Gunnar K Gouras

List of Publications by Year
in descending order

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108	11,254	57631	35952
papers	citations	44 h-index	97 g-index

133	133	133	11451
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Monitoring the interactions between alpha-synuclein and Tau in vitro and in vivo using bimolecular fluorescence complementation. Scientific Reports, 2022, 12, 2987.	1.6	10
2	Parkinsonâ€™s disease and multiple system atrophy patient iPSC-derived oligodendrocytes exhibit alpha-synucleinâ€“induced changes in maturation and immune reactive properties. Proceedings of the National Academy of Sciences of the United States of America, 2022, 119, e2111405119.	3.3	22
3	Correlative imaging to resolve molecular structures in individual cells: Substrate validation study for super-resolution infrared microspectroscopy. Nanomedicine: Nanotechnology, Biology, and Medicine, 2022, 43, 102563.	1.7	6
4	Sphingosine 1-Phoshpate Receptors are Located in Synapses and Control Spontaneous Activity of Mouse Neurons in Culture. Neurochemical Research, 2022, 47, 3114-3125.	1.6	5
5	Accumulation of cellular prion protein within Î²â€amyloid oligomer plaques in aged human brains. Brain Pathology, 2021, 31, e12941.	2.1	17
6	Amyloid Structural Changes Studied by Infrared Microspectroscopy in Bigenic Cellular Models of Alzheimerâ€™s Disease. International Journal of Molecular Sciences, 2021, 22, 3430.	1.8	4
7	Upregulation of APP endocytosis by neuronal aging drives amyloid-dependent synapse loss. Journal of Cell Science, 2021, 134, .	1.2	29
8	Differential seeding and propagating efficiency of Î±-synuclein strains generated in different conditions. Translational Neurodegeneration, 2021, 10, 20.	3.6	11
9	Correlative optical photothermal infrared and X-ray fluorescence for chemical imaging of trace elements and relevant molecular structures directly in neurons. Light: Science and Applications, 2021, 10, 151.	7.7	24
10	Neuronal spreading and plaque induction of intracellular AÎ² and its disruption of AÎ² homeostasis. Acta Neuropathologica, 2021, 142, 669-687.	3.9	17
11	Neuronal Î±â€amylase is important for neuronal activity and glycogenolysis and reduces in presence of amyloid beta pathology. Aging Cell, 2021, 20, e13433.	3.0	7
12	FRET-Based Screening Identifies p38 MAPK and PKC Inhibition as Targets for Prevention of Seeded Î±-Synuclein Aggregation. Neurotherapeutics, 2021, 18, 1692-1709.	2.1	6
13	DNAJB6b is Downregulated in Synucleinopathies. Journal of Parkinson's Disease, 2021, 11, 1-13.	1.5	0
14	In Memoriam for M. Flint Beal. Journal of Alzheimer's Disease, 2021, 83, 1-2.	1.2	1
15	Astrocytic and Neuronal Apolipoprotein E Isoforms Differentially Affect Neuronal Excitability. Frontiers in Neuroscience, 2021, 15, 734001.	1.4	21
16	Nano-Infrared Imaging of Primary Neurons. Cells, 2021, 10, 2559.	1.8	14
17	APOE4 Affects Basal and NMDAR-Mediated Protein Synthesis in Neurons by Perturbing Calcium Homeostasis. Journal of Neuroscience, 2021, 41, 8686-8709.	1.7	16
18	Studies on ApoE in the neurobiology of Alzheimerâ€™s disease. Alzheimer's and Dementia, 2020, 16, e038963.	0.4	0

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19	Synapse disruption by Alzheimerâ€™s AÎ²/APP. Alzheimer's and Dementia, 2020, 16, e043447.	0.4	0
20	Human iPSC-Derived Hippocampal Spheroids: An Innovative Tool for Stratifying Alzheimer Disease Patient-Specific Cellular Phenotypes and Developing Therapies. Stem Cell Reports, 2020, 15, 256-273.	2.3	49
21	Superâ€Resolution Infrared Imaging of Polymorphic Amyloid Aggregates Directly in Neurons. Advanced Science, 2020, 7, 1903004.	5.6	71
22	Aging, Metabolism, Synaptic Activity, and AÎ² in Alzheimer's Disease. Frontiers in Aging Neuroscience, 2019, 11, 185.	1.7	6
23	Poly(propylene imine) dendrimers with histidine-maltose shell as novel type of nanoparticles for synapse and memory protection. Nanomedicine: Nanotechnology, Biology, and Medicine, 2019, 17, 198-209.	1.7	75
24	APP depletion alters selective pre- and post-synaptic proteins. Molecular and Cellular Neurosciences, 2019, 95, 86-95.	1.0	26
25	Prion-like seeding and nucleation of intracellular amyloid-Î². Neurobiology of Disease, 2018, 113, 1-10.	2.1	60
26	Nano-scale Infrared Imaging Of Î²-sheet Structures In Synaptic Junctions Of Primary Neurons Isolated From Transgenic Mice. , 2018, , .		0
27	S100A9-Driven Amyloid-Neuroinflammatory Cascade in Traumatic Brain Injury as a Precursor State for Alzheimerâ€™s Disease. Scientific Reports, 2018, 8, 12836.	1.6	38
28	Plaque formation and the intraneuronal accumulation of Î²â€amyloid in Alzheimer's disease. Pathology International, 2017, 67, 185-193.	0.6	237
29	Pre-plaque conformational changes in Alzheimerâ€™s disease-linked AÎ² and APP. Nature Communications, 2017, 8, 14726.	5.8	74
30	Heterogeneous Association of Alzheimerâ€™s Disease-Linked Amyloid-Î² and Amyloid-Î² Protein Precursor with Synapses. Journal of Alzheimer's Disease, 2017, 60, 511-524.	1.2	20
31	AÎ² accumulation causes MVB enlargement and is modelled by dominant negative VPS4A. Molecular Neurodegeneration, 2017, 12, 61.	4.4	63
32	Dysregulation of Elongation Factor 1A Expression is Correlated with Synaptic Plasticity Impairments in Alzheimerâ€™s Disease. Journal of Alzheimer's Disease, 2016, 54, 669-678.	1.2	17
33	Brain activity and Alzheimerâ€™s disease: a complex relationship. Brain, 2016, 139, 2109-2110.	3.7	3
34	Direct High Affinity Interaction between AÎ²42 and GSK3Î± Stimulates Hyperphosphorylation of Tau. A New Molecular Link in Alzheimerâ€™s Disease?. ACS Chemical Neuroscience, 2016, 7, 161-170.	1.7	40
35	ADAM10 and BACE1 are localized to synaptic vesicles. Journal of Neurochemistry, 2015, 135, 606-615.	2.1	65
36	P1-065: Adam10 and bace1 are localized to synaptic vesicles. , 2015, 11, P363-P364.		0

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37	ESCRTs regulate amyloid precursor protein sorting in multivesicular bodies and intracellular beta amyloid accumulation. Journal of Cell Science, 2015, 128, 2520-8.	1.2	60
38	Î²-amyloid Peptides and Amyloid Plaques in Alzheimer's Disease. Neurotherapeutics, 2015, 12, 3-11.	2.1	195
39	P3-070: DETECTION OF PRE-PLAQUE AMYLOID AGGREGATION USING FTIR. , 2014, 10, P652-P652.		0
40	Lesion of the subiculum reduces the spread of amyloid beta pathology to interconnected brain regions in a mouse model of Alzheimerâ€™s disease. Acta Neuropathologica Communications, 2014, 2, 17.	2.4	17
41	Activity-independent release of the amyloid Î²-peptide from rat brain nerve terminals. Neuroscience Letters, 2014, 566, 125-130.	1.0	16
42	The Inside-Out Amyloid Hypothesis and Synapse Pathology in Alzheimer's Disease. Neurodegenerative Diseases, 2014, 13, 142-146.	0.8	26
43	P1-060: DISTINCT LOCALIZATION OF BETA- AND GAMMA-SECRETASE IN RAT BRAIN SYNAPSES. , 2014, 10, P325-P325.		0
44	O3-05-03: SYNAPTIC ALTERATIONS IN APP KNOCKOUT NEURONS. , 2014, 10, P217-P217.		0
45	P1-088: MODULATION OF BETA-AMYLOID/APP IN ENDOSOMES. , 2014, 10, P334-P334.		0
46	<scp>mTOR</scp>: at the crossroads of aging, chaperones, and <scp>A</scp>lzheimer's disease. Journal of Neurochemistry, 2013, 124, 747-748.	2.1	13
47	Nonsteroidal Selective Androgen Receptor Modulators and Selective Estrogen Receptor Î² Agonists Moderate Cognitive Deficits and Amyloid-Î² Levels in a Mouse Model of Alzheimerâ€™s Disease. ACS Chemical Neuroscience, 2013, 4, 1537-1548.	1.7	50
48	Convergence of Synapses, Endosomes, and Prions in the Biology of Neurodegenerative Diseases. International Journal of Cell Biology, 2013, 2013, 1-6.	1.0	16
49	Accumulation of Intraneuronal Î²-Amyloid 42 Peptides Is Associated with Early Changes in Microtubule-Associated Protein 2 in Neurites and Synapses. PLoS ONE, 2013, 8, e51965.	1.1	48
50	Pathology of Synapses and Dendritic Spines. Neural Plasticity, 2012, 2012, 1-2.	1.0	3
51	Intraneuronal AÃŸ Accumulation, Amyloid Plaques, and Synapse Pathology in AlzheimerÂ's Disease. Neurodegenerative Diseases, 2012, 10, 56-59.	0.8	21
52	Critical role of intraneuronal AÎ² in Alzheimer's disease: Technical challenges in studying intracellular AÎ². Life Sciences, 2012, 91, 1153-1158.	2.0	36
53	Analysis of Vesicular Trafficking in Primary Neurons by Live Imaging. Methods in Molecular Biology, 2011, 793, 343-350.	0.4	4
54	High-Resolution 3D Reconstruction Reveals Intra-Synaptic Amyloid Fibrils. American Journal of Pathology, 2011, 179, 2551-2558.	1.9	27

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55	Coenzyme Q10 Decreases Amyloid Pathology and Improves Behavior in a Transgenic Mouse Model of Alzheimer's Disease. Journal of Alzheimer's Disease, 2011, 27, 211-223.	1.2	127
56	Accumulation of cellular prion protein within dystrophic neurites of amyloid plaques in the Alzheimer's disease brain. Neuropathology, 2011, 31, 208-214.	0.7	29
57	Impaired Î²-Amyloid Secretion in Alzheimer's Disease Pathogenesis. Journal of Neuroscience, 2011, 31, 15384-15390.	1.7	35
58	Degradation of Alzheimer's amyloid fibrils by microglia requires delivery of ClC-7 to lysosomes. Molecular Biology of the Cell, 2011, 22, 1664-1676.	0.9	86
59	Intraneuronal Î²-amyloid accumulation and synapse pathology in Alzheimerâ€™s disease. Acta Neuropathologica, 2010, 119, 523-541.	3.9	341
60	Synapses, synaptic activity and intraneuronal AÎ² in Alzheimerâ€™s disease. Frontiers in Aging Neuroscience, 2010, 2, .	1.7	40
61	Dysregulation of the mTOR Pathway Mediates Impairment of Synaptic Plasticity in a Mouse Model of Alzheimer's Disease. PLoS ONE, 2010, 5, e12845.	1.1	219
62	Effects of Synaptic Modulation on Î²-Amyloid, Synaptophysin, and Memory Performance in Alzheimer's Disease Transgenic Mice. Journal of Neuroscience, 2010, 30, 14299-14304.	1.7	125
63	Co-occurrence of Alzheimer's disease Î²-amyloid and tau pathologies at synapses. Neurobiology of Aging, 2010, 31, 1145-1152.	1.5	116
64	Immunotherapy for Alzheimer disease. MAbs, 2009, 1, 112-114.	2.6	2
65	Synaptic Activity Reduces Intraneuronal AÎ², Promotes APP Transport to Synapses, and Protects against AÎ²-Related Synaptic Alterations. Journal of Neuroscience, 2009, 29, 9704-9713.	1.7	119
66	Triterpenoid CDDOâ€methylamide improves memory and decreases amyloid plaques in a transgenic mouse model of Alzheimerâ€™s disease. Journal of Neurochemistry, 2009, 109, 502-512.	2.1	99
67	Optical visualization of Alzheimerâ€™s pathology via multiphoton-excited intrinsic fluorescence and second harmonic generation. Optics Express, 2009, 17, 3679.	1.7	94
68	Î²-Amyloid Modulation of Synaptic Transmission and Plasticity. Journal of Neuroscience, 2007, 27, 11832-11837.	1.7	107
69	Alzheimerâ€™s disease therapy: focus on synapses. Future Neurology, 2007, 2, 469-470.	0.9	0
70	Internalized Antibodies to the AÎ² Domain of APP Reduce Neuronal AÎ² and Protect against Synaptic Alterations. Journal of Biological Chemistry, 2007, 282, 18895-18906.	1.6	110
71	The Arctic Alzheimer mutation favors intracellular amyloid-Î² production by making amyloid precursor protein less available to Î±-secretase. Journal of Neurochemistry, 2007, 101, 854-862.	2.1	55
72	Neurogenesis as a Therapeutic Strategy for Cognitive Aging and Alzheimers Disease. Current Alzheimer Research, 2006, 3, 3-3.	0.7	5

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73	Editorial - A Milestone for Current Alzheimer Research [Hot Topic: Neurogenesis Catalyst Conference (Guest Editors: Howard M. Fillit & Gunnar Gouras)]. Current Alzheimer Research, 2006, 3, 1-1.	0.7	0
74	beta-Amyloid Accumulation Impairs Multivesicular Body Sorting by Inhibiting the Ubiquitin-Proteasome System. Journal of Neuroscience, 2006, 26, 4277-4288.	1.7	285
75	Regulation of NMDA receptor trafficking by amyloid-Î². Nature Neuroscience, 2005, 8, 1051-1058.	7.1	1,417
76	The immune system, amyloid-beta peptide, and Alzheimer's disease. Immunological Reviews, 2005, 205, 244-256.	2.8	58
77	Beta-amyloid accumulation in APP mutant neurons reduces PSD-95 and GluR1 in synapses. Neurobiology of Disease, 2005, 20, 187-198.	2.1	356
78	Amyloid-Î² oligomers are inefficiently measured by enzyme-linked immunosorbent assay. Annals of Neurology, 2005, 58, 147-150.	2.8	88
79	Molecular Pathology of Dementia. , 2005, 18, 258-269.		0
80	Conditional Inactivation of Presenilin 1 Prevents Amyloid Accumulation and Temporarily Rescues Contextual and Spatial Working Memory Impairments in Amyloid Precursor Protein Transgenic Mice. Journal of Neuroscience, 2005, 25, 6755-6764.	1.7	139
81	Intraneuronal Â-Amyloid Expression Downregulates the Akt Survival Pathway and Blunts the Stress Response. Journal of Neuroscience, 2005, 25, 10960-10969.	1.7	109
82	Intraneuronal AÎ² accumulation and origin of plaques in Alzheimer's disease. Neurobiology of Aging, 2005, 26, 1235-1244.	1.5	306
83	Intraneuronal AÎ² Accumulation-More Evidence, Less Controversy?: Alzheimer Research Forum Live Discussion. Journal of Alzheimer's Disease, 2004, 6, 443-449.	1.2	145
84	Increased plaque burden in brains of APP mutant MnSOD heterozygous knockout mice. Journal of Neurochemistry, 2004, 89, 1308-1312.	2.1	256
85	Oligomerization of Alzheimer's Â-Amyloid within Processes and Synapses of Cultured Neurons and Brain. Journal of Neuroscience, 2004, 24, 3592-3599.	1.7	415
86	P1-195 Oligomerization of Alzheimer's AÎ² within processes and synapses of cultured neurons and brain. Neurobiology of Aging, 2004, 25, S151.	1.5	1
87	Chaperones increase association of tau protein with microtubules. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 721-726.	3.3	421
88	Intraneuronal Alzheimer AÎ²42 Accumulates in Multivesicular Bodies and Is Associated with Synaptic Pathology. American Journal of Pathology, 2002, 161, 1869-1879.	1.9	664
89	Alzheimer beta-amyloid peptides: normal and abnormal localization. Histology and Histopathology, 2002, 17, 239-46.	0.5	39
90	Metal Chelator Decreases Alzheimer Î²-Amyloid Plaques. Neuron, 2001, 30, 641-642.	3.8	45

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91	Current theories for the molecular and cellular pathogenesis of Alzheimer's disease. Expert Reviews in Molecular Medicine, 2001, 3, 1-11.	1.6	6
92	Stimulation of Î²-Amyloid Precursor Protein Trafficking by Insulin Reduces Intraneuronal Î²-Amyloid and Requires Mitogen-Activated Protein Kinase Signaling. Journal of Neuroscience, 2001, 21, 2561-2570.	1.7	460
93	Testosterone reduces neuronal secretion of Alzheimer's beta -amyloid peptides. Proceedings of the National Academy of Sciences of the United States of America, 2000, 97, 1202-1205.	3.3	280
94	Intraneuronal AÎ²42 Accumulation in Human Brain. American Journal of Pathology, 2000, 156, 15-20.	1.9	930
95	Cellular and molecular basis of b-amyloid precursor protein metabolism (Y2K update). Frontiers in Bioscience - Landmark, 2000, 5, d72.	3.0	38
96	Endoplasmic reticulum and trans-Golgi network generate distinct populations of Alzheimer beta -amyloid peptides. Proceedings of the National Academy of Sciences of the United States of America, 1999, 96, 742-747.	3.3	354
97	Estrogen reduces neuronal generation of Alzheimer Î²-amyloid peptides. Nature Medicine, 1998, 4, 447-451.	15.2	545
98	Generation and Regulation of Î²â€Amyloid Peptide Variants by Neurons. Journal of Neurochemistry, 1998, 71, 1920-1925.	2.1	111
99	Cellular and molecular basis of beta-amyloid precursor protein metabolism. Frontiers in Bioscience - Landmark, 1998, 3, d399-407.	3.0	14
100	Evidence for phosphorylation and oligomeric assembly of presenilin 1. Proceedings of the National Academy of Sciences of the United States of America, 1997, 94, 5090-5094.	3.3	159
101	Increased apolipoprotein E ?4 in epilepsy with senile plaques. Annals of Neurology, 1997, 41, 402-404.	2.8	90
102	Apolipoprotein E, A?-Amyloid, and the Molecular Pathology of Alzheimer's Disease Therapeutic Implications. Annals of the New York Academy of Sciences, 1996, 802, 42-49.	1.8	9
103	Decreased senile plaque density in Alzheimer neocortex adjacent to an omental transposition. Neurological Research, 1996, 18, 291-294.	0.6	12
104	Myotonia in colchicine myoneuropathy. , 1996, 19, 870-875.		41
105	Highly selective effects of nerve growth factor, brain-derived neurotrophic factor, and neurotrophin-3 on intact and injured basal forebrain magnocellular neurons. Journal of Comparative Neurology, 1994, 343, 247-262.	0.9	112
106	Tyrosine-hydroxylase-containing neurons in the primate basal forebrain magnocellular complex. Brain Research, 1992, 584, 287-293.	1.1	20
107	Biologic Effects of Nerve Growth Factor on Lesioned Basal Forebrain Neuronsa. Annals of the New York Academy of Sciences, 1991, 640, 102-109.	1.8	15
108	AÎ²/Amyloid Precursor Protein-Induced Hyperexcitability and Dysregulation of Homeostatic Synaptic Plasticity in Neuron Models of Alzheimerâ€™s Disease. Frontiers in Aging Neuroscience, 0, 14, .	1.7	6