156536

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355658

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46
docs citations

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times ranked

9489
citing authors

#	ARTICLE	IF	CITATIONS
1	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.	23.0	406
2	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.	1.5	40
3	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.	3.9	58
4	Discrete Pools of Oligomeric Amyloid- β Track with Spatial Learning Deficits in a Mouse Model of Alzheimer Amyloidosis. <i>American Journal of Pathology</i> , 2018, 188, 739-756.	1.9	16
5	Bidirectional modulation of Alzheimer phenotype by alpha-synuclein in mice and primary neurons. <i>Acta Neuropathologica</i> , 2018, 136, 589-605.	3.9	29
6	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, .	1.6	90
7	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.	3.3	45
8	Specific alterations of tau phosphorylation and neuronal signaling induced by the amyloid- β oligomer A β *56. <i>Neurobiology of Aging</i> , 2016, 39, S27.	1.5	0
9	Soluble Conformers of A β and Tau Alter Selective Proteins Governing Axonal Transport. <i>Journal of Neuroscience</i> , 2016, 36, 9647-9658.	1.7	47
10	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.	1.6	84
11	P2-055: Cellular prion protein and class-specific A β oligomers mediate plaque-associated cytopathology in a mouse model of Alzheimer's disease. , 2015, 11, P504-P504.		1
12	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.	1.7	74
13	Toxic oligomer species of amyloid- β in Alzheimer's disease, a timing issue. <i>Swiss Medical Weekly</i> , 2014, 144, w14021.	0.8	27
14	Brain amyloid- β oligomers in ageing and Alzheimer's disease. <i>Brain</i> , 2013, 136, 1383-1398.	3.7	384
15	Genetic modulation of soluble A β rescues cognitive and synaptic impairment in a mouse model of AD. <i>Molecular Neurodegeneration</i> , 2013, 8, .	4.4	0
16	Breaking the Code of Amyloid- β Oligomers. <i>International Journal of Cell Biology</i> , 2013, 2013, 1-6.	1.0	25
17	Soluble β -Synuclein Is a Novel Modulator of Alzheimer's Disease Pathophysiology. <i>Journal of Neuroscience</i> , 2012, 32, 10253-10266.	1.7	107
18	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.	1.7	254

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19	Soluble A β oligomer production and toxicity. <i>Journal of Neurochemistry</i> , 2012, 120, 125-139.	2.1	312
20	Cognitive effects of cell-derived and synthetically derived A β oligomers. <i>Neurobiology of Aging</i> , 2011, 32, 1784-1794.	1.5	124
21	Detecting A β *56 Oligomers in Brain Tissues. <i>Methods in Molecular Biology</i> , 2010, 670, 45-56.	0.4	35
22	Plaque-bearing mice with reduced levels of oligomeric amyloid- β assemblies have intact memory function. <i>Neuroscience</i> , 2008, 151, 745-749.	1.1	163
23	Oligomers of the amyloid- β protein disrupt working memory: Confirmation with two behavioral procedures. <i>Behavioural Brain Research</i> , 2008, 193, 230-234.	1.2	75
24	Cyclooxygenase-2 inhibition improves amyloid- β -mediated suppression of memory and synaptic plasticity. <i>Brain</i> , 2008, 131, 651-664.	3.7	208
25	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.	1.6	375
26	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.	3.3	107
27	β -Amyloid Modulation of Synaptic Transmission and Plasticity. <i>Journal of Neuroscience</i> , 2007, 27, 11832-11837.	1.7	107
28	A specific amyloid- β protein assembly in the brain impairs memory. <i>Nature</i> , 2006, 440, 352-357.	13.7	2,662
29	Orally available compound prevents deficits in memory caused by the Alzheimer amyloid- β oligomers. <i>Annals of Neurology</i> , 2006, 60, 668-676.	2.8	167
30	NMDA Receptor Activation Inhibits β -Secretase and Promotes Neuronal Amyloid- β Production. <i>Journal of Neuroscience</i> , 2005, 25, 9367-9377.	1.7	178
31	Akt-dependent Expression of NAIP-1 Protects Neurons against Amyloid- β Toxicity. <i>Journal of Biological Chemistry</i> , 2005, 280, 24941-24947.	1.6	51
32	Amyloid Plaques and Amyloid- β Oligomers: An Ongoing Debate. <i>Journal of Neuroscience</i> , 2005, 25, 9319-9320.	1.7	23
33	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.	2.1	47
34	Sp1 and Smad transcription factors co-operate to mediate TGF- β 2-dependent activation of amyloid- β precursor protein gene transcription. <i>Biochemical Journal</i> , 2004, 383, 393-399.	1.7	73
35	Transforming growth factor-beta and ischemic brain injury. <i>Cellular and Molecular Neurobiology</i> , 2003, 23, 539-550.	1.7	90
36	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.	0.2	48

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37	Transforming Growth Factor- β 1 Potentiates Amyloid- β 2 Generation in Astrocytes and in Transgenic Mice. Journal of Biological Chemistry, 2003, 278, 18408-18418.	1.6	127
38	Smad3-Dependent Induction of Plasminogen Activator Inhibitor-1 in Astrocytes Mediates Neuroprotective Activity of Transforming Growth Factor- β 1 against NMDA-Induced Necrosis. Molecular and Cellular Neurosciences, 2002, 21, 634-644.	1.0	77
39	Transforming Growth Factor- β 1 Modulated Cerebral Gene Expression. Journal of Cerebral Blood Flow and Metabolism, 2002, 22, 1114-1123.	2.4	24
40	Increased Expression of Transforming Growth Factor- β 2 after Cerebral Ischemia in the Baboon: An Endogenous Marker of Neuronal Stress?. Journal of Cerebral Blood Flow and Metabolism, 2001, 21, 820-827.	2.4	37
41	Ischemia-Induced Interleukin-6 as a Potential Endogenous Neuroprotective Cytokine against NMDA Receptor-Mediated Excitotoxicity in the Brain. Journal of Cerebral Blood Flow and Metabolism, 2000, 20, 956-966.	2.4	176