Sangram S Sisodia

List of Publications by Year
in descending order

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Version: 2024-02-01

68 papers	15,860 citations	61984 43 h-index	98798 67 g-index
70 all docs	70 docs citations	70 times ranked	12929 citing authors

#	Article	IF	Citations
1	Gut microbiotaâ€“driven brain AÎ² amyloidosis in mice requires microglia. Journal of Experimental Medicine, 2022, 219, .	8.5	44
2	Infection and inflammation: New perspectives on Alzheimer's disease. Brain, Behavior, & Immunity - Health, 2022, 22, 100462.	2.5	17
3	Alteration in synaptic nanoscale organization dictates amyloidogenic processing in Alzheimer's disease. IScience, 2021, 24, 101924.	4.1	13
4	An APP ectodomain mutation outside of the AÎ² domain promotes AÎ² production in vitro and deposition in vivo. Journal of Experimental Medicine, 2021, 218, .	8.5	7
5	Modulation of amyloid deposition and neuroinflammation by the microbiome. Alzheimer's and Dementia, 2020, 16, e044154.	0.8	0
6	Synergistic depletion of gut microbial consortia, but not individual antibiotics, reduces amyloidosis in APPPS1-21 Alzheimerâ€™s transgenic mice. Scientific Reports, 2020, 10, 8183.	3.3	51
7	Negative evidence for a role of APH1B T27I variant in Alzheimerâ€™s disease. Human Molecular Genetics, 2020, 29, 955-966.	2.9	6
8	Deficits in Enrichment-Dependent Neurogenesis and Enhanced Anxiety Behaviors Mediated by Expression of Alzheimer's Disease-Linked Ps1 Variants Are Rescued by Microglial Depletion. Journal of Neuroscience, 2019, 39, 6766-6780.	3.6	22
9	Sex-specific effects of microbiome perturbations on cerebral AÎ² amyloidosis and microglia phenotypes. Journal of Experimental Medicine, 2019, 216, 1542-1560.	8.5	165
10	EC-03-03: GUT MICROBIOME ALTERATIONS IN ALZHEIMER'S DISEASE: PRECLINICAL EVIDENCE. , 2018, 14, P1007-P1007.		0
11	Microglia turnover with aging and in an Alzheimer's model via long-term in vivo single-cell imaging. Nature Neuroscience, 2017, 20, 1371-1376.	14.8	277
12	Antibiotic-induced perturbations in microbial diversity during post-natal development alters amyloid pathology in an aged APPSWE/PS1Î”E9 murine model of Alzheimerâ€™s disease. Scientific Reports, 2017, 7, 10411.	3.3	206
13	Antibiotic-induced perturbations in gut microbial diversity influences neuro-inflammation and amyloidosis in a murine model of Alzheimerâ€™s disease. Scientific Reports, 2016, 6, 30028.	3.3	469
14	Evidence That the â€œLidâ€•Domain of Nicastrin Is Not Essential for Regulating Î³-Secretase Activity. Journal of Biological Chemistry, 2016, 291, 6748-6753.	3.4	6
15	The topology of pen-2, a Î³-secretase subunit, revisited: evidence for a reentrant loop and a single pass transmembrane domain. Molecular Neurodegeneration, 2015, 10, 39.	10.8	14
16	Acne Inversa Caused by Missense Mutations in NCSTN Is Not Fully Compatible with Impairments in Notch Signaling. Journal of Investigative Dermatology, 2015, 135, 618-620.	0.7	28
17	A Synthetic Antibody Fragment Targeting Nicastrin Affects Assembly and Trafficking of Î³-Secretase. Journal of Biological Chemistry, 2014, 289, 34851-34861.	3.4	6
18	Differential Release of Î²-Amyloid from Dendrite- Versus Axon-Targeted APP. Journal of Neuroscience, 2014, 34, 12313-12327.	3.6	38

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19	Soluble Î³-Secretase Modulators Selectively Inhibit the Production of the 42-Amino Acid Amyloid Î² Peptide Variant and Augment the Production of Multiple Carboxy-Truncated Amyloid Î² Species. Biochemistry, 2014, 53, 702-713.	2.5	49
20	Mutant presenilin 1 expression in excitatory neurons impairs enrichment-mediated phenotypes of adult hippocampal progenitor cells. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 9148-9153.	7.1	14
21	Endogenous expression of FAD-linked PS1 impairs proliferation, neuronal differentiation and survival of adult hippocampal progenitors. Molecular Neurodegeneration, 2013, 8, 41.	10.8	15
22	Trafficking and Proteolytic Processing of APP. Cold Spring Harbor Perspectives in Medicine, 2012, 2, a006270-a006270.	6.2	847
23	Identification of a tetratricopeptide repeat-like domain in the nicastrin subunit of Î³-secretase using synthetic antibodies. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 8534-8539.	7.1	32
24	Structure of Î³-Secretase and Its Trimeric Pre-activation Intermediate by Single-particle Electron Microscopy. Journal of Biological Chemistry, 2011, 286, 21440-21449.	3.4	34
25	Amyloid beta from axons and dendrites reduces local spine number and plasticity. Nature Neuroscience, 2010, 13, 190-196.	14.8	295
26	Activation and intrinsic Î³-secretase activity of presenilin 1. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 21435-21440.	7.1	127
27	Modulation of Î³-Secretase Reduces Î²-Amyloid Deposition in a Transgenic Mouse Model of Alzheimer's Disease. Neuron, 2010, 67, 769-780.	8.1	236
28	Non-Cell-Autonomous Effects of Presenilin 1 Variants on Enrichment-Mediated Hippocampal Progenitor Cell Proliferation and Differentiation. Neuron, 2008, 59, 568-580.	8.1	159
29	Expression of a Familial Alzheimer's Disease-Linked Presenilin-1 Variant Enhances Perforant Pathway Lesion-Induced Neuronal Loss in the Entorhinal Cortex. Journal of Neuroscience, 2006, 26, 429-434.	3.6	27
30	A Sequence within the First Transmembrane Domain of PEN-2 Is Critical for PEN-2-mediated Endoproteolysis of Presenilin 1. Journal of Biological Chemistry, 2005, 280, 1992-2001.	3.4	57
31	Evidence That the â€œNFâ€•Motif in Transmembrane Domain 4 of Presenilin 1 Is Critical for Binding with PEN-2. Journal of Biological Chemistry, 2005, 280, 41953-41966.	3.4	70
32	The Notch Ligands, Delta1 and Jagged2, Are Substrates for Presenilin-dependent â€œÎ³-Secretaseâ€•Cleavage. Journal of Biological Chemistry, 2003, 278, 7751-7754.	3.4	183
33	APP Processing and Synaptic Function. Neuron, 2003, 37, 925-937.	8.1	1,423
34	Regulated Hyperaccumulation of Presenilin-1 and the â€œÎ³-Secretaseâ€•Complex. Journal of Biological Chemistry, 2003, 278, 33992-34002.	3.4	94
35	Evidence That Synaptically Released Î²-Amyloid Accumulates as Extracellular Deposits in the Hippocampus of Transgenic Mice. Journal of Neuroscience, 2002, 22, 9785-9793.	3.6	281
36	Î³-Secretase, notch, AÎ² and alzheimer's disease: Where do the presenilins fit in?. Nature Reviews Neuroscience, 2002, 3, 281-290.	10.2	494

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37	Deficient Neurogenesis in Forebrain-Specific Presenilin-1 Knockout Mice Is Associated with Reduced Clearance of Hippocampal Memory Traces. Neuron, 2001, 32, 911-926.	8.1	443
38	Requirement for Presenilin 1 in Facilitating Jagged 2-Mediated Endoproteolysis and Signaling of Notch 1. Journal of Molecular Neuroscience, 2001, 15, 189-204.	2.3	46
39	Alzheimer's Disease: Clinical, Biological, and Therapeutic Perspectives. Journal of Molecular Neuroscience, 2001, 17, 99-99.	2.3	4
40	Characterization of a Presenilin-mediated Amyloid Precursor Protein Carboxyl-terminal Fragment Î³. Journal of Biological Chemistry, 2001, 276, 43756-43760.	3.4	188
41	Multiple Effects of Aspartate Mutant Presenilin 1 on the Processing and Trafficking of Amyloid Precursor Protein. Journal of Biological Chemistry, 2001, 276, 43343-43350.	3.4	87
42	The Value of Transgenic Models for the Study of Neurodegenerative Diseases. Annals of the New York Academy of Sciences, 2000, 920, 179-191.	3.8	51
43	Furin mediates enhanced production of fibrillogenic ABri peptides in familial British dementia. Nature Neuroscience, 1999, 2, 984-988.	14.8	146
44	Transgenic Mouse Models of Alzheimer's Disease and Amyotrophic Lateral Sclerosis. Brain Pathology, 1998, 8, 735-757.	4.1	27
45	ALZHEIMER'S DISEASE: Genetic Studies and Transgenic Models. Annual Review of Genetics, 1998, 32, 461-493.	7.6	384
46	MUTANT GENES IN FAMILIAL ALZHEIMER'S DISEASE AND TRANSGENIC MODELS. Annual Review of Neuroscience, 1998, 21, 479-505.	10.7	572
47	Post-translational Processing and Turnover Kinetics of Presynaptically Targeted Amyloid Precursor Superfamily Proteins in the Central Nervous System. Journal of Biological Chemistry, 1998, 273, 11100-11106.	3.4	69
48	Alzheimer Amyloid Protein Precursor in the Rat Hippocampus: Transport and Processing through the Perforant Path. Journal of Neuroscience, 1998, 18, 9629-9637.	3.6	249
49	Evidence That Levels of Presenilins (PS1 and PS2) Are Coordinately Regulated by Competition for Limiting Cellular Factors. Journal of Biological Chemistry, 1997, 272, 28415-28422.	3.4	302
50	Ectodomain Phosphorylation of Î²-Amyloid Precursor Protein at Two Distinct Cellular Locations. Journal of Biological Chemistry, 1997, 272, 1896-1903.	3.4	69
51	Altered metabolism of familial Alzheimer's disease-linked amyloid precursor protein variants in yeast artificial chromosome transgenic mice. Human Molecular Genetics, 1997, 6, 1535-1541.	2.9	117
52	Amyloid $sZ-protein stimulates parallel increases in cellular levels of its precursor and amyloid precursor-like protein 2 (APLP2) in human cerebrovascular smooth muscle cells. Amyloid: the International Journal of Experimental and Clinical Investigation: the Official Journal of the International Society of Amyloidosis, 1997, 4, 54-60.	3.0	4
53	Accelerated Amyloid Deposition in the Brains of Transgenic Mice Coexpressing Mutant Presenilin 1 and Amyloid Precursor Proteins. Neuron, 1997, 19, 939-945.	8.1	964
54	Hyperaccumulation of FAD-linked presenilin 1 variants in vivo. Nature Medicine, 1997, 3, 756-760.	30.7	140

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55	Presenilin 1 is required for Notch 1 and Dll1 expression in the paraxial mesoderm. Nature, 1997, 387, 288-292.	27.8	730
56	Familial Alzheimer's Diseaseâ€“Linked Presenilin 1 Variants Elevate AÎ²1â€“42/1â€“40 Ratio In Vitro and In Vivo. Neuron, 1996, 17, 1005-1013.	8.1	1,471
57	Endoproteolysis of Presenilin 1 and Accumulation of Processed Derivatives In Vivo. Neuron, 1996, 17, 181-190.	8.1	1,054
58	Inherited Neurodegenerative Diseases and Transgenic Models. Brain Pathology, 1996, 6, 467-480.	4.1	9
59	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
60	Metabolism of the â€œSwedishâ€•Amyloid Precursor Protein Variant in Neuro2a (N2a) Cells. Journal of Biological Chemistry, 1996, 271, 9390-9397.	3.4	286
61	Motor Neuron Disease and Model Systems: Aetiologies, Mechanisms and Therapies. Novartis Foundation Symposium, 1996, 196, 3-17.	1.1	1
62	A mouse model for Down syndrome exhibits learning and behaviour deficits. Nature Genetics, 1995, 11, 177-184.	21.4	854
63	Role of the Î²â€amyloid protein in Alzheimer's disease. FASEB Journal, 1995, 9, 366-370.	0.5	259
64	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.	3.0	18
65	Î²-amyloid precursor protein-deficient mice show reactive gliosis and decreased locomotor activity. Cell, 1995, 81, 525-531.	28.9	648
66	CELLULAR AND MOLECULAR BIOLOGY OF ALZHEIMER'S DISEASE AND ANIMAL MODELS. Annual Review of Medicine, 1994, 45, 435-446.	12.2	127
67	Isolation and characterization of APLP2 encoding a homologue of the Alzheimer's associated amyloid Î² protein precursor. Nature Genetics, 1993, 5, 95-100.	21.4	370
68	Neuronal degeneration in human diseases and animal models. Journal of Neurobiology, 1992, 23, 1277-1294.	3.6	34