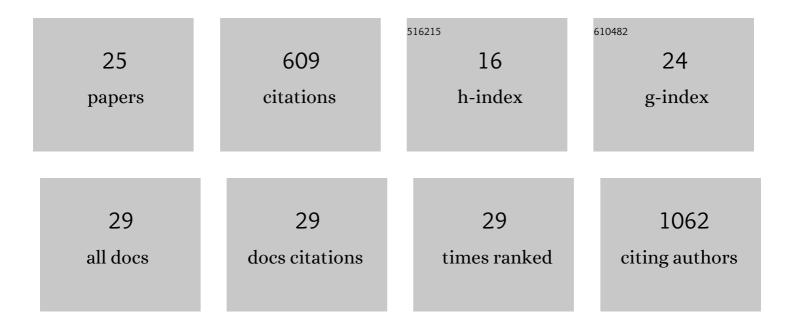
Serena Stanga

List of Publications by Year in descending order

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SEDENA STANCA

#	Article	IF	CITATIONS
1	Activation of the Hepcidin-Ferroportin1 pathway in the brain and astrocytic–neuronal crosstalk to counteract iron dyshomeostasis during aging. Scientific Reports, 2022, 12, .	1.6	4
2	How to Build and to Protect the Neuromuscular Junction: The Role of the Glial Cell Line-Derived Neurotrophic Factor. International Journal of Molecular Sciences, 2021, 22, 136.	1.8	16
3	Cachexia, a Systemic Disease beyond Muscle Atrophy. International Journal of Molecular Sciences, 2020, 21, 8592.	1.8	22
4	Drug Screening and Drug Repositioning as Promising Therapeutic Approaches for Spinal Muscular Atrophy Treatment. Frontiers in Pharmacology, 2020, 11, 592234.	1.6	20
5	Deferasirox-Dependent Iron Chelation Enhances Mitochondrial Dysfunction and Restores p53 Signaling by Stabilization of p53 Family Members in Leukemic Cells. International Journal of Molecular Sciences, 2020, 21, 7674.	1.8	14
6	Mitochondrial Dysfunctions: A Red Thread across Neurodegenerative Diseases. International Journal of Molecular Sciences, 2020, 21, 3719.	1.8	61
7	Mitochondria: A Galaxy in the Hematopoietic and Leukemic Stem Cell Universe. International Journal of Molecular Sciences, 2020, 21, 3928.	1.8	18
8	Presenilin-Deficient Neurons and Astrocytes Display Normal Mitochondrial Phenotypes. Frontiers in Neuroscience, 2020, 14, 586108.	1.4	6
9	Amyloid Precursor Protein (APP) Controls the Expression of the Transcriptional Activator Neuronal PAS Domain Protein 4 (NPAS4) and Synaptic GABA Release. ENeuro, 2020, 7, ENEURO.0322-19.2020.	0.9	24
10	Specificity of presenilinâ€1―and presenilinâ€2â€dependent γâ€secretases towards substrate processing. Journ of Cellular and Molecular Medicine, 2018, 22, 823-833.	al 1.6	23
11	A Role for GDNF and Soluble APP as Biomarkers of Amyotrophic Lateral Sclerosis Pathophysiology. Frontiers in Neurology, 2018, 9, 384.	1.1	33
12	Presenilin 2-Dependent Maintenance of Mitochondrial Oxidative Capacity and Morphology. Frontiers in Physiology, 2017, 8, 796.	1.3	40
13	Glycines from the APP GXXXG/GXXXA Transmembrane Motifs Promote Formation of Pathogenic AÎ ² Oligomers in Cells. Frontiers in Aging Neuroscience, 2016, 8, 107.	1.7	28
14	APPâ€dependent glial cell lineâ€derived neurotrophic factor gene expression drives neuromuscular junction formation. FASEB Journal, 2016, 30, 1696-1711.	0.2	27
15	Analysis by a highly sensitive split luciferase assay of the regions involved in APP dimerization and its impact on processing. FEBS Open Bio, 2015, 5, 763-773.	1.0	25
16	Characterization of Pterocarpus erinaceus kino extract and its gamma-secretase inhibitory properties. Journal of Ethnopharmacology, 2015, 163, 192-202.	2.0	17
17	Gamma-Secretase Inhibitor Activity of a <i>Pterocarpus erinaceus</i> Extract. Neurodegenerative Diseases, 2014, 14, 39-51.	0.8	12
18	Searching for Predictive Blood Biomarkers: Misfolded p53 In Mild Cognitive Impairment. Current Alzheimer Research, 2012, 9, 1191-1197.	0.7	15

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#	Article	IF	Citations
19	The Expanding Universe of Neurotrophic Factors: Therapeutic Potential in Aging and Age-Associated Disorders. Current Pharmaceutical Design, 2010, 16, 698-717.	0.9	46
20	Unfolded p53 in Blood as a Predictive Signature Signature of the Transition from Mild Cognitive Impairment to Alzheimer's Disease. Journal of Alzheimer's Disease, 2010, 20, 97-104.	1.2	31
21	Recruitment of Casein Kinase 2 is Involved in AβPP Processing Following Cholinergic Stimulation. Journal of Alzheimer's Disease, 2010, 20, 1133-1141.	1.2	7
22	Homeodomain Interacting Protein Kinase 2: A Target for Alzheimer's Beta Amyloid Leading to Misfolded p53 and Inappropriate Cell Survival. PLoS ONE, 2010, 5, e10171.	1.1	50
23	Unfolded p53 in the pathogenesis of Alzheimer's disease: is HIPK2 the link?. Aging, 2010, 2, 545-554.	1.4	44
24	Pharmacogenetics and Pharmagenomics, Trends in Normal and Pathological Aging Studies: Focus on p53. Current Pharmaceutical Design, 2008, 14, 2665-2671.	0.9	23
25	APP-deficient neurons show a subtle differential gene expression pattern: impairment in the expression of the activity-dependent transcription factor, NPAS4 Frontiers in Neuroscience, 0, 11, .	1.4	0