Sylvain E LesnÃ©

List of Publications by Year
in descending order

41	6,974	136950 32	315739 38
papers	citations	h-index	g-index

46	46	46	8538
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Amyloid Oligomers: A Joint Experimental/Computational Perspective on Alzheimerâ€™s Disease, Parkinsonâ€™s Disease, Type II Diabetes, and Amyotrophic Lateral Sclerosis. Chemical Reviews, 2021, 121, 2545-2647.	47.7	406
2	Soluble endogenous oligomeric Î±-synuclein species in neurodegenerative diseases: Expression, spreading, and cross-talk. Journal of Parkinson's Disease, 2020, 10, 1-28.	2.8	40
3	Tau is required for progressive synaptic and memory deficits in a transgenic mouse model of Î±-synucleinopathy. Acta Neuropathologica, 2019, 138, 551-574.	7.7	58
4	Discrete Pools of Oligomeric Amyloid-Î² Track with Spatial Learning Deficits in a Mouse Model of Alzheimer Amyloidosis. American Journal of Pathology, 2018, 188, 739-756.	3.8	16
5	Bidirectional modulation of Alzheimer phenotype by alpha-synuclein in mice and primary neurons. Acta Neuropathologica, 2018, 136, 589-605.	7.7	29
6	The amyloid-Î² oligomer AÎ²*56 induces specific alterations in neuronal signaling that lead to tau phosphorylation and aggregation. Science Signaling, 2017, 10, .	3.6	90
7	Selective lowering of synapsins induced by oligomeric Î±-synuclein exacerbates memory deficits. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E4648-E4657.	7.1	45
8	Specific alterations of tau phosphorylation and neuronal signaling induced by the amyloid-Î² oligomer AÎ²*56. Neurobiology of Aging, 2016, 39, S27.	3.1	0
9	Soluble Conformers of AÎ² and Tau Alter Selective Proteins Governing Axonal Transport. Journal of Neuroscience, 2016, 36, 9647-9658.	3.6	47
10	Gain-of-function mutations in protein kinase CÎ± (PKCÎ±) may promote synaptic defects in Alzheimerâ€™s disease. Science Signaling, 2016, 9, ra47.	3.6	84
11	P2-055: Cellular prion protein and class-specific AÎ² oligomers mediate plaque-associated cytopathology in a mouse model of Alzheimer's disease. , 2015, 11, P504-P504.		1
12	Genetic Modulation of Soluble AÂ Rescues Cognitive and Synaptic Impairment in a Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2014, 34, 7871-7885.	3.6	74
13	Toxic oligomer species of amyloid-Î² in Alzheimer's disease, a timing issue. Swiss Medical Weekly, 2014, 144, w14021.	1.6	27
14	Brain amyloid-Î² oligomers in ageing and Alzheimerâ€™s disease. Brain, 2013, 136, 1383-1398.	7.6	384
15	Genetic modulation of soluble AÎ² rescues cognitive and synaptic impairment in a mouse model of AD. Molecular Neurodegeneration, 2013, 8, .	10.8	0
16	Breaking the Code of Amyloid-<mml:math xmlns:mml="http://www.w3.org/1998/Math/MathML" id="M1"><mml:mrow><mml:mi mathvariant="bold-italic">Î²</mml:mi></mml:mrow></mml:math>Oligomers. International Journal of Cell Biology, 2013, 2013, 1-6.	2.5	25
17	Soluble Â-Synuclein Is a Novel Modulator of Alzheimer's Disease Pathophysiology. Journal of Neuroscience, 2012, 32, 10253-10266.	3.6	107
18	The Complex PrP^c-Fyn Couples Human Oligomeric AÎ² with Pathological Tau Changes in Alzheimer's Disease. Journal of Neuroscience, 2012, 32, 16857-16871.	3.6	254

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19	Soluble AÎ² oligomer production and toxicity. Journal of Neurochemistry, 2012, 120, 125-139.	3.9	312
20	Cognitive effects of cell-derived and synthetically derived AÎ² oligomers. Neurobiology of Aging, 2011, 32, 1784-1794.	3.1	124
21	Detecting AÎ²*56 Oligomers in Brain Tissues. Methods in Molecular Biology, 2010, 670, 45-56.	0.9	35
22	Plaque-bearing mice with reduced levels of oligomeric amyloid-Î² assemblies have intact memory function. Neuroscience, 2008, 151, 745-749.	2.3	163
23	Oligomers of the amyloid-Î² protein disrupt working memory: Confirmation with two behavioral procedures. Behavioural Brain Research, 2008, 193, 230-234.	2.2	75
24	Cyclooxygenase-2 inhibition improves amyloid-Î²-mediated suppression of memory and synaptic plasticity. Brain, 2008, 131, 651-664.	7.6	208
25	Accelerating Amyloid-Î² Fibrillization Reduces Oligomer Levels and Functional Deficits in Alzheimer Disease Mouse Models*. Journal of Biological Chemistry, 2007, 282, 23818-23828.	3.4	375
26	Involvement of -site APP cleaving enzyme 1 (BACE1) in amyloid precursor protein-mediated enhancement of memory and activity-dependent synaptic plasticity. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 8167-8172.	7.1	107
27	Î²-Amyloid Modulation of Synaptic Transmission and Plasticity. Journal of Neuroscience, 2007, 27, 11832-11837.	3.6	107
28	A specific amyloid-Î² protein assembly in the brain impairs memory. Nature, 2006, 440, 352-357.	27.8	2,662
29	Orally available compound prevents deficits in memory caused by the Alzheimer amyloid-Î² oligomers. Annals of Neurology, 2006, 60, 668-676.	5.3	167
30	NMDA Receptor Activation Inhibits Î±-Secretase and Promotes Neuronal Amyloid-Î² Production. Journal of Neuroscience, 2005, 25, 9367-9377.	3.6	178
31	Akt-dependent Expression of NAIP-1 Protects Neurons against Amyloid-Î² Toxicity. Journal of Biological Chemistry, 2005, 280, 24941-24947.	3.4	51
32	Amyloid Plaques and Amyloid-Â Oligomers: An Ongoing Debate. Journal of Neuroscience, 2005, 25, 9319-9320.	3.6	23
33	Reduced brain tissue perfusion in TGF-Î²1 transgenic mice showing Alzheimer's disease-like cerebrovascular abnormalities. Neurobiology of Disease, 2005, 19, 38-46.	4.4	47
34	Sp1 and Smad transcription factors co-operate to mediate TGF-Î²-dependent activation of amyloid-Î² precursor protein gene transcription. Biochemical Journal, 2004, 383, 393-399.	3.7	73
35	Transforming growth factor-beta and ischemic brain injury. Cellular and Molecular Neurobiology, 2003, 23, 539-550.	3.3	90
36	Transforming growth factor Î±â€induced expression of typeâ€1 plasminogen activator inhibitor in astrocytes rescues neurons from excitotoxicity. FASEB Journal, 2003, 17, 277-279.	0.5	48

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37	Transforming Growth Factor-Î²1 Potentiates Amyloid-Î² Generation in Astrocytes and in Transgenic Mice. Journal of Biological Chemistry, 2003, 278, 18408-18418.	3.4	127
38	Smad3-Dependent Induction of Plasminogen Activator Inhibitor-1 in Astrocytes Mediates Neuroprotective Activity of Transforming Growth Factor-Î²1 against NMDA-Induced Necrosis. Molecular and Cellular Neurosciences, 2002, 21, 634-644.	2.2	77
39	Transforming Growth Factor-Î²1â€"Modulated Cerebral Gene Expression. Journal of Cerebral Blood Flow and Metabolism, 2002, 22, 1114-1123.	4.3	24
40	Increased Expression of Transforming Growth Factor-Î² after Cerebral Ischemia in the Baboon: An Endogenous Marker of Neuronal Stress?. Journal of Cerebral Blood Flow and Metabolism, 2001, 21, 820-827.	4.3	37
41	Ischemia-Induced Interleukin-6 as a Potential Endogenous Neuroprotective Cytokine against NMDA Receptor-Mediated Excitoxicity in the Brain. Journal of Cerebral Blood Flow and Metabolism, 2000, 20, 956-966.	4.3	176