Christian Tackenberg

List of Publications by Year
in descending order

21 papers	993 citations	516710 16 h-index	752698 20 g-index
24 all docs	24 docs citations	24 times ranked	1435 citing authors

#	Article	IF	Citations
1	Intracerebral Transplantation and In Vivo Bioluminescence Tracking of Human Neural Progenitor Cells in the Mouse Brain. Journal of Visualized Experiments, 2022, , .	0.3	4
2	APOE2, E3, and E4 differentially modulate cellular homeostasis, cholesterol metabolism, and inflammatory response in isogenic iPSC-derived astrocytes. Stem Cell Reports, 2022, 17, 110-126.	4.8	40
3	Isoform- and cell-state-specific lipidation of ApoE in astrocytes. Cell Reports, 2022, 38, 110435.	6.4	35
4	Increased maturation of iPSC-derived neurons in a hydrogel-based 3D culture. Journal of Neuroscience Methods, 2021, 360, 109254.	2.5	16
5	Characterization of the Blood Brain Barrier Disruption in the Photothrombotic Stroke Model. Frontiers in Physiology, 2020, 11, 586226.	2.8	28
6	Familial Alzheimerâ€™s disease mutations at position 22 of the amyloid Î²-peptide sequence differentially affect synaptic loss, tau phosphorylation and neuronal cell death in an ex vivo system. PLoS ONE, 2020, 15, e0239584.	2.5	15
7	A Practical Guide to the Automated Analysis of Vascular Growth, Maturation and Injury in the Brain. Frontiers in Neuroscience, 2020, 14, 244.	2.8	31
8	Alzheimerâ€™s in a dish â€“ induced pluripotent stem cell-based disease modeling. Translational Neurodegeneration, 2019, 8, 21.	8.0	23
9	The secreted APP ectodomain sAPPÎ±, but not sAPPÎ², protects neurons against AÎ² oligomer-induced dendritic spine loss and increased tau phosphorylation. Molecular Brain, 2019, 12, 27.	2.6	36
10	Oxidative stress and altered mitochondrial protein expression in the absence of amyloid-Î² and tau pathology in iPSC-derived neurons from sporadic Alzheimer's disease patients. Stem Cell Research, 2018, 27, 121-130.	0.7	107
11	Genetic ablation of the p66Shc adaptor protein reverses cognitive deficits and improves mitochondrial function in an APP transgenic mouse model of Alzheimerâ€™s disease. Molecular Psychiatry, 2017, 22, 605-614.	7.9	26
12	AÎ²-mediated spine changes in the hippocampus are microtubule-dependent and can be reversed by a subnanomolar concentration of the microtubule-stabilizing agent epothilone D. Neuropharmacology, 2016, 105, 84-95.	4.1	48
13	Calcium flux-independent NMDA receptor activity is required for AÎ² oligomer-induced synaptic loss. Cell Death and Disease, 2015, 6, e1791-e1791.	6.3	71
14	Active vaccination with ankyrin G reduces Î²-amyloid pathology in APP transgenic mice. Molecular Psychiatry, 2013, 18, 358-368.	7.9	23
15	NMDA receptor subunit composition determines beta-amyloid-induced neurodegeneration and synaptic loss. Cell Death and Disease, 2013, 4, e608-e608.	6.3	108
16	Early accumulation of intracellular fibrillar oligomers and late congophilic amyloid angiopathy in mice expressing the Osaka intra-AÎ² APP mutation. Translational Psychiatry, 2012, 2, e183-e183.	4.8	45
17	High-Resolution Imaging and Evaluation of Spines in Organotypic Hippocampal Slice Cultures. Methods in Molecular Biology, 2012, 846, 277-293.	0.9	21
18	Thin, Stubby or Mushroom: Spine Pathology in Alzheimers Disease. Current Alzheimer Research, 2009, 6, 261-268.	1.4	100

#	Article	IF	Citations
19	Divergent Pathways Mediate Spine Alterations and Cell Death Induced by Amyloid-Î², Wild-Type Tau, and R406W Tau. Journal of Neuroscience, 2009, 29, 14439-14450.	3.6	128
20	Tau Aggregation and Progressive Neuronal Degeneration in the Absence of Changes in Spine Density and Morphology after Targeted Expression of Alzheimer's Disease-Relevant Tau Constructs in Organotypic Hippocampal Slices. Journal of Neuroscience, 2006, 26, 6103-6114.	3.6	80
21	Human tau-dependent toxicity in APP transgenic cultures requires calcium influx through N-methyl-D-aspartate receptors. Matters, 0, , .	1.0	1