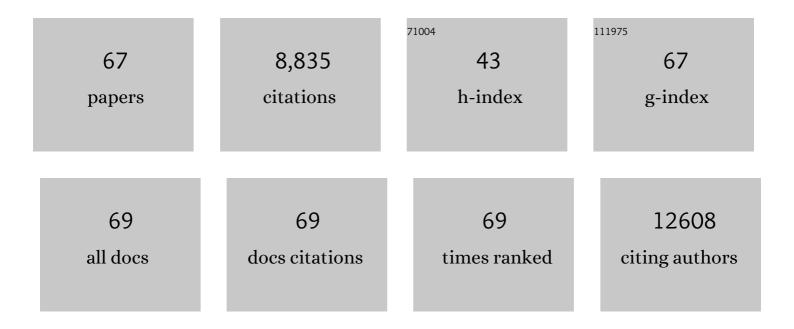
Masashi Kitazawa

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Inflammatory Cytokine IL-1β Downregulates Endothelial LRP1 via MicroRNA-mediated Gene Silencing. Neuroscience, 2021, 453, 69-80.	1.1	4
2	Generation of a humanized Al̂² expressing mouse demonstrating aspects of Alzheimer's disease-like pathology. Nature Communications, 2021, 12, 2421.	5.8	53
3	Effect of Lipopolysaccharide and TNFα on Neuronal Ascorbic Acid Uptake. Mediators of Inflammation, 2021, 2021, 1-11.	1.4	7
4	Calsyntenin-3 interacts with the sodium-dependent vitamin C transporter-2 to regulate vitamin C uptake. International Journal of Biological Macromolecules, 2021, 192, 1178-1184.	3.6	5
5	Genetic Ablation of Hematopoietic Cell Kinase Accelerates Alzheimer's Disease–Like Neuropathology in Tg2576 Mice. Molecular Neurobiology, 2020, 57, 2447-2460.	1.9	15
6	Chronic copper exposure directs microglia towards degenerative expression signatures in wild-type and J20 mouse model of Alzheimer's disease. Journal of Trace Elements in Medicine and Biology, 2020, 62, 126578.	1.5	13
7	miRâ€181a negatively modulates synaptic plasticity in hippocampal cultures and its inhibition rescues memory deficits in a mouse model of Alzheimer's disease. Aging Cell, 2020, 19, e13118.	3.0	42
8	Intra- and extracellular β-amyloid overexpression via adeno-associated virus-mediated gene transfer impairs memory and synaptic plasticity in the hippocampus. Scientific Reports, 2019, 9, 15936.	1.6	12
9	Amyloid-beta impairs TOM1-mediated IL-1R1 signaling. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 21198-21206.	3.3	24
10	Metal Toxicity Links to Alzheimer's Disease and Neuroinflammation. Journal of Molecular Biology, 2019, 431, 1843-1868.	2.0	281
11	Copper-Induced Upregulation of MicroRNAs Directs the Suppression of Endothelial LRP1 in Alzheimer's Disease Model. Toxicological Sciences, 2019, 170, 144-156.	1.4	23
12	Environmental and Dietary Exposure to Copper and Its Cellular Mechanisms Linking to Alzheimer's Disease. Toxicological Sciences, 2018, 163, 338-345.	1.4	59
13	Inhibition of hematopoietic cell kinase dysregulates microglial function and accelerates early stage Alzheimer's diseaseâ€ i ike neuropathology. Clia, 2018, 66, 2700-2718.	2.5	24
14	The emerging risk of exposure to air pollution onÂcognitive decline and Alzheimer's disease – Evidence from epidemiological and animal studies. Biomedical Journal, 2018, 41, 141-162.	1.4	161
15	Inflammatory Cytokine, IL-1β, Regulates Glial Glutamate Transporter via microRNA-181a in vitro. Journal of Alzheimer's Disease, 2018, 63, 965-975.	1.2	16
16	Impaired <scp>AMPA</scp> signaling and cytoskeletal alterations induce early synaptic dysfunction in a mouse model of Alzheimer's disease. Aging Cell, 2018, 17, e12791.	3.0	58
17	Astrocyte transport of glutamate and neuronal activity reciprocally modulate tau pathology in Drosophila. Neuroscience, 2017, 348, 191-200.	1.1	19
18	Impaired In Vivo Gamma Oscillations in the Medial Entorhinal Cortex of Knock-in Alzheimer Model. Frontiers in Systems Neuroscience, 2017, 11, 48.	1.2	52

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19	Copper Exposure Perturbs Brain Inflammatory Responses and Impairs Clearance of Amyloid-Beta. Toxicological Sciences, 2016, 152, 194-204.	1.4	75
20	The Myoblast C2C12 Transfected with Mutant Valosin-Containing Protein Exhibits Delayed Stress Granule Resolution on Oxidative Stress. American Journal of Pathology, 2016, 186, 1623-1634.	1.9	21
21	Ceftriaxone ameliorates tau pathology and cognitive decline via restoration of glial glutamate transporter in a mouse model of Alzheimer's disease. Neurobiology of Aging, 2015, 36, 2260-2271.	1.5	128
22	Infection, systemic inflammation, and Alzheimer's disease. Microbes and Infection, 2015, 17, 549-556.	1.0	81
23	Upregulation of miR-181 Decreases c-Fos and SIRT-1 in the Hippocampus of 3xTg-AD Mice. Journal of Alzheimer's Disease, 2014, 42, 1229-1238.	1.2	77
24	α7 Nicotinic Receptor Agonist Enhances Cognition in Aged 3xTg-AD Mice with Robust Plaques and Tangles. American Journal of Pathology, 2014, 184, 520-529.	1.9	68
25	Endogenous murine tau promotes neurofibrillary tangles in 3xTg-AD mice without affecting cognition. Neurobiology of Disease, 2014, 62, 407-415.	2.1	19
26	Colony-Stimulating Factor 1 Receptor Signaling Is Necessary for Microglia Viability, Unmasking a Microglia Progenitor Cell in the Adult Brain. Neuron, 2014, 82, 380-397.	3.8	1,350
27	Restoration of Lipoxin A4 Signaling Reduces Alzheimer's Disease-Like Pathology in the 3xTg-AD Mouse Model. Journal of Alzheimer's Disease, 2014, 43, 893-903.	1.2	76
28	Neuronal-Specific Overexpression of a Mutant Valosin-Containing Protein Associated with IBMPFD Promotes Aberrant Ubiquitin and TDP-43 Accumulation and Cognitive Dysfunction in Transgenic Mice. American Journal of Pathology, 2013, 183, 504-515.	1.9	35
29	Aspirin-Triggered Lipoxin A4 Stimulates Alternative Activation of Microglia and Reduces Alzheimer Disease–Like Pathology in Mice. American Journal of Pathology, 2013, 182, 1780-1789.	1.9	139
30	Transgenic Mouse Models of Alzheimer Disease: Developing a Better Model as a Tool for Therapeutic Interventions. Current Pharmaceutical Design, 2012, 18, 1131-1147.	0.9	146
31	Calpain Inhibitor A-705253 Mitigates Alzheimer's Disease–Like Pathology and Cognitive Decline in Aged 3xTgAD Mice. American Journal of Pathology, 2012, 181, 616-625.	1.9	80
32	The Homozygote VCPR155H/R155H Mouse Model Exhibits Accelerated Human VCP-Associated Disease Pathology. PLoS ONE, 2012, 7, e46308.	1.1	56
33	Inflammation Induced by Infection Potentiates Tau Pathological Features in Transgenic Mice. American Journal of Pathology, 2011, 178, 2811-2822.	1.9	166
34	Loss of Muscarinic M1 Receptor Exacerbates Alzheimer's Disease–Like Pathology and Cognitive Decline. American Journal of Pathology, 2011, 179, 980-991.	1.9	100
35	Long term changes in phospho-APP and tau aggregation in the 3xTg-AD mice following cerebral ischemia. Neuroscience Letters, 2011, 495, 55-59.	1.0	32
36	Blocking IL-1 Signaling Rescues Cognition, Attenuates Tau Pathology, and Restores Neuronal β-Catenin Pathway Function in an Alzheimer's Disease Model. Journal of Immunology, 2011, 187, 6539-6549.	0.4	359

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37	The 3xTg-AD Mouse Model: Reproducing and Modulating Plaque and Tangle Pathology. Neuromethods, 2011, , 469-482.	0.2	3
38	Amyloid-β protein impairs Ca2+ release and contractility in skeletal muscle. Neurobiology of Aging, 2010, 31, 2080-2090.	1.5	52
39	Memantine Improves Cognition and Reduces Alzheimer's-Like Neuropathology in Transgenic Mice. American Journal of Pathology, 2010, 176, 870-880.	1.9	188
40	Treatment of Alzheimer's Disease with Anti-Homocysteic Acid Antibody in 3xTg-AD Male Mice. PLoS ONE, 2010, 5, e8593.	1.1	31
41	VCP Associated Inclusion Body Myopathy and Paget Disease of Bone Knock-In Mouse Model Exhibits Tissue Pathology Typical of Human Disease. PLoS ONE, 2010, 5, e13183.	1.1	109
42	Immunization with Amyloid-β Attenuates Inclusion Body Myositis-Like Myopathology and Motor Impairment in a Transgenic Mouse Model. Journal of Neuroscience, 2009, 29, 6132-6141.	1.7	22
43	Chronic copper exposure exacerbates both amyloid and tau pathology and selectively dysregulates cdk5 in a mouse model of AD. Journal of Neurochemistry, 2009, 108, 1550-1560.	2.1	139
44	Neural stem cells improve cognition via BDNF in a transgenic model of Alzheimer disease. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 13594-13599.	3.3	761
45	Inflammation induces tau pathology in inclusion body myositis model via glycogen synthase kinaseâ€3β. Annals of Neurology, 2008, 64, 15-24.	2.8	76
46	Environmental neurotoxin dieldrin induces apoptosis via caspase-3-dependent proteolytic activation of protein kinase C delta (PKCdelta): Implications for neurodegeneration in Parkinson's disease. Molecular Brain, 2008, 1, 12.	1.3	58
47	Neural Stem Cells Improve Memory in an Inducible Mouse Model of Neuronal Loss. Journal of Neuroscience, 2007, 27, 11925-11933.	1.7	149
48	Genetically Augmenting Aβ42 Levels in Skeletal Muscle Exacerbates Inclusion Body Myositis-Like Pathology and Motor Deficits in Transgenic Mice. American Journal of Pathology, 2006, 168, 1986-1997.	1.9	62
49	Pathogenic accumulation of APP in fast twitch muscle of IBM patients and a transgenic model. Neurobiology of Aging, 2006, 27, 423-432.	1.5	43
50	Reduction of Soluble Aβ and Tau, but Not Soluble Aβ Alone, Ameliorates Cognitive Decline in Transgenic Mice with Plaques and Tangles. Journal of Biological Chemistry, 2006, 281, 39413-39423.	1.6	262
51	Activation of protein kinase Cδ by proteolytic cleavage contributes to manganese-induced apoptosis in dopaminergic cells: protective role of Bcl-2. Biochemical Pharmacology, 2005, 69, 133-146.	2.0	63
52	Protein Kinase Cδls a Key Downstream Mediator of Manganese-Induced Apoptosis in Dopaminergic Neuronal Cells. Journal of Pharmacology and Experimental Therapeutics, 2005, 313, 46-55.	1.3	143
53	Lipopolysaccharide-Induced Inflammation Exacerbates Tau Pathology by a Cyclin-Dependent Kinase 5-Mediated Pathway in a Transgenic Model of Alzheimer's Disease. Journal of Neuroscience, 2005, 25, 8843-8853.	1.7	607
54	Dieldrin-Induced Neurotoxicity: Relevance to Parkinson's Disease Pathogenesis. NeuroToxicology, 2005, 26, 701-719.	1.4	172

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55	Antipodal Effects of p25 on Synaptic Plasticity, Learning, and Memory—Too Much of a Good Thing Is Bad. Neuron, 2005, 48, 711-712.	3.8	7
56	Amyloid β-Peptide: The Inside Story. Current Alzheimer Research, 2004, 1, 231-239.	0.7	65
57	Microglia as a Potential Bridge between the Amyloid Â-Peptide and Tau. Annals of the New York Academy of Sciences, 2004, 1035, 85-103.	1.8	140
58	Blockade of PKCÂ Proteolytic Activation by Loss of Function Mutants Rescues Mesencephalic Dopaminergic Neurons from Methylcyclopentadienyl Manganese Tricarbonyl (MMT)-Induced Apoptotic Cell Death. Annals of the New York Academy of Sciences, 2004, 1035, 271-289.	1.8	24
59	Dieldrin Promotes Proteolytic Cleavage of Poly(ADP-Ribose) Polymerase and Apoptosis in Dopaminergic Cells: Protective Effect of Mitochondrial Anti-Apoptotic Protein Bcl-2. NeuroToxicology, 2004, 25, 589-598.	1.4	49
60	Caspase-3 dependent proteolytic activation of protein kinase Cdelta mediates and regulates 1-methyl-4-phenylpyridinium (MPP+)-induced apoptotic cell death in dopaminergic cells: relevance to oxidative stress in dopaminergic degeneration. European Journal of Neuroscience, 2003, 18, 1387-1401.	1.2	158
61	Role of Proteolytic Activation of Protein Kinase Cδ in Oxidative Stress-Induced Apoptosis. Antioxidants and Redox Signaling, 2003, 5, 609-620.	2.5	122
62	Amyloid deposition precedes tangle formation in a triple transgenic model of Alzheimer's disease. Neurobiology of Aging, 2003, 24, 1063-1070.	1.5	840
63	Dieldrin induces apoptosis by promoting caspase-3-dependent proteolytic cleavage of protein kinase Cδ in dopaminergic cells: relevance to oxidative stress and dopaminergic degeneration. Neuroscience, 2003, 119, 945-964.	1.1	151
64	Oxidative Stress and Mitochondrial-Mediated Apoptosis in Dopaminergic Cells Exposed to Methylcyclopentadienyl Manganese Tricarbonyl. Journal of Pharmacology and Experimental Therapeutics, 2002, 302, 26-35.	1.3	81
65	Caspase-3-Dependent Proteolytic Cleavage of Protein Kinase Cδ Is Essential for Oxidative Stress-Mediated Dopaminergic Cell Death after Exposure to Methylcyclopentadienyl Manganese Tricarbonyl. Journal of Neuroscience, 2002, 22, 1738-1751.	1.7	210
66	A novel oxidative stress dependent apoptotic pathway in pesticideâ€induced dopaminergic degeneration in PD models. Journal of Neurochemistry, 2002, 81, 76-76.	2.1	1
67	Dieldrin-induced oxidative stress and neurochemical changes contribute to apoptopic cell death in dopaminergic cells. Free Radical Biology and Medicine, 2001, 31, 1473-1485.	1.3	171