

Jos J Lucas

List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

112
papers

8,672
citations

48
h-index

92
g-index

117
ext. papers

9,559
ext. citations

8.5
avg, IF

5.7
L-index

#	Paper	IF	Citations
112	Huntington's disease-specific mis-splicing unveils key effector genes and altered splicing factors. <i>Brain</i> , 2021 , 144, 2009-2023	11.2	4
111	Huntingtin-mediated axonal transport requires arginine methylation by PRMT6. <i>Cell Reports</i> , 2021 , 35, 108980	10.6	3
110	A new non-aggregative splicing isoform of human Tau is decreased in Alzheimer's disease. <i>Acta Neuropathologica</i> , 2021 , 142, 159-177	14.3	3
109	CPEB alteration and aberrant transcriptome-polyadenylation lead to a treatable SLC19A3 deficiency in Huntington's disease. <i>Science Translational Medicine</i> , 2021 , 13, eabe7104	17.5	0
108	Prion-Associated Neurodegeneration Causes Both Endoplasmic Reticulum Stress and Proteasome Impairment in a Murine Model of Spontaneous Disease. <i>International Journal of Molecular Sciences</i> , 2021 , 22,	6.3	4
107	P2X7 Receptor Upregulation in Huntington's Disease Brains. <i>Frontiers in Molecular Neuroscience</i> , 2020 , 13, 567430	6.1	8
106	Pathogenic SREK1 decrease in Huntington's disease lowers TAF1 mimicking X-linked dystonia parkinsonism. <i>Brain</i> , 2020 , 143, 2207-2219	11.2	9
105	Polyadenylation of mRNA as a novel regulatory mechanism of gene expression in temporal lobe epilepsy. <i>Brain</i> , 2020 , 143, 2139-2153	11.2	2
104	High concordance between hippocampal transcriptome of the mouse intra-amygdala kainic acid model and human temporal lobe epilepsy. <i>Epilepsia</i> , 2020 , 61, 2795-2810	6.4	7
103	Differential regulation of Kidins220 isoforms in Huntington's disease. <i>Brain Pathology</i> , 2020 , 30, 120-136		1
102	Impaired development of neocortical circuits contributes to the neurological alterations in DYRK1A haploinsufficiency syndrome. <i>Neurobiology of Disease</i> , 2019 , 127, 210-222	7.5	14
101	Altered Levels and Isoforms of Tau and Nuclear Membrane Invaginations in Huntington's Disease. <i>Frontiers in Cellular Neuroscience</i> , 2019 , 13, 574	6.1	14
100	Autism-like phenotype and risk gene mRNA deadenylation by CPEB4 mis-splicing. <i>Nature</i> , 2018 , 560, 441-446	50.4	62
99	Profiling of Argonaute-2-loaded microRNAs in a mouse model of frontotemporal dementia with parkinsonism-17. <i>International Journal of Physiology, Pathophysiology and Pharmacology</i> , 2018 , 10, 172-183	3.4	2
98	The regulation of proteostasis in glial cells by nucleotide receptors is key in acute neuroinflammation. <i>FASEB Journal</i> , 2018 , 32, 3020-3032	0.9	6
97	Bi-directional genetic modulation of GSK-3 β exacerbates hippocampal neuropathology in experimental status epilepticus. <i>Cell Death and Disease</i> , 2018 , 9, 969	9.8	16
96	MAP2 Splicing is Altered in Huntington's Disease. <i>Brain Pathology</i> , 2017 , 27, 181-189	6	15

95	Tau-positive nuclear indentations in P301S tauopathy mice. <i>Brain Pathology</i> , 2017 , 27, 314-322	6	9
94	Spatiotemporal progression of ubiquitin-proteasome system inhibition after status epilepticus suggests protective adaptation against hippocampal injury. <i>Molecular Neurodegeneration</i> , 2017 , 12, 21	19	11
93	The neuroprotective transcription factor ATF5 is decreased and sequestered into polyglutamine inclusions in Huntington's disease. <i>Acta Neuropathologica</i> , 2017 , 134, 839-850	14.3	7
92	Regulation of proteasome activity by P2Y receptor underlies the neuroprotective effects of extracellular nucleotides. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2017 , 1863, 43-51	6.9	4
91	Faulty splicing and cytoskeleton abnormalities in Huntington's disease. <i>Brain Pathology</i> , 2016 , 26, 772-778		21
90	Prion-mediated neurodegeneration is associated with early impairment of the ubiquitin-proteasome system. <i>Acta Neuropathologica</i> , 2016 , 131, 411-25	14.3	44
89	Impaired PLP-dependent metabolism in brain samples from Huntington disease patients and transgenic R6/1 mice. <i>Metabolic Brain Disease</i> , 2016 , 31, 579-86	3.9	3
88	Altered Machinery of Protein Synthesis in Alzheimer's: From the Nucleolus to the Ribosome. <i>Brain Pathology</i> , 2016 , 26, 593-605	6	96
87	Mice Lacking Functional Fas Death Receptors Are Protected from Kainic Acid-Induced Apoptosis in the Hippocampus. <i>Molecular Neurobiology</i> , 2015 , 52, 120-9	6.2	5
86	Decreased glycogen synthase kinase-3 levels and activity contribute to Huntington's disease. <i>Human Molecular Genetics</i> , 2015 , 24, 5040-52	5.6	27
85	In vivo inhibition of the mitochondrial H ⁺ -ATP synthase in neurons promotes metabolic preconditioning. <i>EMBO Journal</i> , 2014 , 33, 762-78	13	64
84	Reduced striatal dopamine DA D2 receptor function in dominant-negative GSK-3 transgenic mice. <i>European Neuropsychopharmacology</i> , 2014 , 24, 1524-33	1.2	9
83	Huntington's disease is a four-repeat tauopathy with tau nuclear rods. <i>Nature Medicine</i> , 2014 , 20, 881-5	50.5	135
82	Overexpression of synphilin-1 promotes clearance of soluble and misfolded alpha-synuclein without restoring the motor phenotype in aged A30P transgenic mice. <i>Human Molecular Genetics</i> , 2014 , 23, 767-81	5.6	14
81	Ubiquitin-proteasome system involvement in Huntington's disease. <i>Frontiers in Molecular Neuroscience</i> , 2014 , 7, 77	6.1	67
80	Mice with a naturally occurring DISC1 mutation display a broad spectrum of behaviors associated to psychiatric disorders. <i>Frontiers in Behavioral Neuroscience</i> , 2014 , 8, 253	3.5	22
79	Mutant huntingtin affects endocytosis in striatal cells by altering the binding of AP-2 to membranes. <i>Experimental Neurology</i> , 2013 , 241, 75-83	5.7	13
78	GSK3 and tau: two convergence points in Alzheimer's disease. <i>Journal of Alzheimers Disease</i> , 2013 , 33 Suppl 1, S141-4	4.3	162

77	CHOP regulates the p53-MDM2 axis and is required for neuronal survival after seizures. <i>Brain</i> , 2013 , 136, 577-92	11.2	74
76	Protective neuronal induction of ATF5 in endoplasmic reticulum stress induced by status epilepticus. <i>Brain</i> , 2013 , 136, 1161-76	11.2	33
75	Neuronal apoptosis and motor deficits in mice with genetic inhibition of GSK-3 are Fas-dependent. <i>PLoS ONE</i> , 2013 , 8, e70952	3.7	5
74	Age-dependent decline of motor neocortex but not hippocampal performance in heterozygous BDNF mice correlates with a decrease of cortical PSD-95 but an increase of hippocampal TrkB levels. <i>Experimental Neurology</i> , 2012 , 237, 335-45	5.7	21
73	βSynuclein levels affect autophagosome numbers in vivo and modulate Huntington disease pathology. <i>Autophagy</i> , 2012 , 8, 431-2	10.2	17
72	Looking for novel functions of tau. <i>Biochemical Society Transactions</i> , 2012 , 40, 653-5	5.1	15
71	βSynuclein accumulates in huntingtin inclusions but forms independent filaments and its deficiency attenuates early phenotype in a mouse model of Huntington's disease. <i>Human Molecular Genetics</i> , 2012 , 21, 495-510	5.6	25
70	Striatal-enriched protein tyrosine phosphatase expression and activity in Huntington's disease: a STEP in the resistance to excitotoxicity. <i>Journal of Neuroscience</i> , 2011 , 31, 8150-62	6.6	55
69	GSK-3 Mouse Models to Study Neuronal Apoptosis and Neurodegeneration. <i>Frontiers in Molecular Neuroscience</i> , 2011 , 4, 45	6.1	49
68	Impaired ATF6 processing, decreased Rheb and neuronal cell cycle re-entry in Huntington's disease. <i>Neurobiology of Disease</i> , 2011 , 41, 23-32	7.5	35
67	Loss of striatal type 1 cannabinoid receptors is a key pathogenic factor in Huntington's disease. <i>Brain</i> , 2011 , 134, 119-36	11.2	154
66	Increased neurotransmitter release at the neuromuscular junction in a mouse model of polyglutamine disease. <i>Journal of Neuroscience</i> , 2011 , 31, 1106-13	6.6	34
65	GSK3β overexpression induces neuronal death and a depletion of the neurogenic niches in the dentate gyrus. <i>Hippocampus</i> , 2011 , 21, 910-22	3.5	61
64	PH domain leucine-rich repeat protein phosphatase 1 contributes to maintain the activation of the PI3K/Akt pro-survival pathway in Huntington's disease striatum. <i>Cell Death and Differentiation</i> , 2010 , 17, 324-35	12.7	42
63	Centro de Biología Molecular "Severo Ochoa": a center for basic research into Alzheimer's disease. <i>Journal of Alzheimers Disease</i> , 2010 , 21, 325-35	4.3	
62	Tau kinase I overexpression induces dentate gyrus degeneration. <i>Neurodegenerative Diseases</i> , 2010 , 7, 13-5	2.3	4
61	Acute polyglutamine expression in inducible mouse model unravels ubiquitin/proteasome system impairment and permanent recovery attributable to aggregate formation. <i>Journal of Neuroscience</i> , 2010 , 30, 3675-88	6.6	76
60	GSK3: a possible link between beta amyloid peptide and tau protein. <i>Experimental Neurology</i> , 2010 , 223, 322-5	5.7	200

59	Presynaptic dysfunction in Huntington's disease. <i>Biochemical Society Transactions</i> , 2010 , 38, 488-92	5.1	26
58	Tau phosphorylation in hippocampus results in toxic gain-of-function. <i>Biochemical Society Transactions</i> , 2010 , 38, 977-80	5.1	21
57	Tau-knockout mice show reduced GSK3-induced hippocampal degeneration and learning deficits. <i>Neurobiology of Disease</i> , 2010 , 37, 622-9	7.5	87
56	Protein oxidation in Huntington disease affects energy production and vitamin B6 metabolism. <i>Free Radical Biology and Medicine</i> , 2010 , 49, 612-21	7.8	66
55	NFAT/Fas signaling mediates the neuronal apoptosis and motor side effects of GSK-3 inhibition in a mouse model of lithium therapy. <i>Journal of Clinical Investigation</i> , 2010 , 120, 2432-45	15.9	64
54	Accumulation of ubiquitin conjugates in a polyglutamine disease model occurs without global ubiquitin/proteasome system impairment. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2009 , 106, 13986-91	11.5	72
53	Altered P2X7-receptor level and function in mouse models of Huntington's disease and therapeutic efficacy of antagonist administration. <i>FASEB Journal</i> , 2009 , 23, 1893-906	0.9	180
52	Reduced calcineurin protein levels and activity in exon-1 mouse models of Huntington's disease: role in excitotoxicity. <i>Neurobiology of Disease</i> , 2009 , 36, 461-9	7.5	32
51	Effects of partial suppression of parkin on huntingtin mutant R6/1 mice. <i>Brain Research</i> , 2009 , 1281, 91-100	3.7	21
50	Tauopathies with parkinsonism: clinical spectrum, neuropathologic basis, biological markers, and treatment options. <i>European Journal of Neurology</i> , 2009 , 16, 297-309	6	143
49	The role of GSK3 in Alzheimer disease. <i>Brain Research Bulletin</i> , 2009 , 80, 248-50	3.9	59
48	Hippocampal neuronal subpopulations are differentially affected in double transgenic mice overexpressing frontotemporal dementia and parkinsonism linked to chromosome 17 tau and glycogen synthase kinase-3beta. <i>Neuroscience</i> , 2008 , 157, 772-80	3.9	8
47	Lithium, a potential protective drug in Alzheimer's disease. <i>Neurodegenerative Diseases</i> , 2008 , 5, 247-9	2.3	37
46	Co-expression of FTDP-17 Human Tau and GSK-3[̢] (or APPSW) in Transgenic Mice: Induction of Tau Polymerization and Neurodegeneration 2008 , 337-342		
45	A mouse model to study tau pathology related with tau phosphorylation and assembly. <i>Journal of the Neurological Sciences</i> , 2007 , 257, 250-4	3.2	7
44	BH3-only proteins Bid and Bim(EL) are differentially involved in neuronal dysfunction in mouse models of Huntington's disease. <i>Journal of Neuroscience Research</i> , 2007 , 85, 2756-69	4.4	29
43	Neuronal apoptosis and reversible motor deficit in dominant-negative GSK-3 conditional transgenic mice. <i>EMBO Journal</i> , 2007 , 26, 2743-54	13	54
42	Glycogen synthase kinase-3 inhibition is integral to long-term potentiation. <i>European Journal of Neuroscience</i> , 2007 , 25, 81-6	3.5	268

41	Is the ubiquitin-proteasome system impaired in Huntington's disease?. <i>Cellular and Molecular Life Sciences</i> , 2007 , 64, 2245-57	10.3	54
40	N-terminal cleavage of GSK-3 by calpain: a new form of GSK-3 regulation. <i>Journal of Biological Chemistry</i> , 2007 , 282, 22406-13	5.4	99
39	Testing the possible inhibition of proteasome by direct interaction with ubiquitylated and aggregated huntingtin. <i>Brain Research Bulletin</i> , 2007 , 72, 121-3	3.9	6
38	Full reversal of Alzheimer's disease-like phenotype in a mouse model with conditional overexpression of glycogen synthase kinase-3. <i>Journal of Neuroscience</i> , 2006 , 26, 5083-90	6.6	217
37	Cooexpression of FTDP-17 tau and GSK-3beta in transgenic mice induce tau polymerization and neurodegeneration. <i>Neurobiology of Aging</i> , 2006 , 27, 1258-68	5.6	96
36	Reduced expression of the TrkB receptor in Huntington's disease mouse models and in human brain. <i>European Journal of Neuroscience</i> , 2006 , 23, 649-58	3.5	107
35	Inhibition of 26S proteasome activity by huntingtin filaments but not inclusion bodies isolated from mouse and human brain. <i>Journal of Neurochemistry</i> , 2006 , 98, 1585-96	6	77
34	Chronic lithium administration to FTDP-17 tau and GSK-3beta overexpressing mice prevents tau hyperphosphorylation and neurofibrillary tangle formation, but pre-formed neurofibrillary tangles do not revert. <i>Journal of Neurochemistry</i> , 2006 , 99, 1445-55	6	169
33	The Ups in Neurodegenerative Diseases and Aging Huntington's Disease 2006 , 225-235		
32	The ubiquitin-proteasome system in Huntington's disease. <i>Neuroscientist</i> , 2005 , 11, 583-94	7.6	42
31	Full motor recovery despite striatal neuron loss and formation of irreversible amyloid-like inclusions in a conditional mouse model of Huntington's disease. <i>Journal of Neuroscience</i> , 2005 , 25, 9773-81	6.6	61
30	Assembly in vitro of tau protein and its implications in Alzheimer's disease. <i>Current Alzheimer Research</i> , 2004 , 1, 97-101	3	21
29	Biochemical, ultrastructural, and reversibility studies on huntingtin filaments isolated from mouse and human brain. <i>Journal of Neuroscience</i> , 2004 , 24, 9361-71	6.6	47
28	Glycogen synthase kinase-3 plays a crucial role in tau exon 10 splicing and intranuclear distribution of SC35. Implications for Alzheimer's disease. <i>Journal of Biological Chemistry</i> , 2004 , 279, 3801-6	5.4	103
27	Enhanced induction of the immunoproteasome by interferon gamma in neurons expressing mutant Huntingtin. <i>Neurotoxicity Research</i> , 2004 , 6, 463-8	4.3	36
26	Tau in neurodegenerative diseases: tau phosphorylation and assembly. <i>Neurotoxicity Research</i> , 2004 , 6, 477-82	4.3	39
25	Testing the ubiquitin-proteasome hypothesis of neurodegeneration in vivo. <i>Trends in Neurosciences</i> , 2004 , 27, 66-9	13.3	33
24	Role of tau protein in both physiological and pathological conditions. <i>Physiological Reviews</i> , 2004 , 84, 361-84	47.9	641

23	Proteasomal expression, induction of immunoproteasome subunits, and local MHC class I presentation in myofibrillar myopathy and inclusion body myositis. <i>Journal of Neuropathology and Experimental Neurology</i> , 2004 , 63, 484-98	3.1	69
22	Neuronal induction of the immunoproteasome in Huntington's disease. <i>Journal of Neuroscience</i> , 2003 , 23, 11653-61	6.6	218
21	GSK-3 dependent phosphoepitopes recognized by PHF-1 and AT-8 antibodies are present in different tau isoforms. <i>Neurobiology of Aging</i> , 2003 , 24, 1087-94	5.6	36
20	Structural insights and biological effects of glycogen synthase kinase 3-specific inhibitor AR-A014418. <i>Journal of Biological Chemistry</i> , 2003 , 278, 45937-45	5.4	393
19	Loss of mRNA levels, binding and activation of GTP-binding proteins for cannabinoid CB1 receptors in the basal ganglia of a transgenic model of Huntington's disease. <i>Brain Research</i> , 2002 , 929, 236-42	3.7	93
18	Sulfo-glycosaminoglycan content affects PHF-tau solubility and allows the identification of different types of PHFs. <i>Brain Research</i> , 2002 , 935, 65-72	3.7	17
17	Spatial learning deficit in transgenic mice that conditionally over-express GSK-3beta in the brain but do not form tau filaments. <i>Journal of Neurochemistry</i> , 2002 , 83, 1529-33	6	291
16	Nuclear localization of N-terminal mutant huntingtin is cell cycle dependent. <i>European Journal of Neuroscience</i> , 2002 , 16, 355-9	3.5	19
15	Decreased nuclear beta-catenin, tau hyperphosphorylation and neurodegeneration in GSK-3beta conditional transgenic mice. <i>EMBO Journal</i> , 2001 , 20, 27-39	13	670
14	FTDP-17 mutations in tau transgenic mice provoke lysosomal abnormalities and Tau filaments in forebrain. <i>Molecular and Cellular Neurosciences</i> , 2001 , 18, 702-14	4.8	176
13	Proteasomal-dependent aggregate reversal and absence of cell death in a conditional mouse model of Huntington's disease. <i>Journal of Neuroscience</i> , 2001 , 21, 8772-81	6.6	136
12	Modulation of the effects of cocaine by 5-HT1B receptors: a comparison of knockouts and antagonists. <i>Pharmacology Biochemistry and Behavior</i> , 2000 , 67, 559-66	3.9	81
11	Reversal of neuropathology and motor dysfunction in a conditional model of Huntington's disease. <i>Cell</i> , 2000 , 101, 57-66	56.2	899
10	Nuclear localization of beta-catenin in adult mouse thalamus correlates with low levels of GSK-3beta. <i>NeuroReport</i> , 1999 , 10, 2699-703	1.7	12
9	Increased vulnerability to cocaine in mice lacking the serotonin-1B receptor. <i>Nature</i> , 1998 , 393, 175-8	50.4	276
8	Absence of fenfluramine-induced anorexia and reduced c-Fos induction in the hypothalamus and central amygdaloid complex of serotonin 1B receptor knock-out mice. <i>Journal of Neuroscience</i> , 1998 , 18, 5537-44	6.6	136
7	5-Hydroxytryptamine1B receptors modulate the effect of cocaine on c-fos expression: converging evidence using 5-hydroxytryptamine1B knockout mice and the 5-hydroxytryptamine1B/1D antagonist GR127935. <i>Molecular Pharmacology</i> , 1997 , 51, 755-63	4.3	81
6	Peripheral noxious stimulation induces CREM expression in dorsal horn: involvement of glutamate. <i>European Journal of Neuroscience</i> , 1997 , 9, 2778-83	3.5	12

5	New players in the 5-HT receptor field: genes and knockouts. <i>Trends in Pharmacological Sciences</i> , 1995 , 16, 246-52	13.2	102
4	Molecular mechanisms of pain: serotonin1A receptor agonists trigger transactivation by c-fos of the prodynorphin gene in spinal cord neurons. <i>Neuron</i> , 1993 , 10, 599-611	13.9	90
3	Co-induction of jun B and c-fos in a subset of neurons in the spinal cord. <i>Oncogene</i> , 1991 , 6, 223-7	9.2	54
2	Animal Models with Modified Expression of GSK-3 for the Study of Its Physiology and of Its Implications in Human Pathologies203-219		
1	Huntington's disease-specific mis-splicing captured by human-mouse intersect-RNA-seq unveils pathogenic effectors and reduced splicing factors		4