Gopal Thinakaran

List of Publications by Year
in descending order

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104 papers	14,256 citations	26630 56 h-index	33894 99 g-index
105 all docs	105 docs citations	105 times ranked	11778 citing authors

#	Article	IF	Citations
1	Cinnabarinic Acid-Induced Stanniocalcin 2 Confers Cytoprotection against Alcohol-Induced Liver Injury. Journal of Pharmacology and Experimental Therapeutics, 2022, 381, 1-11.	2.5	8
2	Enhanced cleavage of APP by co-expressed Bace1 alters the distribution of APP and its fragments in neuronal and non-neuronal cells. Molecular Neurobiology, 2022, 59, 3073-3090.	4.0	8
3	BIN1 is a key regulator of proinflammatory and neurodegeneration-related activation in microglia. Molecular Neurodegeneration, 2022, 17, 33.	10.8	26
4	Preclinical validation of a potent Î³-secretase modulator for Alzheimerâ€™s disease prevention. Journal of Experimental Medicine, 2021, 218, .	8.5	39
5	Neuronal BIN1 Regulates Presynaptic Neurotransmitter Release and Memory Consolidation. Cell Reports, 2020, 30, 3520-3535.e7.	6.4	59
6	Reduction of the expression of the late-onset Alzheimerâ€™s disease (AD) risk-factor BIN1 does not affect amyloid pathology in an AD mouse model. Journal of Biological Chemistry, 2019, 294, 4477-4487.	3.4	33
7	APP-Mediated Signaling Prevents Memory Decline in Alzheimerâ€™s Disease Mouse Model. Cell Reports, 2019, 27, 1345-1355.e6.	6.4	20
8	Matrix metalloproteinase 13, a new target for therapy in Alzheimer's disease. Genes and Diseases, 2019, 6, 1-2.	3.4	7
9	Aberrant accrual of BIN1 near Alzheimerâ€™s disease amyloid deposits in transgenic models. Brain Pathology, 2019, 29, 485-501.	4.1	25
10	Insulin-Like Growth Factor-II/Cation-Independent Mannose 6-Phosphate Receptor in Neurodegenerative Diseases. Molecular Neurobiology, 2017, 54, 2636-2658.	4.0	41
11	Lack of BACE1 S-palmitoylation reduces amyloid burden and mitigates memory deficits in transgenic mouse models of Alzheimerâ€™s disease. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E9665-E9674.	7.1	51
12	BIN1 localization is distinct from Tau tangles in Alzheimerâ€™s disease. Matters, 2017, 2017, .	1.0	13
13	Presynaptic dystrophic neurites surrounding amyloid plaques are sites of microtubule disruption, BACE1 elevation, and increased Aî² generation in Alzheimerâ€™s disease. Acta Neuropathologica, 2016, 132, 235-256.	7.7	193
14	A Greek Tragedy: The Growing Complexity of Alzheimer Amyloid Precursor Protein Proteolysis. Journal of Biological Chemistry, 2016, 291, 19235-19244.	3.4	151
15	Predominant expression of Alzheimerâ€™s disease-associated BIN1 in mature oligodendrocytes and localization to white matter tracts. Molecular Neurodegeneration, 2016, 11, 59.	10.8	95
16	APP Receptor? To Be or Not To Be. Trends in Pharmacological Sciences, 2016, 37, 390-411.	8.7	107
17	Significance of transcytosis in Alzheimer's disease: BACE1 takes the scenic route to axons. BioEssays, 2015, 37, 888-898.	2.5	12
18	Physiologically generated presenilin 1 lacking exon 8 fails to rescue brain PS1âˆ’/âˆ’ phenotype and forms complexes with wildtype PS1 and nicastrin. Scientific Reports, 2015, 5, 17042.	3.3	4

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19	APLP2 Regulates Refractive Error and Myopia Development in Mice and Humans. PLoS Genetics, 2015, 11, e1005432.	3.5	77
20	Rheb GTPase Regulates Î²-Secretase Levels and Amyloid Î² Generation. Journal of Biological Chemistry, 2014, 289, 5799-5808.	3.4	49
21	Sorting the Role of SORLA in Alzheimerâ€™s Disease. Science Translational Medicine, 2014, 6, 223fs8.	12.4	10
22	Axonal BACE1 dynamics and targeting in hippocampal neurons: a role for Rab11 GTPase. Molecular Neurodegeneration, 2014, 9, 1.	10.8	130
23	Increasing membrane cholesterol of neurons in culture recapitulates Alzheimerâ€™s disease early phenotypes. Molecular Neurodegeneration, 2014, 9, 60.	10.8	76
24	Overexpression of the IGF-II/M6P Receptor in Mouse Fibroblast Cell Lines Differentially Alters Expression Profiles of Genes Involved in Alzheimerâ€™s Disease-Related Pathology. PLoS ONE, 2014, 9, e98057.	2.5	5
25	A Paired RNAi and RabGAP Overexpression Screen Identifies Rab11 as a Regulator of Î²-Amyloid Production. Cell Reports, 2013, 5, 1536-1551.	6.4	120
26	A Function for EHD Family Proteins in Unidirectional Retrograde Dendritic Transport of BACE1 and Alzheimerâ€™s Disease AÎ² Production. Cell Reports, 2013, 5, 1552-1563.	6.4	65
27	Ca2+ Influx through Store-operated Ca2+ Channels Reduces Alzheimer Disease Î²-Amyloid Peptide Secretion. Journal of Biological Chemistry, 2013, 288, 26955-26966.	3.4	35
28	Alterations in Gene Expression in Mutant Amyloid Precursor Protein Transgenic Mice Lacking Niemann-Pick Type C1 Protein. PLoS ONE, 2013, 8, e54605.	2.5	6
29	Trafficking and Proteolytic Processing of APP. Cold Spring Harbor Perspectives in Medicine, 2012, 2, a006270-a006270.	6.2	847
30	Novel GÎ±_S-Protein Signaling Associated with Membrane-Tethered Amyloid Precursor Protein Intracellular Domain. Journal of Neuroscience, 2012, 32, 1714-1729.	3.6	42
31	Differential Regulation of Amyloid Precursor Protein/Presenilin 1 Interaction during Ab40/42 Production Detected Using Fusion Constructs. PLoS ONE, 2012, 7, e48551.	2.5	4
32	Stringently regulated p23 expression is critical for coordinated movement in mice: implications for Alzheimerâ€™s disease. Molecular Neurodegeneration, 2012, 7, L2.	10.8	1
33	Transgenic neuronal overexpression reveals that stringently regulated p23 expression is critical for coordinated movement in mice. Molecular Neurodegeneration, 2011, 6, 87.	10.8	19
34	Stanniocalcin 2 Is a Negative Modulator of Store-Operated Calcium Entry. Molecular and Cellular Biology, 2011, 31, 3710-3722.	2.3	62
35	Loss of Cleavage at Î²â€²-Site Contributes to Apparent Increase in Î²-Amyloid Peptide (AÎ²) Secretion by Î²-Secretase (BACE1)-Glycosylphosphatidylinositol (GPI) Processing of Amyloid Precursor Protein. Journal of Biological Chemistry, 2011, 286, 26166-26177.	3.4	30
36	Modeling Presenilin-Dependent Familial Alzheimer's Disease: Emphasis on Presenilin Substrate-Mediated Signaling and Synaptic Function. International Journal of Alzheimer's Disease, 2010, 2010, 1-11.	2.0	25

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37	Mutation Analysis of the Presenilin 1 N-terminal Domain Reveals a Broad Spectrum of Î³-Secretase Activity toward Amyloid Precursor Protein and Other Substrates. Journal of Biological Chemistry, 2010, 285, 38042-38052.	3.4	28
38	Reduced Alzheimer's Disease Î²-Amyloid Deposition in Transgenic Mice ExpressingS-Palmitoylation-Deficient APH1aL and Nicastrin. Journal of Neuroscience, 2010, 30, 16160-16169.	3.6	37
39	Membrane rafts in Alzheimer's disease beta-amyloid production. Biochimica Et Biophysica Acta - Molecular and Cell Biology of Lipids, 2010, 1801, 860-867.	2.4	240
40	Psychosine Accumulates in Membrane Microdomains in the Brain of Krabbe Patients, Disrupting the Raft Architecture. Journal of Neuroscience, 2009, 29, 6068-6077.	3.6	140
41	S-Palmitoylation of Î³-Secretase Subunits Nicastrin and APH-1. Journal of Biological Chemistry, 2009, 284, 1373-1384.	3.4	61
42	Alzheimer Disease AÎ² Production in the Absence of S-Palmitoylation-dependent Targeting of BACE1 to Lipid Rafts. Journal of Biological Chemistry, 2009, 284, 3793-3803.	3.4	137
43	Steadyâ€state increase of cAMPâ€response element binding protein, Rac, and PAK signaling in presenilinâ€deficient neurons. Journal of Neurochemistry, 2008, 104, 1637-1648.	3.9	9
44	Localization and regional distribution of p23/TMP21 in the brain. Neurobiology of Disease, 2008, 32, 37-49.	4.4	27
45	Amyloid Precursor Protein Trafficking, Processing, and Function. Journal of Biological Chemistry, 2008, 283, 29615-29619.	3.4	906
46	Evidence That CD147 Modulation of Î²-Amyloid (AÎ²) Levels Is Mediated by Extracellular Degradation of Secreted AÎ². Journal of Biological Chemistry, 2008, 283, 19489-19498.	3.4	46
47	Thematic Minireview Series on the Molecular Basis of Alzheimer Disease. Journal of Biological Chemistry, 2008, 283, 29613-29614.	3.4	3
48	Biogenesis of Î³-secretase early in the secretory pathway. Journal of Cell Biology, 2007, 179, 951-963.	5.2	62
49	Dual roles of the transmembrane protein p23/TMP21 in the modulation of amyloid precursor protein metabolism. Molecular Neurodegeneration, 2007, 2, 4.	10.8	68
50	Amyloidogenic processing of Î²-amyloid precursor protein in intracellular compartments. Neurology, 2006, 66, S69-73.	1.1	216
51	Pathological and physiological functions of presenilins. Molecular Neurodegeneration, 2006, 1, 4.	10.8	124
52	Presenilins and Alzheimer disease: the calcium conspiracy. Nature Neuroscience, 2006, 9, 1354-1355.	14.8	26
53	Spatial Segregation of Î³-Secretase and Substrates in DistinctMembraneDomains. Journal of Biological Chemistry, 2005, 280, 25892-25900.	3.4	203
54	Î³-Secretase Is a Functional Component of Phagosomes. Journal of Biological Chemistry, 2005, 280, 36310-36317.	3.4	32

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55	Presenilin Attenuates Receptor-Mediated Signaling and Synaptic Function. Journal of Neuroscience, 2005, 25, 1540-1549.	3.6	72
56	Nicastrin Is Critical for Stability and Trafficking but Not Association of Other Presenilin/Î³-Secretase Components. Journal of Biological Chemistry, 2005, 280, 17020-17026.	3.4	105
57	Characterization of Stanniocalcin 2, a Novel Target of the Mammalian Unfolded Protein Response with Cytoprotective Properties. Molecular and Cellular Biology, 2004, 24, 9456-9469.	2.3	166
58	Presenilins and Î³-Secretase Inhibitors Affect Intracellular Trafficking and Cell Surface Localization of the Î³-Secretase Complex Components. Journal of Biological Chemistry, 2004, 279, 40560-40566.	3.4	42
59	Association of Î³-Secretase with Lipid Rafts in Post-Golgi and Endosome Membranes. Journal of Biological Chemistry, 2004, 279, 44945-44954.	3.4	372
60	Identification of the role of presenilins beyond Alzheimer?s disease. Pharmacological Research, 2004, 50, 411-418.	7.1	58
61	The role of presenilin cofactors in the Î³-secretase complex. Nature, 2003, 422, 438-441.	27.8	839
62	Presenilin-1 Regulates Intracellular Trafficking and Cell Surface Delivery of Î²-Amyloid Precursor Protein. Journal of Biological Chemistry, 2003, 278, 3446-3454.	3.4	123
63	PEN-2 and APH-1 Coordinately Regulate Proteolytic Processing of Presenilin 1. Journal of Biological Chemistry, 2003, 278, 7850-7854.	3.4	202
64	Investigation of Unfolded-Protein Response in Cells Expressing Familial Alzheimer's Disease-Linked Presenilin Variants. , 2003, 232, 203-216.		1
65	Presenilin 1 Is Required for Maturation and Cell Surface Accumulation of Nicastrin. Journal of Biological Chemistry, 2002, 277, 19236-19240.	3.4	166
66	Proteolytic Processing of Familial British Dementia-associated BRI Variants. Journal of Biological Chemistry, 2002, 277, 1872-1877.	3.4	53
67	A Role for Presenilin 1 in Regulating the Delivery of Amyloid Precursor Protein to the Cell Surface. Neurobiology of Disease, 2002, 11, 64-82.	4.4	65
68	Metabolism of Presenilins<. Journal of Molecular Neuroscience, 2001, 17, 183-192.	2.3	13
69	Endoplasmic Reticulum Stress-induced Cysteine Protease Activation in Cortical Neurons. Journal of Biological Chemistry, 2001, 276, 44736-44743.	3.4	89
70	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
71	Lessons from presenilin domain analysis: endoproteolytic processing and enhanced AÎ²42 production mediated by FAD-linked variants. , 2001, , 167-175.		0
72	Determining the Transmembrane Topology of the Presenilins. , 2000, 32, 283-296.		0

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73	Upregulation of BiP and CHOP by the unfolded-protein response is independent of presenilin expression. Nature Cell Biology, 2000, 2, 863-870.	10.3	136
74	The Nonconserved Hydrophilic Loop Domain of Presenilin (PS) Is Not Required for PS Endoproteolysis or Enhanced AÎ²42 Production Mediated by Familial Early Onset Alzheimer's Disease-linked PS Variants. Journal of Biological Chemistry, 2000, 275, 17136-17142.	3.4	61
75	Subcellular Localization of Presenilins: Association with a Unique Membrane Pool in Cultured Cells. Neurobiology of Disease, 2000, 7, 99-117.	4.4	54
76	Amyloid Precursor Proteins Inhibit Heme Oxygenase Activity and Augment Neurotoxicity in Alzheimer's Disease. Neuron, 2000, 28, 461-473.	8.1	168
77	Familial British Dementia: Expression and Metabolism of BRI. Annals of the New York Academy of Sciences, 2000, 920, 93-99.	3.8	18
78	Evidence That Intramolecular Associations between Presenilin Domains Are Obligatory for Endoproteolytic Processing. Journal of Biological Chemistry, 1999, 274, 13818-13823.	3.4	69
79	Amyloid Precursor-like Protein 2 Promotes Cell Migration toward Fibronectin and Collagen IV. Journal of Biological Chemistry, 1999, 274, 27249-27256.	3.4	31
80	Furin mediates enhanced production of fibrillogenic ABri peptides in familial British dementia. Nature Neuroscience, 1999, 2, 984-988.	14.8	146
81	Function and Dysfunction of the Presenilins. American Journal of Human Genetics, 1999, 65, 7-12.	6.2	73
82	The role of presenilins in Alzheimerâ€™s disease. Journal of Clinical Investigation, 1999, 104, 1321-1327.	8.2	59
83	Estrogen reduces neuronal generation of Alzheimer Î²-amyloid peptides. Nature Medicine, 1998, 4, 447-451.	30.7	545
84	Immunohistochemical and in situ analysis of amyloid precursor-like protein-1 and amyloid precursor-like protein-2 expression in Alzheimer disease and aged control brains. Brain Research, 1998, 804, 45-51.	2.2	39
85	Effects of PS1 Deficiency on Membrane Protein Trafficking in Neurons. Neuron, 1998, 21, 1213-1221.	8.1	359
86	Stable Association of Presenilin Derivatives and Absence of Presenilin Interactions with APP. Neurobiology of Disease, 1998, 4, 438-453.	4.4	187
87	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.	4.4	96
88	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
89	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
90	Familial Amyotrophic Lateral Sclerosis and Alzheimerâ€™s Disease. Advances in Experimental Medicine and Biology, 1998, , 145-159.	1.6	7

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91	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
92	Endoproteolytic Processing and Stabilization of Wild-type and Mutant Presenilin. Journal of Biological Chemistry, 1997, 272, 24536-24541.	3.4	190
93	Ectodomain Phosphorylation of Î²-Amyloid Precursor Protein at Two Distinct Cellular Locations. Journal of Biological Chemistry, 1997, 272, 1896-1903.	3.4	69
94	Processing of presenilin 1 in brains of patients with AlzheimerÊ¼s disease and controls. NeuroReport, 1997, 8, 1717-1721.	1.2	31
95	Identification of Candidate Proteins Binding to Prion Protein. Neurobiology of Disease, 1997, 3, 339-355.	4.4	111
96	Hyperaccumulation of FAD-linked presenilin 1 variants in vivo. Nature Medicine, 1997, 3, 756-760.	30.7	140
97	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
98	Protein Topology of Presenilin 1. Neuron, 1996, 17, 1023-1030.	8.1	381
99	Endoproteolysis of Presenilin 1 and Accumulation of Processed Derivatives In Vivo. Neuron, 1996, 17, 181-190.	8.1	1,054
100	Expression of Presenilin 1 and 2 (PS1 and PS2) in Human and Murine Tissues. Journal of Neuroscience, 1996, 16, 7513-7525.	3.6	279
101	Comparative evaluation of synaptophysin-based methods for quantification of synapses. Journal of Neurocytology, 1996, 25, 821-828.	1.5	261
102	Metabolism of the â€œSwedishâ€•Amyloid Precursor Protein Variant in Neuro2a (N2a) Cells. Journal of Biological Chemistry, 1996, 271, 9390-9397.	3.4	286
103	The Unfolded Protein Response-mediated Upregulation of BiP and CHOP Is not Affected by Presenilin Expression. , 0, , 559-567.		0
104	APP Biology, Processing and Function. , 0, , 17-34.		2