Alexandre Umpierrez Amaral

List of Publications by Year
in descending order

77 papers	1,510 citations	331670 21 h-index	395702 33 g-index
77 all docs	77 docs citations	77 times ranked	1819 citing authors

#	Article	IF	Citations
1	Mitochondrial dysfunction in fatty acid oxidation disorders: insights from human and animal studies. Bioscience Reports, 2016, 36, e00281.	2.4	138
2	Î±-Ketoisocaproic acid and leucine provoke mitochondrial bioenergetic dysfunction in rat brain. Brain Research, 2010, 1324, 75-84.	2.2	75
3	Experimental Evidence that Phenylalanine Provokes Oxidative Stress in Hippocampus and Cerebral Cortex of Developing Rats. Cellular and Molecular Neurobiology, 2010, 30, 317-326.	3.3	58
4	Conventional and ultrasound-assisted methods for extraction of bioactive compounds from red araÃ§Ã¡ peel (Psidium cattleianum Sabine). Arabian Journal of Chemistry, 2020, 13, 5800-5809.	4.9	56
5	Experimental Evidence that Methylmalonic Acid Provokes Oxidative Damage and Compromises Antioxidant Defenses in Nerve Terminal and Striatum of Young Rats. Cellular and Molecular Neurobiology, 2011, 31, 775-785.	3.3	49
6	Long-chain 3-hydroxy fatty acids accumulating in long-chain 3-hydroxyacyl-CoA dehydrogenase and mitochondrial trifunctional protein deficiencies uncouple oxidative phosphorylation in heart mitochondria. Journal of Bioenergetics and Biomembranes, 2013, 45, 47-57.	2.3	39
7	Induction of oxidative stress by the metabolites accumulating in 3-methylglutaconic aciduria in cerebral cortex of young rats. Life Sciences, 2008, 82, 652-662.	4.3	35
8	Disturbance of brain energy and redox homeostasis provoked by sulfite and thiosulfate: Potential pathomechanisms involved in the neuropathology of sulfite oxidase deficiency. Gene, 2013, 531, 191-198.	2.2	35
9	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.	4.0	35
10	Neurochemical evidence that phytanic acid induces oxidative damage and reduces the antioxidant defenses in cerebellum and cerebral cortex of rats. Life Sciences, 2010, 87, 275-280.	4.3	33
11	Sulfite disrupts brain mitochondrial energy homeostasis and induces mitochondrial permeability transition pore opening via thiol group modification. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 1413-1422.	3.8	31
12	Disturbance of mitochondrial energy homeostasis caused by the metabolites accumulating in LCHAD and MTP deficiencies in rat brain. Life Sciences, 2010, 86, 825-831.	4.3	30
13	Evidence that 3â€hydroxyâ€3â€methylglutaric acid promotes lipid and protein oxidative damage and reduces the nonenzymatic antioxidant defenses in rat cerebral cortex. Journal of Neuroscience Research, 2008, 86, 683-693.	2.9	29
14	Induction of oxidative stress in brain of glutaryl-CoA dehydrogenase deficient mice by acute lysine administration. Molecular Genetics and Metabolism, 2012, 106, 31-38.	1.1	29
15	Disruption of brain redox homeostasis in glutaryl-CoA dehydrogenase deficient mice treated with high dietary lysine supplementation. Molecular Genetics and Metabolism, 2013, 108, 30-39.	1.1	29
16	2â€Methylcitric acid impairs glutamate metabolism and induces permeability transition in brain mitochondria. Journal of Neurochemistry, 2016, 137, 62-75.	3.9	27
17	Glycine Provokes Lipid Oxidative Damage and Reduces the Antioxidant Defenses in Brain Cortex of Young Rats. Cellular and Molecular Neurobiology, 2009, 29, 253-261.	3.3	24
18	Marked reduction of Na+, K+-ATPase and creatine kinase activities induced by acute lysine administration in glutaryl-CoA dehydrogenase deficient mice. Molecular Genetics and Metabolism, 2012, 107, 81-86.	1.1	24

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19	Reduction of Na+, K+-ATPase activity and expression in cerebral cortex of glutaryl-CoA dehydrogenase deficient mice: A possible mechanism for brain injury in glutaric aciduria type I. Molecular Genetics and Metabolism, 2012, 107, 375-382.	1.1	24
20	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.	2.9	24
21	Induction of oxidative stress by the metabolites accumulating in isovaleric acidemia in brain cortex of young rats. Free Radical Research, 2008, 42, 707-715.	3.3	22
22	Striatum is more vulnerable to oxidative damage induced by the metabolites accumulating in 3â€hydroxyâ€3â€methylglutarylâ€CoA lyase deficiency as compared to liver. International Journal of Developmental Neuroscience, 2009, 27, 351-356.	1.6	22
23	Mitochondrial bioenergetics deregulation caused by long-chain 3-hydroxy fatty acids accumulating in LCHAD and MTP deficiencies in rat brain: A possible role of mPTP opening as a pathomechanism in these disorders?. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2014, 1842, 1658-1667.	3.8	22
24	Redox homeostasis is compromised in vivo by the metabolites accumulating in 3-hydroxy-3-methylglutaryl-CoA lyase deficiency in rat cerebral cortex and liver. Free Radical Research, 2013, 47, 1066-1075.	3.3	21
25	Disturbance of mitochondrial functions provoked by the major long-chain 3-hydroxylated fatty acids accumulating in MTP and LCHAD deficiencies in skeletal muscle. Toxicology in Vitro, 2016, 36, 1-9.	2.4	20
26	Experimental evidence that ornithine and homocitrulline disrupt energy metabolism in brain of young rats. Brain Research, 2009, 1291, 102-112.	2.2	19
27	Uncoupling, metabolic inhibition and induction of mitochondrial permeability transition in rat liver mitochondria caused by the major long-chain hydroxyl monocarboxylic fatty acids accumulating in LCHAD deficiency. Biochimica Et Biophysica Acta - Bioenergetics, 2015, 1847, 620-628.	1.0	19
28	Metabolite accumulation in <scp>VLCAD</scp> deficiency markedly disrupts mitochondrial bioenergetics and Ca²⁺ homeostasis in the heart. FEBS Journal, 2018, 285, 1437-1455.	4.7	19
29	Lysine induces lipid and protein damage and decreases reduced glutathione concentrations in brain of young rats. International Journal of Developmental Neuroscience, 2008, 26, 693-698.	1.6	18
30	Deregulation of mitochondrial functions provoked by longâ€chain fatty acid accumulating in longâ€chain 3â€hydroxyacylâ€CoA dehydrogenase and mitochondrial permeability transition deficiencies in rat heart â€“ mitochondrial permeability transition pore opening as a potential contributing pathomechanism of cardiac alterations in these disorders. FEBS Journal, 2015, 282, 4714-4726.	4.7	17
31	Bioenergetics dysfunction, mitochondrial permeability transition pore opening and lipid peroxidation induced by hydrogen sulfide as relevant pathomechanisms underlying the neurological dysfunction characteristic of ethylmalonic encephalopathy. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2017, 1863, 2192-2201.	3.8	17
32	Pathogenesis of brain damage in glutaric acidemia type I: Lessons from the genetic mice model. International Journal of Developmental Neuroscience, 2019, 78, 215-221.	1.6	17
33	Pristanic acid promotes oxidative stress in brain cortex of young rats: A possible pathophysiological mechanism for brain damage in peroxisomal disorders. Brain Research, 2011, 1382, 259-265.	2.2	16
34	Disruption of Mitochondrial Homeostasis by Phytanic Acid in Cerebellum of Young Rats. Cerebellum, 2013, 12, 362-369.	2.5	16
35	Disturbance of energy and redox homeostasis and reduction of Na+,K+-ATPase activity provoked by in vivo intracerebral administration of ethylmalonic acid to young rats. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2015, 1852, 759-767.	3.8	16
36	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.	2.7	16

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37	Ethylmalonic acid impairs brain mitochondrial succinate and malate transport. Molecular Genetics and Metabolism, 2012, 105, 84-90.	1.1	15
38	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.	1.0	15
39	Î±-Ketoadipic Acid and Î±-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.	2.7	15
40	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.	0.6	14
41	Experimental evidence that overexpression of NR2B glutamate receptor subunit is associated with brain vacuolation in adult glutaryl-CoA dehydrogenase deficient mice: A potential role for glutamatergic-induced excitotoxicity in GA I neuropathology. Journal of the Neurological Sciences, 2015, 359, 133-140.	0.6	14
42	Ornithine and Homocitrulline Impair Mitochondrial Function, Decrease Antioxidant Defenses and Induce Cell Death in Menadione-Stressed Rat Cortical Astrocytes: Potential Mechanisms of Neurological Dysfunction in HHH Syndrome. Neurochemical Research, 2016, 41, 2190-2198.	3.3	14
43	Guanosine Neuroprotection of Presynaptic Mitochondrial Calcium Homeostasis in a Mouse Study with Amyloid-Î² Oligomers. Molecular Neurobiology, 2020, 57, 4790-4809.	4.0	14
44	Experimental evidence that bioenergetics disruption is not mainly involved in the brain injury of glutaryl-CoA dehydrogenase deficient mice submitted to lysine overload. Brain Research, 2015, 1620, 116-129.	2.2	13
45	Glycine intrastriatal administration induces lipid and protein oxidative damage and alters the enzymatic antioxidant defenses in rat brain. Life Sciences, 2011, 89, 276-281.	4.3	12
46	Disruption of redox homeostasis and histopathological alterations caused by in vivo intrastriatal administration of D-2-hydroxyglutaric acid to young rats. Neuroscience, 2014, 277, 281-293.	2.3	12
47	Impairment of <scp>GABA</scp>ergic system contributes to epileptogenesis in glutaric acidemia type I. Epilepsia, 2017, 58, 1771-1781.	5.1	12
48	Pathophysiology of maple syrup urine disease: Focus on the neurotoxic role of the accumulated branched-chain amino acids and branched-chain Î±-keto acids. Neurochemistry International, 2022, 157, 105360.	3.8	12
49	D-Serine induces lipid and protein oxidative damage and decreases glutathione levels in brain cortex of rats. Brain Research, 2009, 1256, 34-42.	2.2	11
50	Ethylmalonic Acid Induces Permeability Transition in Isolated Brain Mitochondria. Neurotoxicity Research, 2014, 26, 168-178.	2.7	11
51	Disruption of mitochondrial functions and oxidative stress contribute to neurologic dysfunction in organic acidurias. Archives of Biochemistry and Biophysics, 2020, 696, 108646.	3.0	11
52	Disturbance of bioenergetics and calcium homeostasis provoked by metabolites accumulating in propionic acidemia in heart mitochondria of developing rats. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2020, 1866, 165682.	3.8	11
53	Influence of ketone bodies on oxidative stress parameters in brain of developing rats in vitro. Metabolic Brain Disease, 2008, 23, 411-425.	2.9	10
54	Neurochemical Evidence that Lysine Inhibits Synaptic Na+,K+-ATPase Activity and Provokes Oxidative Damage in Striatum of Young Rats In vivo. Neurochemical Research, 2011, 36, 205-214.	3.3	10

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55	Acute lysine overload provokes marked striatum injury involving oxidative stress signaling pathways in glutaryl-CoA dehydrogenase deficient mice. Neurochemistry International, 2019, 129, 104467.	3.8	10
56	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.	3.8	10
57	Creatine administration prevents Na+,K+-ATPase inhibition induced by intracerebroventricular administration of isovaleric acid in cerebral cortex of young rats. Brain Research, 2009, 1262, 81-88.	2.2	9
58	Evidence that the major metabolites accumulating in hyperornithinemiaâ€“hyperammonemiaâ€“homocitrullinuria syndrome induce oxidative stress in brain of young rats. International Journal of Developmental Neuroscience, 2009, 27, 635-641.	1.6	9
59	<scp>d</scp>â€Serine administration provokes lipid oxidation and decreases the antioxidant defenses in rat striatum. International Journal of Developmental Neuroscience, 2010, 28, 297-301.	1.6	9
60	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.	4.0	9
61	Experimental evidence that pristanic acid disrupts mitochondrial homeostasis in brain of young rats. Journal of Neuroscience Research, 2012, 90, 597-605.	2.9	8
62	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.	3.8	8
63	Experimental evidence that maleic acid markedly compromises glutamate oxidation through inhibition of glutamate dehydrogenase and Î±-ketoglutarate dehydrogenase activities in kidney of developing rats. Molecular and Cellular Biochemistry, 2019, 458, 99-112.	3.1	8
64	Disturbance of mitochondrial functions associated with permeability transition pore opening induced by cis-5-tetradecenoic and myristic acids in liver of adolescent rats. Mitochondrion, 2020, 50, 1-13.	3.4	8
65	Lipopolysaccharide-Elicited Systemic Inflammation Induces Selective Vulnerability of Cerebral Cortex and Striatum of Developing Glutaryl-CoA Dehydrogenase Deficient (Gcdhâ^'/â^') Mice to Oxidative Stress. Neurotoxicity Research, 2020, 38, 1024-1036.	2.7	8
66	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.	2.3	8
67	Evidence that 2-methylacetoacetate induces oxidative stress in rat brain. Metabolic Brain Disease, 2010, 25, 261-267.	2.9	7
68	NMDA Receptors and Oxidative Stress Induced by the Major Metabolites Accumulating in HMG Lyase Deficiency Mediate Hypophosphorylation of Cytoskeletal Proteins in Brain From Adolescent Rats: Potential Mechanisms Contributing to the Neuropathology of This Disease. Neurotoxicity Research, 2015, 28, 239-252.	2.7	7
69	Higher Vulnerability of Menadione-Exposed Cortical Astrocytes of Glutaryl-CoA Dehydrogenase Deficient Mice to Oxidative Stress, Mitochondrial Dysfunction, and Cell Death: Implications for the Neurodegeneration in Glutaric Aciduria Type I. Molecular Neurobiology, 2017, 54, 4795-4805.	4.0	7
70	Evidence that thiol group modification and reactive oxygen species are involved in hydrogen sulfide-induced mitochondrial permeability transition pore opening in rat cerebellum. Mitochondrion, 2019, 47, 141-150.	3.4	7
71	Impairment of mitochondrial bioenergetics and permeability transition induction caused by major long-chain fatty acids accumulating in VLCAD deficiency in skeletal muscle as potential pathomechanisms of myopathy. Toxicology in Vitro, 2020, 62, 104665.	2.4	7
72	Recent Advances in the Pathophysiology of Fatty Acid Oxidation Defects: Secondary Alterations of Bioenergetics and Mitochondrial Calcium Homeostasis Caused by the Accumulating Fatty Acids. Frontiers in Genetics, 2020, 11, 598976.	2.3	7

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73	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.	2.7	6
74	High vulnerability of the heart and liver to 3â€hydroxypalmitic acidâ€“induced disruption of mitochondrial functions in intact cell systems. Journal of Cellular Biochemistry, 2018, 119, 7678-7686.	2.6	4
75	Ethylmalonic acid impairs bioenergetics by disturbing succinate and glutamate oxidation and induces mitochondrial permeability transition pore opening in rat cerebellum. Journal of Neurochemistry, 2021, 158, 262-281.	3.9	3
76	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.	2.3	3
77	The Effect of Periodontal Diseases and Cognitive Deficit on Behavioral State, Oxidative Stress Parameters and Alveolar Bone Loss in Rats. Journal of the International Academy of Periodontology, 2020, 22, 156-165.	0.7	1