Takashi Saito

List of Publications by Year
in descending order

145 papers	9,553 citations	57681 46 h-index	53065 89 g-index
168 all docs	168 docs citations	168 times ranked	15095 citing authors

#	Article	IF	Citations
1	Presubiculum principal cells are preserved from degeneration in knock-in APP/TAU mouse models of Alzheimerâ€™s disease. Seminars in Cell and Developmental Biology, 2023, 139, 55-72.	2.3	8
2	¹¹C-PiB and¹²⁴I-Antibody PET Provide Differing Estimates of Brain Amyloid-Î² After Therapeutic Intervention. Journal of Nuclear Medicine, 2022, 63, 302-309.	2.8	19
3	Somatostatin-evoked AÎ² catabolism in the brain: Mechanistic involvement of Î±-endosulfine-KATP channel pathway. Molecular Psychiatry, 2022, 27, 1816-1828.	4.1	11
4	Deficiency of MTH1 and/or OGG1 increases the accumulation of 8-oxoguanine in the brain of the AppNL-G-F/NL-G-F knock-in mouse model of Alzheimerâ€™s disease, accompanied by accelerated microgliosis and reduced anxiety-like behavior. Neuroscience Research, 2022, 177, 118-134.	1.0	3
5	Therapeutic effects of anti-amyloid Î² antibody after intravenous injection and efficient nose-to-brain delivery in Alzheimerâ€™s disease mouse model. Drug Delivery and Translational Research, 2022, , 1.	3.0	2
6	Disrupted neural correlates of anesthesia and sleep reveal early circuit dysfunctions in Alzheimer models. Cell Reports, 2022, 38, 110268.	2.9	13
7	Assessing Sex-Specific Circadian, Metabolic, and Cognitive Phenotypes in the AÎ²PP/PS1 and APPNL-F/NL-F Models of Alzheimerâ€™s Disease. Journal of Alzheimer's Disease, 2022, 85, 1077-1093.	1.2	5
8	Periodontal Infection Aggravates C1q-Mediated Microglial Activation and Synapse Pruning in Alzheimerâ€™s Mice. Frontiers in Immunology, 2022, 13, 816640.	2.2	15
9	Astrocytes deficient in circadian clock gene Bmal1 show enhanced activation responses to amyloid-beta pathology without changing plaque burden. Scientific Reports, 2022, 12, 1796.	1.6	22
10	Lipid flippase dysfunction as a therapeutic target for endosomal anomalies in Alzheimerâ€™s disease. IScience, 2022, 25, 103869.	1.9	7
11	Recent Advances in the Modeling of Alzheimerâ€™s Disease. Frontiers in Neuroscience, 2022, 16, 807473.	1.4	55
12	AAVâ€mediated delivery of an antiâ€BACE1 VHH alleviates pathology in an Alzheimer's disease model. EMBO Molecular Medicine, 2022, 14, e09824.	3.3	13
13	Epigenetic repression of Wnt receptors in AD: a role for Sirtuin2-induced H4K16ac deacetylation of Frizzled1 and Frizzled7 promoters. Molecular Psychiatry, 2022, 27, 3024-3033.	4.1	16
14	Endothelial expression of human amyloid precursor protein leads to amyloid Î² in the blood and induces cerebral amyloid angiopathy in knock-in mice. Journal of Biological Chemistry, 2022, 298, 101880.	1.6	8
15	Assessments of prolonged effects of desflurane and sevoflurane on motor learning deficits in aged AppNL-G-F/NL-G-F mice. Molecular Brain, 2022, 15, 32.	1.3	2
16	Amelioration of Alzheimerâ€™s Disease by Gut-Pancreas-Liver-Brain Interaction in an App Knock-In Mouse Model. Life, 2022, 12, 34.	1.1	3
17	High Correlation among Brain-Derived Major Protein Levels in Cerebrospinal Fluid: Implication for Amyloid-Beta and Tau Protein Changes in Alzheimerâ€™s Disease. Metabolites, 2022, 12, 355.	1.3	3
18	Impairment of ciliary dynamics in an APP knock-in mouse model of Alzheimer's disease. Biochemical and Biophysical Research Communications, 2022, 610, 85-91.	1.0	4

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19	Expression of Olfactory-Related Genes in the Olfactory Epithelium of an Alzheimerâ€™s Disease Mouse Model. Journal of Alzheimer's Disease, 2022, , 1-7.	1.2	1
20	Effects of highâ€fat diet on nutrient metabolism and cognitive functions in young <scp> APPKI ^{NLâ€Gâ€F/NLâ€Gâ€F} </scp> mice. Neuropsychopharmacology Reports, 2022, , .	1.1	1
21	Propolis Promotes Memantine-Dependent Rescue of Cognitive Deficits in APP-KI Mice. Molecular Neurobiology, 2022, 59, 4630-4646.	1.9	4
22	Early memory deficits and extensive brain network disorganization in the App/MAPT double knock-in mouse model of familial Alzheimerâ€™s disease. Aging Brain, 2022, 2, 100042.	0.7	5
23	An isogenic panel of <i>App</i> knock-in mouse models: Profiling Î²-secretase inhibition and endosomal abnormalities. Science Advances, 2022, 8, .	4.7	6
24	Terminal complement pathway activation drives synaptic loss in Alzheimerâ€™s disease models. Acta Neuropathologica Communications, 2022, 10, .	2.4	19
25	Hippocampal neural circuit connectivity alterations in an Alzheimer's disease mouse model revealed by monosynaptic rabies virus tracing. Neurobiology of Disease, 2022, 172, 105820.	2.1	8
26	Tau-binding protein PRMT8 facilitates vacuole degeneration in the brain. Journal of Biochemistry, 2022, 172, 233-243.	0.9	2
27	Early-life stress induces the development of Alzheimer's disease pathology via angiopathy. Experimental Neurology, 2021, 337, 113552.	2.0	17
28	Pulse-Chase Proteomics of the App Knockin Mouse Models of Alzheimerâ€™s Disease Reveals that Synaptic Dysfunction Originates in Presynaptic Terminals. Cell Systems, 2021, 12, 141-158.e9.	2.9	32
29	A potential defense mechanism against amyloid deposition in cerebellum. Biochemical and Biophysical Research Communications, 2021, 535, 25-32.	1.0	7
30	Microglial gene signature reveals loss of homeostatic microglia associated with neurodegeneration of Alzheimerâ€™s disease. Acta Neuropathologica Communications, 2021, 9, 1.	2.4	172
31	Integrated analysis of behavioral, epigenetic, and gut microbiome analyses in AppNL-G-F, AppNL-F, and wild type mice. Scientific Reports, 2021, 11, 4678.	1.6	38
32	Extracellular Release of ILEI/FAM3C and Amyloid-Î² Is Associated with the Activation of Distinct Synapse Subpopulations. Journal of Alzheimer's Disease, 2021, 80, 159-174.	1.2	5
33	PET imaging of colony-stimulating factor 1 receptor: A head-to-head comparison of a novel radioligand, ¹¹C-GW2580, and ¹¹C-CPPC, in mouse models of acute and chronic neuroinflammation and a rhesus monkey. Journal of Cerebral Blood Flow and Metabolism, 2021, 41, 2410-2422.	2.4	36
34	Enhancing calmodulin binding to ryanodine receptor is crucial to limit neuronal cell loss in Alzheimer disease. Scientific Reports, 2021, 11, 7289.	1.6	14
35	Early identification of Alzheimer's disease in mouse models: Application of deep neural network algorithm to cognitive behavioral parameters. IScience, 2021, 24, 102198.	1.9	14
36	Plaque associated microglia hyper-secrete extracellular vesicles and accelerate tau propagation in a humanized APP mouse model. Molecular Neurodegeneration, 2021, 16, 18.	4.4	97

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37	Tooth Loss Induces Memory Impairment and Gliosis in App Knock-In Mouse Models of Alzheimerâ€™s Disease. Journal of Alzheimer's Disease, 2021, 80, 1687-1704.	1.2	11
38	Modality-Specific Impairment of Hippocampal CA1 Neurons of Alzheimerâ€™s Disease Model Mice. Journal of Neuroscience, 2021, 41, 5315-5329.	1.7	11
39	Multi-scale network imaging in a mouse model of amyloidosis. Cell Calcium, 2021, 95, 102365.	1.1	9
40	Knock-in models related to Alzheimerâ€™s disease: synaptic transmission, plaques and the role of microglia. Molecular Neurodegeneration, 2021, 16, 47.	4.4	27
41	A highâ€fat diet exacerbates the Alzheimer's disease pathology in the hippocampus of the <i>App</i><i>^{NLâˆ’F/NLâˆ’F}</i> knockâ€in mouse model. Aging Cell, 2021, 20, e13429.	3.0	19
42	Suppression of amyloidâ€Î² secretion from neurons by <i>cis</i>â€9, <i>trans</i>â€11â€octadecadienoic acid, an isomer of conjugated linoleic acid. Journal of Neurochemistry, 2021, 159, 603-617.	2.1	3
43	Casein Kinase 2 dependent phosphorylation of eIF4B regulates BACE1 expression in Alzheimerâ€™s disease. Cell Death and Disease, 2021, 12, 769.	2.7	8
44	Neuronal Cell Cycle Re-Entry Enhances Neuropathological Features in AppNLF Knock-In Mice. Journal of Alzheimer's Disease, 2021, 82, 1683-1702.	1.2	7
45	Widespread Reduced Density of Noradrenergic Locus Coeruleus Axons in the App Knock-In Mouse Model of Amyloid-Î² Amyloidosis. Journal of Alzheimer's Disease, 2021, 82, 1513-1530.	1.2	7
46	A third-generation mouse model of Alzheimer's disease shows early and increased cored plaque pathology composed of wild-type human amyloid Î² peptide. Journal of Biological Chemistry, 2021, 297, 101004.	1.6	16
47	Transferrin Biosynthesized in the Brain Is a Novel Biomarker for Alzheimerâ€™s Disease. Metabolites, 2021, 11, 616.	1.3	16
48	The AppNL-G-F mouse retina is a site for preclinical Alzheimerâ€™s disease diagnosis and research. Acta Neuropathologica Communications, 2021, 9, 6.	2.4	22
49	Distinct microglial response against Alzheimer's amyloid and tau pathologies characterized by P2Y12 receptor. Brain Communications, 2021, 3, fcab011.	1.5	41
50	HMGB1 signaling phosphorylates Ku70 and impairs DNA damage repair in Alzheimerâ€™s disease pathology. Communications Biology, 2021, 4, 1175.	2.0	14
51	Microglia and CD206+ border-associated mouse macrophages maintain their embryonic origin during Alzheimerâ€™s disease. ELife, 2021, 10, .	2.8	16
52	Identification and drug-induced reversion of molecular signatures of Alzheimerâ€™s disease onset and progression in AppNL-G-F, AppNL-F, and 3xTg-AD mouse models. Genome Medicine, 2021, 13, 168.	3.6	7
53	Microglia-Based Sex-Biased Neuropathology in Early-Stage Alzheimerâ€™s Disease Model Mice and the Potential Pharmacologic Efficacy of Dioscin. Cells, 2021, 10, 3261.	1.8	5
54	MUTYH Actively Contributes to Microglial Activation and Impaired Neurogenesis in the Pathogenesis of Alzheimerâ€™s Disease. Oxidative Medicine and Cellular Longevity, 2021, 2021, 1-30.	1.9	17

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55	Contribution of GABAergic interneurons to amyloid-Î² plaque pathology in an APP knock-in mouse model. Molecular Neurodegeneration, 2020, 15, 3.	4.4	26
56	Increased levels of AÎ²42 decrease the lifespan of ob/ob mice with dysregulation of microglia and astrocytes. FASEB Journal, 2020, 34, 2425-2435.	0.2	15
57	Gene-environment interaction promotes Alzheimer's risk as revealed by synergy of repeated mild traumatic brain injury and mouse App knock-in. Neurobiology of Disease, 2020, 145, 105059.	2.1	2
58	Disrupted Place Cell Remapping and Impaired Grid Cells in a Knockin Model of Alzheimer's Disease. Neuron, 2020, 107, 1095-1112.e6.	3.8	82
59	Touchscreen-based location discrimination and paired associate learning tasks detect cognitive impairment at an early stage in an App knock-in mouse model of Alzheimerâ€™s disease. Molecular Brain, 2020, 13, 147.	1.3	13
60	Prodromal Alzheimerâ€™s Disease: Constitutive Upregulation of Neuroglobin Prevents the Initiation of Alzheimerâ€™s Pathology. Frontiers in Neuroscience, 2020, 14, 562581.	1.4	8
61	Impact of Hyperhomocysteinemia and Different Dietary Interventions on Cognitive Performance in a Knock-in Mouse Model for Alzheimerâ€™s Disease. Nutrients, 2020, 12, 3248.	1.7	8
62	The two faces of synaptic failure in AppNL-G-F knock-in mice. Alzheimer's Research and Therapy, 2020, 12, 100.	3.0	25
63	Astaxanthin Ameliorated Parvalbumin-Positive Neuron Deficits and Alzheimerâ€™s Disease-Related Pathological Progression in the Hippocampus of AppNL-G-F/NL-G-F Mice. Frontiers in Pharmacology, 2020, 11, 307.	1.6	27
64	Amyloid Î² induces interneuron-specific changes in the hippocampus of APPNL-F mice. PLoS ONE, 2020, 15, e0233700.	1.1	17
65	Oral glutathione administration inhibits the oxidative stress and the inflammatory responses in AppNLâ^ˆG-F/NLâ^ˆG-F knock-in mice. Neuropharmacology, 2020, 168, 108026.	2.0	26
66	Analysis of Taste Sensitivities in App Knock-In Mouse Model of Alzheimerâ€™s Disease. Journal of Alzheimer's Disease, 2020, 76, 997-1004.	1.2	5
67	Î²-amyloid redirects norepinephrine signaling to activate the pathogenic GSK3Î²/tau cascade. Science Translational Medicine, 2020, 12, .	5.8	86
68	Nrf2 Suppresses Oxidative Stress and Inflammation in <i>App</i> Knock-In Alzheimerâ€™s Disease Model Mice. Molecular and Cellular Biology, 2020, 40, .	1.1	98
69	YAP-dependent necrosis occurs in early stages of Alzheimerâ€™s disease and regulates mouse model pathology. Nature Communications, 2020, 11, 507.	5.8	62
70	Versatile whole-organ/body staining and imaging based on electrolyte-gel properties of biological tissues. Nature Communications, 2020, 11, 1982.	5.8	134
71	Proteomics Time-Course Study of App Knock-In Mice Reveals Novel Presymptomatic AÎ²42-Induced Pathways to Alzheimerâ€™s Disease Pathology. Journal of Alzheimer's Disease, 2020, 75, 321-335.	1.2	9
72	Retinal Thickness Changes Over Time in a Murine AD Model APPNL-F/NL-F. Frontiers in Aging Neuroscience, 2020, 12, 625642.	1.7	10

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73	Progressive Changes in Sleep and Its Relations to Amyloid-Î² Distribution and Learning in Single<i>App</i>Knock-In Mice. ENeuro, 2020, 7, ENEURO.0093-20.2020.	0.9	9
74	Fibrillar AÎ² triggers microglial proteome alterations and dysfunction in Alzheimer mouse models. ELife, 2020, 9, .	2.8	80
75	Looking beyond the standard version of the Morris water task in the assessment of mouse models of cognitive deficits. Hippocampus, 2019, 29, 3-14.	0.9	12
76	Longitudinal PET Monitoring of Amyloidosis and Microglial Activation in a Second-Generation Amyloid-Î² Mouse Model. Journal of Nuclear Medicine, 2019, 60, 1787-1793.	2.8	41
77	Humanization of the entire murine Mapt gene provides a murine model of pathological human tau propagation. Journal of Biological Chemistry, 2019, 294, 12754-12765.	1.6	114
78	Serine Phosphorylation of IRS1 Correlates with AÎ²-Unrelated Memory Deficits and Elevation in AÎ² Level Prior to the Onset of Memory Decline in AD. Nutrients, 2019, 11, 1942.	1.7	13
79	ABCA7 haplodeficiency disturbs microglial immune responses in the mouse brain. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 23790-23796.	3.3	43
80	Guidelines for the use of flow cytometry and cell sorting in immunological studies (second edition). European Journal of Immunology, 2019, 49, 1457-1973.	1.6	766
81	Inhibition of p38 MAPK in the brain through nasal administration of p38 inhibitor loaded in chitosan nanocapsules. Nanomedicine, 2019, 14, 2409-2422.	1.7	11
82	Network-guided analysis of hippocampal proteome identifies novel proteins that colocalize with AÎ² in a mice model of early-stage Alzheimerâ€™s disease. Neurobiology of Disease, 2019, 132, 104603.	2.1	13
83	An impaired intrinsic microglial clock system induces neuroinflammatory alterations in the early stage of amyloid precursor protein knock-in mouse brain. Journal of Neuroinflammation, 2019, 16, 173.	3.1	33
84	Amyloid Î² oligomers constrict human capillaries in Alzheimerâ€™s disease via signaling to pericytes. Science, 2019, 365, .	6.0	436
85	App mice overall do not show impaired motivation, but cored amyloid plaques in the striatum are inversely correlated with motivation. Neurochemistry International, 2019, 129, 104470.	1.9	5
86	An App knock-in mouse inducing the formation of a toxic conformer of AÎ² as a model for evaluating only oligomer-induced cognitive decline in Alzheimer's disease. Biochemical and Biophysical Research Communications, 2019, 515, 462-467.	1.0	14
87	Tau binding protein CAPON induces tau aggregation and neurodegeneration. Nature Communications, 2019, 10, 2394.	5.8	59
88	Temporal progression of Alzheimer's disease in brains and intestines of transgenic mice. Neurobiology of Aging, 2019, 81, 166-176.	1.5	31
89	Aminophospholipids are signal-transducing TREM2 ligands on apoptotic cells. Scientific Reports, 2019, 9, 7508.	1.6	61
90	SIRT3 mediates hippocampal synaptic adaptations to intermittent fasting and ameliorates deficits in APP mutant mice. Nature Communications, 2019, 10, 1886.	5.8	114

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91	Amyloid-Î² plaque formation and reactive gliosis are required for induction of cognitive deficits in App knock-in mouse models of Alzheimerâ€™s disease. BMC Neuroscience, 2019, 20, 13.	0.8	37
92	Reducing ADAMTS-3 Inhibits Amyloid Î² Deposition in <i>App</i> Knock-in Mouse. Biological and Pharmaceutical Bulletin, 2019, 42, 354-356.	0.6	8
93	Aberrant Excitatoryâ€“Inhibitory Synaptic Mechanisms in Entorhinal Cortex Microcircuits During the Pathogenesis of Alzheimerâ€™s Disease. Cerebral Cortex, 2019, 29, 1834-1850.	1.6	90
94	Insoluble AÎ² overexpression in an <i>App</i> knock-in mouse model alters microstructure and gamma oscillations in the prefrontal cortex, and impacts on anxiety-related behaviours. DMM Disease Models and Mechanisms, 2019, 12, .	1.2	25
95	Biology of splicing in Alzheimer's disease research. Progress in Molecular Biology and Translational Science, 2019, 168, 79-84.	0.9	0
96	Subtle behavioral changes and increased prefrontal-hippocampal network synchronicity in APPNLâ€'Gâ€'F mice before prominent plaque deposition. Behavioural Brain Research, 2019, 364, 431-441.	1.2	63
97	The Disease-modifying Drug Candidate, SAK3 Improves Cognitive Impairment and Inhibits Amyloid beta Deposition in App Knock-in Mice. Neuroscience, 2018, 377, 87-97.	1.1	22
98	Spatial reversal learning defect coincides with hypersynchronous telencephalic BOLD functional connectivity in APPNL-F/NL-F knock-in mice. Scientific Reports, 2018, 8, 6264.	1.6	41
99	Reduced expression of Na+/Ca2+ exchangers is associated with cognitive deficits seen in Alzheimer's disease model mice. Neuropharmacology, 2018, 131, 291-303.	2.0	23
100	Loss of kallikreinâ€related peptidase 7 exacerbates amyloid pathology in Alzheimer's disease model mice. EMBO Molecular Medicine, 2018, 10, .	3.3	39
101	Endoplasmic reticulum stress responses in mouse models of Alzheimer's disease: Overexpression paradigm versus knockin paradigm. Journal of Biological Chemistry, 2018, 293, 3118-3125.	1.6	53
102	Generation of App knock-in mice reveals deletion mutations protective against Alzheimerâ€™s disease-like pathology. Nature Communications, 2018, 9, 1800.	5.8	33
103	Near-Infrared Photoactivatable Oxygenation Catalysts of Amyloid Peptide. CheM, 2018, 4, 807-820.	5.8	59
104	Reduction in open field activity in the absence of memory deficits in the AppNLâ€'Gâ€'F knock-in mouse model of Alzheimerâ€™s disease. Behavioural Brain Research, 2018, 336, 177-181.	1.2	50
105	Istradefylline reduces memory deficits in aging mice with amyloid pathology. Neurobiology of Disease, 2018, 110, 29-36.	2.1	75
106	T-type calcium channel enhancer SAK3 promotes dopamine and serotonin releases in the hippocampus in naive and amyloid precursor protein knock-in mice. PLoS ONE, 2018, 13, e0206986.	1.1	20
107	Transmission of amyloid-Î² protein pathology from cadaveric pituitary growth hormone. Nature, 2018, 564, 415-419.	13.7	122
108	Neuroinflammation in mouse models of Alzheimer's disease. Clinical and Experimental Neuroimmunology, 2018, 9, 211-218.	0.5	77

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109	The intellectual disability gene PQBP1 rescues Alzheimerâ€™s disease pathology. Molecular Psychiatry, 2018, 23, 2090-2110.	4.1	41
110	Novel Quantitative Analyses of Spontaneous Synaptic Events in Cortical Pyramidal Cells Reveal Subtle Parvalbumin-Expressing Interneuron Dysfunction in a Knock-In Mouse Model of Alzheimerâ€™s Disease. ENeuro, 2018, 5, ENEURO.0059-18.2018.	0.9	18
111	Cognitive and emotional alterations in App knock-in mouse models of AÎ² amyloidosis. BMC Neuroscience, 2018, 19, 46.	0.8	51
112	Concurrent cell typeâ€“specific isolation and profiling of mouse brains in inflammation and Alzheimerâ€™s disease. JCI Insight, 2018, 3, .	2.3	39
113	Circadian and Brain State Modulation of Network Hyperexcitability in Alzheimerâ€™s Disease. ENeuro, 2018, 5, ENEURO.0426-17.2018.	0.9	33
114	PLD3 gene and processing of APP. Nature, 2017, 541, E1-E2.	13.7	42
115	Tetraspanin 6: a pivotal protein of the multiple vesicular body determining exosome release and lysosomal degradation of amyloid precursor protein fragments. Molecular Neurodegeneration, 2017, 12, 25.	4.4	70
116	An immunoaffinity-based method for isolating ultrapure adult astrocytes based on ATP1B2 targeting by the ACSA-2 antibody. Journal of Biological Chemistry, 2017, 292, 8874-8891.	1.6	73
117	<scp>APP</scp> mouse models for Alzheimer's disease preclinical studies. EMBO Journal, 2017, 36, 2473-2487.	3.5	530
118	Comparative profiling of cortical gene expression in Alzheimerâ€™s disease patients and mouse models demonstrates a link between amyloidosis and neuroinflammation. Scientific Reports, 2017, 7, 17762.	1.6	138
119	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
120	Time-course global proteome analyses reveal an inverse correlation between AÎ² burden and immunoglobulin M levels in the APPNL-F mouse model of Alzheimer disease. PLoS ONE, 2017, 12, e0182844.	1.1	6
121	Familial Alzheimerâ€™s Disease Mutations in Presenilin Generate Amyloidogenic AÎ² Peptide Seeds. Neuron, 2016, 90, 410-416.	3.8	86
122	Cognitive deficits in single App knock-in mouse models. Neurobiology of Learning and Memory, 2016, 135, 73-82.	1.0	158
123	Calpain Activation in Alzheimer's Model Mice Is an Artifact of APP and Presenilin Overexpression. Journal of Neuroscience, 2016, 36, 9933-9936.	1.7	98
124	Chronic Neuroinflammation Underlying Pathogenesis of Alzheimerâ€™s Disease. , 2016, , 661-671.		1
125	HMGB1, a pathogenic molecule that induces neurite degeneration via TLR4-MARCKS, is a potential therapeutic target for Alzheimerâ€™s disease. Scientific Reports, 2016, 6, 31895.	1.6	111
126	Bisecting GlcNAc modification stabilizes BACE1 protein under oxidative stress conditions. Biochemical Journal, 2016, 473, 21-30.	1.7	65

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127	Autophagy-Related Protein 7 Deficiency in Amyloid Î² (Aî²) Precursor Protein Transgenic Mice Decreases Aî² in the Multivesicular Bodies and Induces Aî² Accumulation in the Golgi. American Journal of Pathology, 2015, 185, 305-313.	1.9	70
128	An aberrant sugar modification of <scp>BACE</scp>1 blocks its lysosomal targeting in <scp>A</scp>lzheimer's disease. EMBO Molecular Medicine, 2015, 7, 175-189.	3.3	147
129	Loss of GPR3 reduces the amyloid plaque burden and improves memory in Alzheimerâ€™s disease mouse models. Science Translational Medicine, 2015, 7, 309ra164.	5.8	61
130	Neuronal Store-Operated Calcium Entry and Mushroom Spine Loss in Amyloid Precursor Protein Knock-In Mouse Model of Alzheimer's Disease. Journal of Neuroscience, 2015, 35, 13275-13286.	1.7	158
131	ScaleS: an optical clearing palette for biological imaging. Nature Neuroscience, 2015, 18, 1518-1529.	7.1	511
132	New Mouse Model of Alzheimerâ€™s. ACS Chemical Neuroscience, 2014, 5, 499-502.	1.7	70
133	Single App knock-in mouse models of Alzheimer's disease. Nature Neuroscience, 2014, 17, 661-663.	7.1	846
134	Aî² Secretion and Plaque Formation Depend on Autophagy. Cell Reports, 2013, 5, 61-69.	2.9	386
135	Cell Surface Expression of the Major Amyloid-Î² Peptide (Aî²)-degrading Enzyme, Neprilysin, Depends on Phosphorylation by Mitogen-activated Protein Kinase/Extracellular Signal-regulated Kinase Kinase (MEK) and Dephosphorylation by Protein Phosphatase 1a. Journal of Biological Chemistry, 2012, 287, 29362-29372.	1.6	35
136	Potent amyloidogenicity and pathogenicity of Aî²43. Nature Neuroscience, 2011, 14, 1023-1032.	7.1	245
137	Brain Endothelial Cells Produce Amyloid Î² from Amyloid Precursor Protein 770 and Preferentially Secrete the O-Glycosylated Form. Journal of Biological Chemistry, 2010, 285, 40097-40103.	1.6	93
138	Interleukinâ€•1Î² upâ€•regulates TACE to enhance Î±â€•cleavage of APP in neurons: resulting decrease in Aî² production. Journal of Neurochemistry, 2008, 104, 1387-1393.	2.1	89
139	A secreted type of Î²1,6 Nâ€•acetylglucosaminyltransferase V (GnTâ€•V), a novel angiogenesis inducer, is regulated by Î³â€•secretase. FASEB Journal, 2006, 20, 2451-2459.	0.2	27
140	Somatostatin regulates brain amyloid Î² peptide Aî²42 through modulation of proteolytic degradation. Nature Medicine, 2005, 11, 434-439.	15.2	335
141	NFAM1, an immunoreceptor tyrosine-based activation motif-bearing molecule that regulates B cell development and signaling. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 8126-8131.	3.3	93
142	Alzheimer's Disease, Neuropeptides, Neuropeptidase, and Amyloid-Â Peptide Metabolism. Science of Aging Knowledge Environment: SAGE KE, 2003, 2003, 1pe-1.	0.9	41
143	A Secreted Type of Î²1,6-N-Acetylglucosaminyltransferase V (GnT-V) Induces Tumor Angiogenesis without Mediation of Glycosylation. Journal of Biological Chemistry, 2002, 277, 17002-17008.	1.6	77
144	Domain-specific Mutations of a Transforming Growth Factor (TGF)-Î²1 Latency-associated Peptide Cause Camurati-Engelmann Disease Because of the Formation of a Constitutively Active Form of TGF-Î²1. Journal of Biological Chemistry, 2001, 276, 11469-11472.	1.6	89

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145	Domain-specific mutations in TGFB1 result in Camurati-Engelmann disease. Nature Genetics, 2000, 26, 19-20.	9.4	239