Lars N G Nilsson

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	The <i>Uppsala APP</i> deletion causes early onset autosomal dominant Alzheimer's disease by altering APP processing and increasing amyloid β fibril formation. Science Translational Medicine, 2021, 13, .	12.4	23
2	CSF sTREM2 and Tau Work Together in Predicting Increased Temporal Lobe Atrophy in Older Adults. Cerebral Cortex, 2020, 30, 2295-2306.	2.9	15
3	Analyzing microglial-associated Aβ in Alzheimer's disease transgenic mice with a novel mid-domain Aβ-antibody. Scientific Reports, 2020, 10, 10590.	3.3	3
4	Cerebrospinal fluid sTREM2 in Alzheimer's disease: comparisons between clinical presentation and AT classification. Scientific Reports, 2020, 10, 15886.	3.3	23
5	A high cerebrospinal fluid soluble TREM2 level is associated with slow clinical progression of Alzheimer's disease. Alzheimer's and Dementia: Diagnosis, Assessment and Disease Monitoring, 2020, 12, e12128.	2.4	16
6	Systemic LPS-induced Aβ-solubilization and clearance in AβPP-transgenic mice is diminished by heparanase overexpression. Scientific Reports, 2019, 9, 4600.	3.3	10
7	Glial activation and inflammation along the Alzheimer's disease continuum. Journal of Neuroinflammation, 2019, 16, 46.	7.2	175
8	An improved CPRG colorimetric ligand-receptor signal transduction assay based on beta-galactosidase activity in mammalian BWZ-reporter cells. Journal of Pharmacological and Toxicological Methods, 2018, 90, 67-75.	0.7	1
9	CSF sTREM2 in delirium—relation to Alzheimer's disease CSF biomarkers Aβ42, t-tau and p-tau. Journal of Neuroinflammation, 2018, 15, 304.	7.2	36
10	The Alzheimer's disease risk factors apolipoprotein E and TREM2 are linked in a receptor signaling pathway. Journal of Neuroinflammation, 2017, 14, 59.	7.2	59
11	Cerebrospinal fluid soluble TREM2 in aging and Alzheimer's disease. Alzheimer's Research and Therapy, 2016, 8, 17.	6.2	105
12	¹¹ C and ¹⁸ F Radiolabeling of Tetra- and Pentathiophenes as PET-Ligands for Amyloid Protein Aggregates. ACS Medicinal Chemistry Letters, 2016, 7, 368-373.	2.8	10
13	Identification of amyloid beta mid-domain fragments in human cerebrospinal fluid. Biochimie, 2015, 113, 86-92.	2.6	8
14	Overexpression of Heparanase Lowers the Amyloid Burden in Amyloid-Î ² Precursor Protein Transgenic Mice. Journal of Biological Chemistry, 2015, 290, 5053-5064.	3.4	41
15	Isobaric Quantification of Cerebrospinal Fluid Amyloid-β Peptides in Alzheimer's Disease: C-Terminal Truncation Relates to Early Measures of Neurodegeneration. Journal of Proteome Research, 2015, 14, 4834-4843.	3.7	7
16	Brainwide distribution and variance of amyloid-beta deposits in tg-ArcSwe mice. Neurobiology of Aging, 2014, 35, 556-564.	3.1	19
17	Elevated mRNA-Levels of Gonadotropin-Releasing Hormone and Its Receptor in Plaque-Bearing Alzheimer's Disease Transgenic Mice. PLoS ONE, 2014, 9, e103607.	2.5	19
18	Lack of exon 10 in the murine tau gene results in mild sensorimotor defects with aging. BMC Neuroscience, 2013, 14, 148.	1.9	11

LARS N G NILSSON

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19	Transient OGG1, APE1, PARP1 and Polβ expression in an Alzheimer's disease mouse model. Mechanisms of Ageing and Development, 2013, 134, 467-477.	4.6	25
20	Elevated MARK2-Dependent Phosphorylation of Tau in Alzheimer's Disease. Journal of Alzheimer's Disease, 2013, 33, 699-713.	2.6	48
21	The Arctic AβPP mutation leads to Alzheimer's disease pathology with highly variable topographic deposition of differentially truncated Aβ. Acta Neuropathologica Communications, 2013, 1, 60.	5.2	38
22	Specific Uptake of an Amyloid-β Protofibril-Binding Antibody-Tracer in AβPP Transgenic Mouse Brain. Journal of Alzheimer's Disease, 2013, 37, 29-40.	2.6	65
23	USE OF FUSED CIRCULATIONS TO INVESTIGATE THE ROLE OF APOLIPOPROTEIN E AS AMYLOID CATALYST AND PERIPHERAL SINK IN ALZHEIMER'S DISEASE. Technology and Innovation, 2012, 14, 199-208.	0.2	10
24	Heparanase overexpression impairs inflammatory response and macrophage-mediated clearance of amyloid-Î ² in murine brain. Acta Neuropathologica, 2012, 124, 465-478.	7.7	57
25	The Arctic amyloid-β precursor protein (AβPP) mutation results in distinct plaques and accumulation of N- and C-truncated AĨ². Neurobiology of Aging, 2012, 33, 1010.e1-1010.e13.	3.1	31
26	Observations in APP Bitransgenic Mice Suggest that Diffuse and Compact Plaques Form via Independent Processes in Alzheimer's Disease. American Journal of Pathology, 2011, 178, 2286-2298.	3.8	38
27	Increased mRNA Levels of <i>TCF7L2</i> and <i>MYC</i> of the Wnt Pathway in Tg-ArcSwe Mice and Alzheimer's Disease, 2011, 2011, 1-7.	2.0	15
28	Loss of Astrocyte Polarization in the Tg-ArcSwe Mouse Model of Alzheimer's Disease. Journal of Alzheimer's Disease, 2011, 27, 711-722.	2.6	165
29	The CCAAT/enhancer binding protein (C/EBP) δ is differently regulated by fibrillar and oligomeric forms of the Alzheimer amyloid-β peptide. Journal of Neuroinflammation, 2011, 8, 34.	7.2	28
30	Sensitive detection of Aβ protofibrils by proximity ligation - relevance for Alzheimer's disease. BMC Neuroscience, 2010, 11, 124.	1.9	33
31	Animal models of amyloidâ€Î²â€related pathologies in Alzheimer's disease. FEBS Journal, 2010, 277, 1389-14	09.7	87
32	Translating research on brain aging into public health: a new type of immunotherapy for Alzheimer's disease. Nutrition Reviews, 2010, 68, S128-S134.	5.8	2
33	Appearance of <i>Cxcl10</i> â€expressing cell clusters is common for traumatic brain injury and neurodegenerative disorders. European Journal of Neuroscience, 2010, 31, 852-863.	2.6	36
34	Genetic Deletion and Pharmacological Inhibition of Nogo-66 Receptor Impairs Cognitive Outcome after Traumatic Brain Injury in Mice. Journal of Neurotrauma, 2010, 27, 1297-1309.	3.4	42
35	Impaired behavior of female tg-ArcSwe APP mice in the IntelliCage: A longitudinal study. Behavioural Brain Research, 2010, 215, 83-94.	2.2	41
36	An amyloid-β protofibril-selective antibody prevents amyloid formation in a mouse model of Alzheimer's disease. Neurobiology of Disease, 2009, 36, 425-434.	4.4	89

LARS N G NILSSON

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37	Genetic and pharmacological evidence of intraneuronal AÎ ² accumulation in APP transgenic mice. FEBS Letters, 2009, 583, 3021-3026.	2.8	18
38	Amyloidâ€Ĵ² protofibril levels correlate with spatial learning in Arctic Alzheimer's disease transgenic mice. FEBS Journal, 2009, 276, 995-1006.	4.7	79
39	A highly insoluble state of AÎ ² similar to that of Alzheimer's disease brain is found in Arctic APP transgenic mice. Neurobiology of Aging, 2009, 30, 1393-1405.	3.1	79
40	Heparan Sulfate Accumulation with Aβ Deposits in Alzheimer's Disease and Tg2576 Mice is Contributed by Glial Cells. Brain Pathology, 2008, 18, 548-561.	4.1	71
41	Local impact of perivascular plaques on cerebral blood flow dynamics in a transgenic mouse model of Alzheimer's disease. , 2008, , .		Ο
42	Sensitive ELISA detection of amyloidâ€Ĵ² protofibrils in biological samples. Journal of Neurochemistry, 2007, 103, 334-345.	3.9	174
43	Imaging Distinct Conformational States of Amyloid-β Fibrils in Alzheimer's Disease Using Novel Luminescent Probes. ACS Chemical Biology, 2007, 2, 553-560.	3.4	177
44	Docosahexaenoic acid stimulates nonâ€amyloidogenic APP processing resulting in reduced Aβ levels in cellular models of Alzheimer's disease. European Journal of Neuroscience, 2007, 26, 882-889.	2.6	51
45	The Arctic Alzheimer mutation favors intracellular amyloid-β production by making amyloid precursor protein less available to α-secretase. Journal of Neurochemistry, 2007, 101, 854-862.	3.9	55
46	The Arctic Alzheimer mutation facilitates early intraneuronal Aβ aggregation and senile plaque formation in transgenic mice. Neurobiology of Aging, 2006, 27, 67-77.	3.1	221
47	Amyloidâ€Î² oligomers are inefficiently measured by enzymeâ€linked immunosorbent assay. Annals of Neurology, 2005, 58, 147-150.	5.3	88
48	Apolipoprotein is required for the formation of filamentous amyloid, but not for amorphous Aβ deposition, in an AβPP/PS double transgenic mouse model of Alzheimer's disease. Journal of Alzheimer's Disease, 2004, 6, 509-514.	2.6	13
49	Use of multimetric statistical analysis to characterize and discriminate between the performance of four Alzheimer's transgenic mouse lines differing in Aβ deposition. Behavioural Brain Research, 2004, 153, 107-121.	2.2	37
50	Cognitive impairment in PDAPP mice depends on ApoE and ACT-catalyzed amyloid formation. Neurobiology of Aging, 2004, 25, 1153-1167.	3.1	70
51	Effect of cytokines, dexamethazone and the A/T-signal peptide polymorphism on the expression of alpha1-antichymotrypsin in astrocytes: significance for Alzheimer's disease. Neurochemistry International, 2001, 39, 361-370.	3.8	27
52	The inflammation-induced pathological chaperones ACT and apo-E are necessary catalysts of Alzheimer amyloid formation. Neurobiology of Aging, 2001, 22, 923-930.	3.1	79
53	α-1-Antichymotrypsin Promotes β-Sheet Amyloid Plaque Deposition in a Transgenic Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2001, 21, 1444-1451.	3.6	133
54	Translation of the Alzheimer Amyloid Precursor Protein mRNA Is Up-regulated by Interleukin-1 through 5â€2-Untranslated Region Sequences. Journal of Biological Chemistry, 1999, 274, 6421-6431.	3.4	256

LARS N G NILSSON

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55	Coexistence of somatostatin receptor subtypes in the human neuroblastoma cell line LA-N-2. FEBS Letters, 1997, 401, 83-88.	2.8	6
56	Influence of place learning on somatostatin levels in the rat brain following environmental deprivation. Regulatory Peptides, 1995, 58, 11-18.	1.9	3
57	Diminution of preprosomatostatin-mRNA in cerebral cortex of the aged rat. Neurochemistry International, 1995, 27, 481-487.	3.8	1
58	Somatostatinergic phenotype markers in the human neuroblastoma cell-line LA-N-2. FEBS Letters, 1995, 372, 88-92.	2.8	4
59	Effects of nucleus basalis lesion on muscarinic receptor subtypes. Experimental Brain Research, 1993, 97, 225-32.	1.5	15
60	Decrease of somatostatin receptor binding in the rat cerebral cortex after ibotenic acid lesion of the nucleus basalis magnocellularis: a quantitative autoradiographic study. Brain Research, 1993, 628, 31-38.	2.2	7
61	Environmental influence on somatostatin levels and gene expression in the rat brain. Brain Research, 1993, 628, 93-98.	2.2	24
62	Important Role of Hepatitis C Virus Infection as a Cause of Chronic Liver Disease in Somalia. Scandinavian Journal of Infectious Diseases, 1993, 25, 559-564.	1.5	19
63	Characterization and quantification of 125I-bolton hunter substance P binding sites in human brain. Neurochemistry International, 1991, 18, 399-404.	3.8	9
64	Regional distribution of somatostatin receptor binding and modulation of adenylyl cyclase activity in Alzheimer's disease brain. Journal of the Neurological Sciences, 1991, 105, 225-233.	0.6	56
65	Impaired astrocytic Ca2+ signaling in awake-behaving Alzheimer's disease transgenic mice. ELife, 0, 11, .	6.0	15