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List of Publications by Year in Descending Order

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

48 papers	977 citations	19 h-index	29 g-index
51 ext. papers	1,073 ext. citations	4.3 avg, IF	3.38 L-index

#	Paper	IF	Citations
48	Methylmalonic Acid Compromises Respiration and Reduces the Expression of Differentiation Markers of SH-SY5Y Human Neuroblastoma Cells. *ACS Chemical Neuroscience*, **2021**, 12, 2608-2618	5.7	0
47	Targeting mitochondria in melanoma: Interplay between MAPK signaling pathway and mitochondrial dynamics. *Biochemical Pharmacology*, **2020**, 178, 114104	6	9
46	Methylmalonic Acid Compromises Energy Metabolism and Decreases the Expression of Neuronal Differentiation Genes in SH-SY5Y Human Neuroblastoma Cells. *FASEB Journal*, **2020**, 34, 1-1	0.9	
45	Lipopolysaccharide-Elicited Systemic Inflammation Induces Selective Vulnerability of Cerebral Cortex and Striatum of Developing Glutaryl-CoA Dehydrogenase Deficient (Gcdh) Mice to Oxidative Stress. *Neurotoxicity Research*, **2020**, 38, 1024-1036	4.3	4
44	Long Lasting High Lysine Diet Aggravates White Matter Injury in Glutaryl-CoA Dehydrogenase Deficient (Gcdh-/-) Mice. *Molecular Neurobiology*, **2019**, 56, 648-657	6.2	6
43	Bradykinin-potentiating PEPTIDE-10C, an argininosuccinate synthetase activator, protects against HO-induced oxidative stress in SH-SY5Y neuroblastoma cells. *Peptides*, **2018**, 103, 90-97	3.8	11
42	Experimental Evidence that 3-Methylglutaric Acid Disturbs Mitochondrial Function and Induced Oxidative Stress in Rat Brain Synaptosomes: New Converging Mechanisms. *Neurochemical Research*, **2016**, 41, 2619-2626	4.6	13
41	3-Hydroxy-3-methylglutaric and 3-methylglutaric acids impair redox status and energy production and transfer in rat heart: relevance for the pathophysiology of cardiac dysfunction in 3-hydroxy-3-methylglutaryl-coenzyme A lyase deficiency. *Free Radical Research*, **2016**, 50, 997-1010	4	13
40	Reactive nitrogen species mediate oxidative stress and astrogliosis provoked by in vivo administration of phytanic acid in cerebellum of adolescent rats: A potential contributing pathomechanism of cerebellar injury in peroxisomal disorders. *Neuroscience*, **2015**, 304, 122-32	3.9	16
39	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, 201-13	7.8	21
38	Toxic synergism between quinolinic acid and organic acids accumulating in glutaric acidemia type I and in disorders of propionate metabolism in rat brain synaptosomes: Relevance for metabolic acidemias. *Neuroscience*, **2015**, 308, 64-74	3.9	19
37	The effect of WIN 55,212-2 suggests a cannabinoid-sensitive component in the early toxicity induced by organic acids accumulating in glutaric acidemia type I and in related disorders of propionate metabolism in rat brain synaptosomes. *Neuroscience*, **2015**, 310, 578-88	3.9	10
36	Striatal neuronal death mediated by astrocytes from the Gcdh-/- mouse model of glutaric acidemia type I. *Human Molecular Genetics*, **2015**, 24, 4504-15	5.6	18
35	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, 116-29	3.7	12
34	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, 759-67	6.9	16
33	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, 105-13	3.2	12
32	Disruption of oxidative phosphorylation and synaptic Na(+), K(+)-ATPase activity by pristanic acid in cerebellum of young rats. *Life Sciences*, **2014**, 94, 67-73	6.8	

31	Pristanic acid provokes lipid, protein, and DNA oxidative damage and reduces the antioxidant defenses in cerebellum of young rats. *Cerebellum*, **2014**, 13, 751-9	4.3	7
30	Glycine intracerebroventricular administration disrupts mitochondrial energy homeostasis in cerebral cortex and striatum of young rats. *Neurotoxicity Research*, **2013**, 24, 502-11	4.3	8
29	Marked inhibition of Na+, K(+)- ATPase activity and the respiratory chain by phytanic acid in cerebellum from young rats: possible underlying mechanisms of cerebellar ataxia in Refsum disease. *Journal of Bioenergetics and Biomembranes*, **2013**, 45, 137-44	3.7	14
28	Disruption of brain redox homeostasis in glutaryl-CoA dehydrogenase deficient mice treated with high dietary lysine supplementation. *Molecular Genetics and Metabolism*, **2013**, 108, 30-9	3.7	27
27	Neurochemical evidence that the metabolites accumulating in 3-methylcrotonyl-CoA carboxylase deficiency induce oxidative damage in cerebral cortex of young rats. *Cellular and Molecular Neurobiology*, **2013**, 33, 137-46	4.6	12
26	3-Methylcrotonylglycine disrupts mitochondrial energy homeostasis and inhibits synaptic Na(+),K(+)-ATPase activity in brain of young rats. *Cellular and Molecular Neurobiology*, **2012**, 32, 297-307	4.6	13
25	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, 81-6	3.7	23
24	Ethylmalonic acid impairs brain mitochondrial succinate and malate transport. *Molecular Genetics and Metabolism*, **2012**, 105, 84-90	3.7	13
23	Disruption of redox homeostasis in cerebral cortex of developing rats by acylcarnitines accumulating in medium-chain acyl-CoA dehydrogenase deficiency. *International Journal of Developmental Neuroscience*, **2012**, 30, 383-90	2.7	11
22	Neurochemical evidence that 3-methylglutaric acid inhibits synaptic Na+,K+-ATPase activity probably through oxidative damage in brain cortex of young rats. *International Journal of Developmental Neuroscience*, **2011**, 29, 1-7	2.7	24
21	Creatine administration prevents Na+,K+-ATPase inhibition induced by intracerebroventricular administration of isovaleric acid in cerebral cortex of young rats. *Brain Research*, **2009**, 1262, 81-8	3.7	8
20	Striatum is more vulnerable to oxidative damage induced by the metabolites accumulating in 3-hydroxy-3-methylglutaryl-CoA lyase deficiency as compared to liver. *International Journal of Developmental Neuroscience*, **2009**, 27, 351-6	2.7	20
19	Chronic early postnatal glutaric acid administration causes cognitive deficits in the water maze. *Behavioural Brain Research*, **2008**, 187, 411-6	3.4	9
18	Inhibition of brain energy metabolism by the branched-chain amino acids accumulating in maple syrup urine disease. *Neurochemical Research*, **2008**, 33, 114-24	4.6	46
17	Isovaleric acid reduces Na+, K+-ATPase activity in synaptic membranes from cerebral cortex of young rats. *Cellular and Molecular Neurobiology*, **2007**, 27, 529-40	4.6	30
16	Evidence that the inhibitory effects of guanidinoacetate on the activities of the respiratory chain, Na+,K+-ATPase and creatine kinase can be differentially prevented by taurine and vitamins E and C administration in rat striatum in vivo. *Biochimica Et Biophysica Acta - Molecular Basis of Disease*, **2007**, 1772, 563-9	6.9	17
15	Evidence that glutaric acid reduces glutamate uptake by cerebral cortex of infant rats. *Life Sciences*, **2007**, 81, 1668-76	6.8	30
14	Evidence that quinolinic acid severely impairs energy metabolism through activation of NMDA receptors in striatum from developing rats. *Journal of Neurochemistry*, **2006**, 99, 1531-42	6	49

13	Glutaric acid administration impairs energy metabolism in midbrain and skeletal muscle of young rats. *Neurochemical Research*, **2005**, 30, 1123-31	4.6	29
12	Inhibition of energy metabolism by 2-methylacetoacetate and 2-methyl-3-hydroxybutyrate in cerebral cortex of developing rats. *Journal of Inherited Metabolic Disease*, **2005**, 28, 501-15	5.4	16
11	Inhibition of energy metabolism in cerebral cortex of young rats by the medium-chain fatty acids accumulating in MCAD deficiency. *Brain Research*, **2004**, 1030, 141-51	3.7	30
10	Evidence that 3-hydroxyglutaric acid interacts with NMDA receptors in synaptic plasma membranes from cerebral cortex of young rats. *Neurochemistry International*, **2004**, 45, 1087-94	4.4	42
9	Inhibition of creatine kinase activity from rat cerebral cortex by D-2-hydroxyglutaric acid in vitro. *Neurochemistry International*, **2004**, 44, 45-52	4.4	39
8	3-hydroxyglutaric acid enhances glutamate uptake into astrocytes from cerebral cortex of young rats. *Neurochemistry International*, **2004**, 44, 345-53	4.4	23
7	Evidence that antioxidants prevent the inhibition of Na+,K(+)-ATPase activity induced by octanoic acid in rat cerebral cortex in vitro. *Neurochemical Research*, **2003**, 28, 1255-63	4.6	16
6	Effects of L-2-hydroxyglutaric acid on various parameters of the glutamatergic system in cerebral cortex of rats. *Metabolic Brain Disease*, **2003**, 18, 233-43	3.9	13
5	Ethylmalonic acid inhibits mitochondrial creatine kinase activity from cerebral cortex of young rats in vitro. *Neurochemical Research*, **2003**, 28, 771-7	4.6	25
4	Inhibition of brain energy metabolism by the alpha-keto acids accumulating in maple syrup urine disease. *Biochimica Et Biophysica Acta - Molecular Basis of Disease*, **2003**, 1639, 232-8	6.9	67
3	L-2-hydroxyglutaric acid inhibits mitochondrial creatine kinase activity from cerebellum of developing rats. *International Journal of Developmental Neuroscience*, **2003**, 21, 217-24	2.7	27
2	Inhibition of creatine kinase activity in vitro by ethylmalonic acid in cerebral cortex of young rats. *Neurochemical Research*, **2002**, 27, 1633-9	4.6	39
1	Inhibition of cytochrome c oxidase activity in rat cerebral cortex and human skeletal muscle by D-2-hydroxyglutaric acid in vitro. *Biochimica Et Biophysica Acta - Molecular Basis of Disease*, **2002**, 1586, 81-91	6.9	69