Michael K Lee

List of Publications by Year
in descending order

106 papers	25,062 citations	28736 57 h-index	36203 101 g-index
110 all docs	110 docs citations	110 times ranked	22108 citing authors

#	Article	IF	Citations
1	Astrocyteâ€neuronal network interplay is disrupted in Alzheimer's disease mice. Glia, 2022, 70, 368-378.	2.5	33
2	Loss of tau expression attenuates neurodegeneration associated with Î±-synucleinopathy. Translational Neurodegeneration, 2022, 11, .	3.6	7
3	Changes in Drp1 Function and Mitochondrial Morphology Are Associated with the Î±-Synuclein Pathology in a Transgenic Mouse Model of Parkinsonâ€™s Disease. Cells, 2021, 10, 885.	1.8	27
4	UNC-45A Is Highly Expressed in the Proliferative Cells of the Mouse Genital Tract and in the Microtubule-Rich Areas of the Mouse Nervous System. Cells, 2021, 10, 1604.	1.8	2
5	Partial inhibition of mitochondrial complex I ameliorates Alzheimerâ€™s disease pathology and cognition in APP/PS1 female mice. Communications Biology, 2021, 4, 61.	2.0	35
6	Î³-Glutamyl-Transpeptidase-Resistant Glutathione Analog Attenuates Progression of Alzheimerâ€™s Disease-like Pathology and Neurodegeneration in a Mouse Model. Antioxidants, 2021, 10, 1796.	2.2	8
7	Ablating Tau Reduces Hyperexcitability and Moderates Electroencephalographic Slowing in Transgenic Mice Expressing A53T Human Î±-Synuclein. Frontiers in Neurology, 2020, 11, 563.	1.1	19
8	Î±-Synucleinopathy associated c-Abl activation causes p53-dependent autophagy impairment. Molecular Neurodegeneration, 2020, 15, 27.	4.4	35
9	Pharmacological and chemogenetic orexin/hypocretin intervention ameliorates Hipp-dependent memory impairment in the A53T mice model of Parkinsonâ€™s disease. Molecular Brain, 2019, 12, 87.	1.3	25
10	Tau is required for progressive synaptic and memory deficits in a transgenic mouse model of Î±-synucleinopathy. Acta Neuropathologica, 2019, 138, 551-574.	3.9	58
11	Optic Nerve Sheath Fenestration for Treatment of Retrolaminar Silicone Oil Migration. Ophthalmic Plastic and Reconstructive Surgery, 2019, 35, e31-e34.	0.4	2
12	UNC-45A Is a Novel Microtubule-Associated Protein and Regulator of Paclitaxel Sensitivity in Ovarian Cancer Cells. Molecular Cancer Research, 2019, 17, 370-383.	1.5	21
13	Toxic properties of microsome-associated alpha-synuclein species in mouse primary neurons. Neurobiology of Disease, 2018, 111, 36-47.	2.1	21
14	Î±-synuclein Induces Mitochondrial Dysfunction through Spectrin and the Actin Cytoskeleton. Neuron, 2018, 97, 108-124.e6.	3.8	181
15	A53T Mutant Alpha-Synuclein Induces Tau-Dependent Postsynaptic Impairment Independently of Neurodegenerative Changes. Journal of Neuroscience, 2018, 38, 9754-9767.	1.7	65
16	Bidirectional modulation of Alzheimer phenotype by alpha-synuclein in mice and primary neurons. Acta Neuropathologica, 2018, 136, 589-605.	3.9	29
17	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.	3.3	45
18	UNC-45A is required for neurite extension via controlling NMII activation. Molecular Biology of the Cell, 2017, 28, 1337-1346.	0.9	16

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19	Amplification of distinct Î±-synuclein fibril conformers through protein misfolding cyclic amplification. Experimental and Molecular Medicine, 2017, 49, e314-e314.	3.2	39
20	Conditional Deletion ofPrnpRescues Behavioral and Synaptic Deficits after Disease Onset in Transgenic Alzheimer's Disease. Journal of Neuroscience, 2017, 37, 9207-9221.	1.7	45
21	Cp/Heph mutant mice have ironâ€induced neurodegeneration diminished by deferiprone. Journal of Neurochemistry, 2015, 135, 958-974.	2.1	35
22	Method for Measuring the Activity of Deubiquitinating Enzymes in Cell Lines and Tissue Samples. Journal of Visualized Experiments, 2015, , e52784.	0.2	3
23	UNC-45A Is a Nonmuscle Myosin IIA Chaperone Required for NK Cell Cytotoxicity via Control of Lytic Granule Secretion. Journal of Immunology, 2015, 195, 4760-4770.	0.4	29
24	Small-Molecule RA-9 Inhibits Proteasome-Associated DUBs and Ovarian Cancer In Vitro and In Vivo via Exacerbating Unfolded Protein Responses. Clinical Cancer Research, 2014, 20, 3174-3186.	3.2	54
25	Neuropsychiatric symptoms in Alzheimer's disease: Past progress and anticipation of the future. Alzheimer's and Dementia, 2013, 9, 602-608.	0.4	292
26	Current Clinical Practices of the Rhinoplasty Society Members. Annals of Plastic Surgery, 2013, 71, 453-455.	0.5	16
27	Accumulation of Toxic Î±-Synuclein Oligomer within Endoplasmic Reticulum Occurs in Î±-SynucleinopathyIn Vivo. Journal of Neuroscience, 2012, 32, 3301-3305.	1.7	272
28	Neurodegenerative phenotypes in an A53T Â-synuclein transgenic mouse model are independent of LRRK2. Human Molecular Genetics, 2012, 21, 2420-2431.	1.4	84
29	Endoplasmic Reticulum Stress Is Important for the Manifestations of Î±-SynucleinopathyIn Vivo. Journal of Neuroscience, 2012, 32, 3306-3320.	1.7	319
30	Magnetization transfer and adiabatic R1Ï•MRI in the brainstem of Parkinsonâ€™s disease. Parkinsonism and Related Disorders, 2012, 18, 623-625.	1.1	14
31	Passive (Amyloid-Î²) Immunotherapy Attenuates Monoaminergic Axonal Degeneration in the AÎ²PPswe/PS1dE9 Mice. Journal of Alzheimer's Disease, 2011, 23, 271-279.	1.2	16
32	Dopaminergic Neuronal Loss, Reduced Neurite Complexity and Autophagic Abnormalities in Transgenic Mice Expressing G2019S Mutant LRRK2. PLoS ONE, 2011, 6, e18568.	1.1	338
33	Resistance to MPTP-Neurotoxicity in Î±-Synuclein Knockout Mice Is Complemented by Human Î±-Synuclein and Associated with Increased Î²-Synuclein and Akt Activation. PLoS ONE, 2011, 6, e16706.	1.1	57
34	Synphilin-1 attenuates neuronal degeneration in the A53T Â-synuclein transgenic mouse model. Human Molecular Genetics, 2010, 19, 2087-2098.	1.4	65
35	Increased Expression of Î±-Synuclein Reduces Neurotransmitter Release by Inhibiting Synaptic Vesicle Reclustering after Endocytosis. Neuron, 2010, 65, 66-79.	3.8	885
36	Regulation of Neuronal Survival Factor MEF2D by Chaperone-Mediated Autophagy. Science, 2009, 323, 124-127.	6.0	282

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37	Differential regulation of small heat shock proteins in transgenic mouse models of neurodegenerative diseases. Neurobiology of Aging, 2008, 29, 586-597.	1.5	44
38	Ubiquitin Proteasome System Stress Underlies Synergistic Killing of Ovarian Cancer Cells by Bortezomib and a Novel HDAC6 Inhibitor. Clinical Cancer Research, 2008, 14, 7340-7347.	3.2	109
39	Amyloid Pathology Is Associated with Progressive Monoaminergic Neurodegeneration in a Transgenic Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2008, 28, 13805-13814.	1.7	180
40	Lysine 63-linked ubiquitination promotes the formation and autophagic clearance of protein inclusions associated with neurodegenerative diseases. Human Molecular Genetics, 2008, 17, 431-439.	1.4	379
41	Wild-type and mutant Î±-synuclein induce a multi-component gene expression profile consistent with shared pathophysiology in different transgenic mouse models of PD. Experimental Neurology, 2007, 204, 421-432.	2.0	46
42	Myosin II Co-Chaperone General Cell UNC-45 Overexpression Is Associated with Ovarian Cancer, Rapid Proliferation, and Motility. American Journal of Pathology, 2007, 171, 1640-1649.	1.9	45
43	Parkinson's Disease Â-Synuclein Transgenic Mice Develop Neuronal Mitochondrial Degeneration and Cell Death. Journal of Neuroscience, 2006, 26, 41-50.	1.7	620
44	Locomotor hyperactivity and alterations in dopamine neurotransmission are associated with overexpression of A53T mutant human Î±-synuclein in mice. Neurobiology of Disease, 2006, 21, 431-443.	2.1	113
45	Ubiquitin-Proteasome System Stress Sensitizes Ovarian Cancer to Proteasome Inhibitorâ€“Induced Apoptosis. Cancer Research, 2006, 66, 3754-3763.	0.4	123
46	Phosphorylation of Ser-129 Is the Dominant Pathological Modification of Î±-Synuclein in Familial and Sporadic Lewy Body Disease. Journal of Biological Chemistry, 2006, 281, 29739-29752.	1.6	1,113
47	Inclusion Body Formation and Neurodegeneration Are Parkin Independent in a Mouse Model of Â-Synucleinopathy. Journal of Neuroscience, 2006, 26, 3685-3696.	1.7	86
48	Vision for the future. , 2006, , 175-186.		0
49	Antiapoptotic property of human Î±-synuclein in neuronal cell lines is associated with the inhibition of caspase-3 but not caspase-9 activity. Journal of Neurochemistry, 2005, 93, 1542-1550.	2.1	30
50	Selected genetically engineered models relevant to human neurodegenerative disease. , 2005, , 176-195.		1
51	Association of DJ-1 and parkin mediated by pathogenic DJ-1 mutations and oxidative stress. Human Molecular Genetics, 2005, 14, 71-84.	1.4	231
52	Aggregation promoting C-terminal truncation of Â-synuclein is a normal cellular process and is enhanced by the familial Parkinson's disease-linked mutations. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 2162-2167.	3.3	405
53	Â-Synuclein Phosphorylation Enhances Eosinophilic Cytoplasmic Inclusion Formation in SH-SY5Y Cells. Journal of Neuroscience, 2005, 25, 5544-5552.	1.7	237
54	AÎ² deposition is associated with enhanced cortical Î±-synuclein lesions in Lewy body diseases. Neurobiology of Aging, 2005, 26, 1183-1192.	1.5	200

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55	Effects of Î±-Synuclein Immunization in a Mouse Model of Parkinsonâ€™s Disease. Neuron, 2005, 46, 857-868.	3.8	533
56	Axonal transport of human Î±â€synuclein slows with aging but is not affected by familial Parkinson's diseaseâ€linked mutations. Journal of Neurochemistry, 2004, 88, 401-410.	2.1	70
57	Stabilization of Â-Synuclein Protein with Aging and Familial Parkinson's Disease-Linked A53T Mutation. Journal of Neuroscience, 2004, 24, 7400-7409.	1.7	166
58	Mutant presenilins specifically elevate the levels of the 42 residue Î²-amyloid peptide in vivo: evidence for augmentation of a 42-specific Î³ secretase. Human Molecular Genetics, 2004, 13, 159-170.	1.4	1,350
59	Human Â-synuclein-harboring familial Parkinson's disease-linked Ala-53 -> Thr mutation causes neurodegenerative disease with Â-synuclein aggregation in transgenic mice. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 8968-8973.	3.3	730
60	Animal Models of PD. Neuron, 2002, 35, 219-222.	3.8	131
61	Accumulation of proteolytic fragments of mutant presenilin 1 and accelerated amyloid deposition are co-regulated in transgenic mice. Neurobiology of Aging, 2002, 23, 171-177.	1.5	18
62	Evidence That Synaptically Released Î²-Amyloid Accumulates as Extracellular Deposits in the Hippocampus of Transgenic Mice. Journal of Neuroscience, 2002, 22, 9785-9793.	1.7	281
63	Advances in genetic models of Parkinson's disease. Clinical Neuroscience Research, 2001, 1, 456-466.	0.8	8
64	The Value of Transgenic Models for the Study of Neurodegenerative Diseases. Research and Perspectives in Alzheimer's Disease, 2001, , 67-86.	0.1	0
65	Enhanced Synaptic Potentiation in Transgenic Mice Expressing presenilin 1 Familial Alzheimer's Disease Mutation Is Normalized with a Benzodiazepine. Neurobiology of Disease, 2000, 7, 54-63.	2.1	62
66	The Value of Transgenic Models for the Study of Neurodegenerative Diseases. Annals of the New York Academy of Sciences, 2000, 920, 179-191.	1.8	51
67	Age-Dependent Emergence and Progression of a Tauopathy in Transgenic Mice Overexpressing the Shortest Human Tau Isoform. Neuron, 1999, 24, 751-762.	3.8	564
68	Neuropathology of preclinical and clinical lateonset Alzheimer's disease. Annals of Neurology, 1998, 43, 673-676.	2.8	83
69	Axonal Transport of Mutant Superoxide Dismutase 1 and Focal Axonal Abnormalities in the Proximal Axons of Transgenic Mice. Neurobiology of Disease, 1998, 5, 27-35.	2.1	96
70	Familial Amyotrophic Lateral Sclerosis and Alzheimerâ€™s Disease. Advances in Experimental Medicine and Biology, 1998, , 145-159.	0.8	7
71	Transgenic Models of Amyotrophic Lateral Sclerosis and Alzheimer's Disease. , 1998, , 107-123.		0
72	ALS-Linked SOD1 Mutant G85R Mediates Damage to Astrocytes and Promotes Rapidly Progressive Disease with SOD1-Containing Inclusions. Neuron, 1997, 18, 327-338.	3.8	1,239

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73	Accelerated Amyloid Deposition in the Brains of Transgenic Mice Coexpressing Mutant Presenilin 1 and Amyloid Precursor Proteins. Neuron, 1997, 19, 939-945.	3.8	964
74	Identification and characterization of a mouse homologue of the Spinal Muscular Atrophy-determining gene, survival motor neuron. Gene, 1997, 204, 47-53.	1.0	44
75	Mutant presenilins of Alzheimer's disease increase production of 42-residue amyloid Î²-protein in both transfected cells and transgenic mice. Nature Medicine, 1997, 3, 67-72.	15.2	1,271
76	Hyperaccumulation of FAD-linked presenilin 1 variants in vivo. Nature Medicine, 1997, 3, 756-760.	15.2	140
77	Perspectives on the Mechanisms of Familial Amyotrophic Lateral Sclerosis Caused by Mutations in Superoxide Dismutase 1. , 1997, , 295-314.		0
78	Transgenic models of neurodegenerative diseases. Current Opinion in Neurobiology, 1996, 6, 651-660.	2.0	30
79	Familial Alzheimer's Diseaseâ€“Linked Presenilin 1 Variants Elevate AÎ²1â€“42/1â€“40 Ratio In Vitro and In Vivo. Neuron, 1996, 17, 1005-1013.	3.8	1,471
80	Endoproteolysis of Presenilin 1 and Accumulation of Processed Derivatives In Vivo. Neuron, 1996, 17, 181-190.	3.8	1,054
81	Altered Reactivity of Superoxide Dismutase in Familial Amyotrophic Lateral Sclerosis. Science, 1996, 271, 515-518.	6.0	715
82	Expression of Presenilin 1 and 2 (PS1 and PS2) in Human and Murine Tissues. Journal of Neuroscience, 1996, 16, 7513-7525.	1.7	279
83	Assessment of normal and mutant human presenilin function in Caenorhabditis elegans. Proceedings of the National Academy of Sciences of the United States of America, 1996, 93, 14940-14944.	3.3	383
84	Mechanisms of selective motor neuron death in transgenic mouse models of motor neuron disease. Neurology, 1996, 47, S54-61; discussion S61-2.	1.5	57
85	Neuronal Intermediate Filaments. Annual Review of Neuroscience, 1996, 19, 187-217.	5.0	419
86	Transgenic and gene-targeting approaches to model disorders of motor neurons. Seminars in Neuroscience, 1996, 8, 163-169.	2.3	2
87	Inherited Neurodegenerative Diseases and Transgenic Models. Brain Pathology, 1996, 6, 467-480.	2.1	9
88	A vector for expressing foreign genes in the brains and hearts of transgenic mice. Genetic Analysis, Techniques and Applications, 1996, 13, 159-163.	1.5	323
89	Neurofilament subunit NF-H modulates axonal diameter by selectively slowing neurofilament transport.. Journal of Cell Biology, 1996, 135, 711-724.	2.3	173
90	Subunit composition of neurofilaments specifies axonal diameter.. Journal of Cell Biology, 1996, 133, 1061-1069.	2.3	159

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91	The Microtubule-associated Protein Tau Is Extensively Modified with O-linked N-acetylglucosamine. Journal of Biological Chemistry, 1996, 271, 28741-28744.	1.6	296
92	Title is missing!. Cold Spring Harbor Symposia on Quantitative Biology, 1996, 61, 709-723.	2.0	31
93	Motor Neuron Disease and Model Systems: Aetiologies, Mechanisms and Therapies. Novartis Foundation Symposium, 1996, 196, 3-17.	1.2	1
94	Superoxide Dismutase 1 Subunits with Mutations Linked to Familial Amyotrophic Lateral Sclerosis Do Not Affect Wild-type Subunit Function. Journal of Biological Chemistry, 1995, 270, 3234-3238.	1.6	142
95	Nucleotide sequence of the chromosome 14-encodedS182cDNA and revised secondary structure prediction. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 1995, 2, 188-190.	1.4	18
96	Mutations associated with amyotrophic lateral sclerosis convert superoxide dismutase from an antiapoptotic gene to a proapoptotic gene: studies in yeast and neural cells.. Proceedings of the National Academy of Sciences of the United States of America, 1995, 92, 3024-3028.	3.3	318
97	An adverse property of a familial ALS-linked SOD1 mutation causes motor neuron disease characterized by vacuolar degeneration of mitochondria. Neuron, 1995, 14, 1105-1116.	3.8	1,394
98	A mutant neurofilament subunit causes massive, selective motor neuron death: Implications for the pathogenesis of human motor neuron disease. Neuron, 1994, 13, 975-988.	3.8	368
99	Neurofilament function and dysfunction: involvement in axonal growth and neuronal disease. Current Opinion in Cell Biology, 1994, 6, 34-40.	2.6	140
100	Superoxide dismutase 1 with mutations linked to familial amyotrophic lateral sclerosis possesses significant activity.. Proceedings of the National Academy of Sciences of the United States of America, 1994, 91, 8292-8296.	3.3	548
101	Neurofilaments are obligate heteropolymers in vivo. Journal of Cell Biology, 1993, 122, 1337-1350.	2.3	370
102	Characterization of posttranslational modifications in neuron-specific class III beta-tubulin by mass spectrometry.. Proceedings of the National Academy of Sciences of the United States of America, 1991, 88, 4685-4689.	3.3	246
103	The expression and posttranslational modification of a neuron-specific ?-tubulin isotype during chick embryogenesis. Cytoskeleton, 1990, 17, 118-132.	4.4	550
104	Posttranslational modification of class III beta-tubulin.. Proceedings of the National Academy of Sciences of the United States of America, 1990, 87, 7195-7199.	3.3	189
105	Memory enhancement with posttraining intraventricular glucose injections in rats.. Behavioral Neuroscience, 1988, 102, 591-595.	0.6	97
106	Memory enhancement with posttraining intraventricular glucose injections in rats. Behavioral Neuroscience, 1988, 102, 591-5.	0.6	33