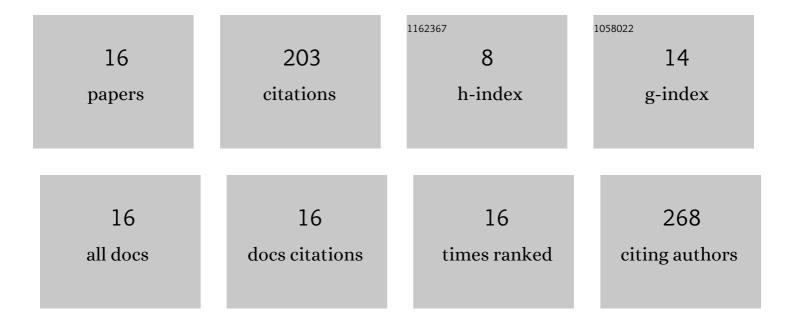
Rafael Teixeira Ribeiro

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	S-adenosylmethionine induces mitochondrial dysfunction, permeability transition pore opening and redox imbalance in subcellular preparations of rat liver. Journal of Bioenergetics and Biomembranes, 2021, 53, 525-539.	1.0	3
2	Neuronal Death, Glial Reactivity, Microglia Activation, Oxidative Stress and Bioenergetics Impairment Caused by Intracerebroventricular Administration of D-2-hydroxyglutaric Acid to Neonatal Rats. Neuroscience, 2021, 471, 115-132.	1.1	8
3	Free Radical Scavengers Prevent Argininosuccinic Acid-Induced Oxidative Stress in the Brain of Developing Rats: a New Adjuvant Therapy for Argininosuccinate Lyase Deficiency?. Molecular Neurobiology, 2020, 57, 1233-1244.	1.9	10
4	Disruption of Brain Redox Homeostasis, Microglia Activation and Neuronal Damage Induced by Intracerebroventricular Administration of S-Adenosylmethionine to Developing Rats. Molecular Neurobiology, 2019, 56, 2760-2773.	1.9	16
5	Acute lysine overload provokes marked striatum injury involving oxidative stress signaling pathways in glutaryl-CoA dehydrogenase deficient mice. Neurochemistry International, 2019, 129, 104467.	1.9	10
6	l-Carnitine prevents oxidative stress in striatum of glutaryl-CoA dehydrogenase deficient mice submitted to lysine overload. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2019, 1865, 2420-2427.	1.8	10
7	Induction of Neuroinflammatory Response and Histopathological Alterations Caused by Quinolinic Acid Administration in the Striatum of Glutaryl-CoA Dehydrogenase Deficient Mice. Neurotoxicity Research, 2018, 33, 593-606.	1.3	6
8	Experimental Evidence that In Vivo Intracerebral Administration of L-2-Hydroxyglutaric Acid to Neonatal Rats Provokes Disruption of Redox Status and Histopathological Abnormalities in the Brain. Neurotoxicity Research, 2018, 33, 681-692.	1.3	16
9	S-Adenosylmethionine Promotes Oxidative Stress and Decreases Na+, K+-ATPase Activity in Cerebral Cortex Supernatants of Adolescent Rats: Implications for the Pathogenesis of S-Adenosylhomocysteine Hydrolase Deficiency. Molecular Neurobiology, 2018, 55, 5868-5878.	1.9	9
10	α-Ketoadipic Acid and α-Aminoadipic Acid Cause Disturbance of Glutamatergic Neurotransmission and Induction of Oxidative Stress In Vitro in Brain of Adolescent Rats. Neurotoxicity Research, 2017, 32, 276-290.	1.3	15
11	Mevalonolactone disrupts mitochondrial functions and induces permeability transition pore opening in rat brain mitochondria: Implications for the pathogenesis of mevalonic aciduria. Neurochemistry International, 2017, 108, 133-145.	1.9	8
12	cis-4-Decenoic and decanoic acids impair mitochondrial energy, redox and Ca 2+ homeostasis and induce mitochondrial permeability transition pore opening in rat brain and liver: Possible implications for the pathogenesis of MCAD deficiency. Biochimica Et Biophysica Acta - Bioenergetics, 2016, 1857, 1363-1372.	0.5	15
13	Oxidative Stress, Disrupted Energy Metabolism, and Altered Signaling Pathways in Glutaryl-CoA Dehydrogenase Knockout Mice: Potential Implications of Quinolinic Acid Toxicity in the Neuropathology of Glutaric Acidemia Type I. Molecular Neurobiology, 2016, 53, 6459-6475.	1.9	35
14	In vivo intracerebral administration of L-2-hydroxyglutaric acid provokes oxidative stress and histopathological alterations in striatum and cerebellum of adolescent rats. Free Radical Biology and Medicine, 2015, 83, 201-213.	1.3	24
15	Acute lysine overload provokes protein oxidative damage and reduction of antioxidant defenses in the brain of infant glutaryl-CoA dehydrogenase deficient mice: A role for oxidative stress in GA I neuropathology. Journal of the Neurological Sciences, 2014, 344, 105-113.	0.3	14
16	Disturbance of Mitochondrial Dynamics, Endoplasmic Reticulum–Mitochondria Crosstalk, Redox Homeostasis, and Inflammatory Response in the Brain of Glutaryl-CoA Dehydrogenase-Deficient Mice: Neuroprotective Effects of Bezafibrate. Molecular Neurobiology, 0, , .	1.9	4