Alberto Ferri

List of Publications by Year
in descending order

59 papers	3,242 citations	159525 30 h-index	149623 56 g-index
61 all docs	61 docs citations	61 times ranked	4291 citing authors

#	Article	IF	Citations
1	Lipid catabolism and mitochondrial uncoupling are stimulated in brown adipose tissue of amyotrophic lateral sclerosis mouse models. Genes and Diseases, 2023, 10, 321-324.	1.5	1
2	Repurposing of Trimetazidine for amyotrophic lateral sclerosis: A study in SOD1^{G93A}mice. British Journal of Pharmacology, 2022, 179, 1732-1752.	2.7	21
3	Proteome data of neuroblastoma cells overexpressing Neuroglobin. Data in Brief, 2022, 41, 107843.	0.5	8
4	Mechanistic Insights of Mitochondrial Dysfunction in Amyotrophic Lateral Sclerosis: An Update on a Lasting Relationship. Metabolites, 2022, 12, 233.	1.3	11
5	Skeletal Muscle in ALS: An Unappreciated Therapeutic Opportunity?. Cells, 2021, 10, 525.	1.8	32
6	Microglia Morphological Changes in the Motor Cortex of hSOD1G93A Transgenic ALS Mice. Brain Sciences, 2021, 11, 807.	1.1	6
7	Skeletal Muscle Metabolism: Origin or Prognostic Factor for Amyotrophic Lateral Sclerosis (ALS) Development?. Cells, 2021, 10, 1449.	1.8	11
8	Protein Aggregation Landscape in Neurodegenerative Diseases: Clinical Relevance and Future Applications. International Journal of Molecular Sciences, 2021, 22, 6016.	1.8	28
9	Overexpression of Neuroglobin Promotes Energy Metabolism and Autophagy Induction in Human Neuroblastoma SH-SY5Y Cells. Cells, 2021, 10, 3394.	1.8	14
10	Impact of the chronic disease self-management program (CDSMP) on self-perceived frailty condition: the EU-EFFICHRONIC project. Therapeutic Advances in Chronic Disease, 2021, 12, 204062232110567.	1.1	5
11	A multimolecular signaling complex including PrPCand LRP1 is strictly dependent on lipid rafts and is essential for the function of tissue plasminogen activator. Journal of Neurochemistry, 2020, 152, 468-481.	2.1	24
12	Altered skeletal muscle glucose-fatty acid flux in amyotrophic lateral sclerosis. Brain Communications, 2020, 2, fcaa154.	1.5	32
13	Editorial: Mitochondrial Proteomics: Understanding Mitochondria Function and Dysfunction Through the Characterization of Their Proteome. Frontiers in Cell and Developmental Biology, 2020, 8, 608753.	1.8	0
14	Skeletal-Muscle Metabolic Reprogramming in ALS-SOD1G93A Mice Predates Disease Onset and Is A Promising Therapeutic Target. IScience, 2020, 23, 101087.	1.9	55
15	Histaminergic transmission slows progression of amyotrophic lateral sclerosis. Journal of Cachexia, Sarcopenia and Muscle, 2019, 10, 872-893.	2.9	27
16	Neuroglobin overexpression plays a pivotal role in neuroprotection through mitochondrial raft-like microdomains in neuroblastoma SK-N-BE2 cells. Molecular and Cellular Neurosciences, 2018, 88, 167-176.	1.0	18
17	Differential toxicity of TAR DNAâ€binding protein 43 isoforms depends on their submitochondrial localization in neuronal cells. Journal of Neurochemistry, 2018, 146, 585-597.	2.1	39
18	SIRT3 and mitochondrial metabolism in neurodegenerative diseases. Neurochemistry International, 2017, 109, 184-192.	1.9	89

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19	What is â€œHyperâ€•in the ALS Hypermetabolism?. Mediators of Inflammation, 2017, 2017, 1-11.	1.4	64
20	Improvement of skeletal muscle performance in ageing by the metabolic modulator Trimetazidine. Journal of Cachexia, Sarcopenia and Muscle, 2016, 7, 449-457.	2.9	44
21	Role of mitochondrial raft-like microdomains in the regulation of cell apoptosis. Apoptosis: an International Journal on Programmed Cell Death, 2015, 20, 621-634.	2.2	46
22	CNF1 Enhances Brain Energy Content and Counteracts Spontaneous Epileptiform Phenomena in Aged DBA/2J Mice. PLoS ONE, 2015, 10, e0140495.	1.1	9
23	Acute focal brain damage alters mitochondrial dynamics and autophagy in axotomized neurons. Cell Death and Disease, 2014, 5, e1545-e1545.	2.7	57
24	Glutaredoxin 1 is a major player in copper metabolism in neuroblastoma cells. Biochimica Et Biophysica Acta - General Subjects, 2014, 1840, 255-261.	1.1	14
25	Mitochondria and ALS: Implications from novel genes and pathways. Molecular and Cellular Neurosciences, 2013, 55, 44-49.	1.0	81
26	CNF1 Increases Brain Energy Level, Counteracts Neuroinflammatory Markers and Rescues Cognitive Deficits in a Murine Model of Alzheimer's Disease. PLoS ONE, 2013, 8, e65898.	1.1	37
27	Caspase-3 triggers early synaptic dysfunction in a mouse model of Alzheimer's disease. Nature Neuroscience, 2011, 14, 69-76.	7.1	479
28	Mitochondrial redox signalling by p66Shc mediates ALS-like disease through Rac1 inactivation. Human Molecular Genetics, 2011, 20, 4196-4208.	1.4	41
29	Copper depletion increases the mitochondrial-associated SOD1 in neuronal cells. BioMetals, 2011, 24, 269-278.	1.8	13
30	HIF1-positive and HIF1-negative glioblastoma cells compete in vitro but cooperate in tumor growth in vivo. International Journal of Oncology, 2010, 36, 785-91.	1.4	12
31	Inactivation of cytochrome c oxidase by mutant SOD1s in mouse motoneuronal NSCâ€34 cells is independent from copper availability but is because of nitric oxide. Journal of Neurochemistry, 2010, 112, 183-192.	2.1	25
32	Glutaredoxin 2 prevents aggregation of mutant SOD1 in mitochondria and abolishes its toxicity. Human Molecular Genetics, 2010, 19, 4529-4542.	1.4	79
33	Dynamic NAD(P)H post-synaptic autofluorescence signals for the assessment of mitochondrial function in a neurodegenerative disease: Monitoring the primary motor cortex of G93A mice, an amyotrophic lateral sclerosis model. Mitochondrion, 2010, 10, 108-114.	1.6	14
34	The Proinflammatory Action of Microglial P2 Receptors Is Enhanced in SOD1 Models for Amyotrophic Lateral Sclerosis. Journal of Immunology, 2009, 183, 4648-4656.	0.4	105
35	Oligomerization of Mutant SOD1 in Mitochondria of Motoneuronal Cells Drives Mitochondrial Damage and Cell Toxicity. Antioxidants and Redox Signaling, 2009, 11, 1547-1558.	2.5	79
36	Impairment of mitochondrial calcium handling in a mtSOD1 cell culture model of motoneuron disease. BMC Neuroscience, 2009, 10, 64.	0.8	92

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37	Oxidative inactivation of calcineurin by Cu,Zn superoxide dismutase G93A, a mutant typical of familial amyotrophic lateral sclerosis. Journal of Neurochemistry, 2008, 79, 531-538.	2.1	33
38	Inflammatory cytokines increase mitochondrial damage in motoneuronal cells expressing mutant SOD1. Neurobiology of Disease, 2008, 32, 454-460.	2.1	16
39	Amyotrophic Lateral Sclerosis: From Current Developments in the Laboratory to Clinical Implications. Antioxidants and Redox Signaling, 2008, 10, 405-444.	2.5	131
40	Cysteine 111 Affects Aggregation and Cytotoxicity of Mutant Cu,Zn-superoxide Dismutase Associated with Familial Amyotrophic Lateral Sclerosis. Journal of Biological Chemistry, 2008, 283, 866-874.	1.6	110
41	Superoxide dismutase 1 modulates expression of transferrin receptor. Journal of Biological Inorganic Chemistry, 2006, 11, 489-498.	1.1	41
42	Apaf1 mediates apoptosis and mitochondrial damage induced by mutant human SOD1s typical of familial amyotrophic lateral sclerosis. Neurobiology of Disease, 2006, 21, 69-79.	2.1	25
43	Familial ALS-superoxide dismutases associate with mitochondria and shift their redox potentials. Proceedings of the National Academy of Sciences of the United States of America, 2006, 103, 13860-13865.	3.3	231
44	Impairment of glutamate transport and increased vulnerability to oxidative stress in neuroblastoma SH-SY5Y cells expressing a Cu,Zn superoxide dismutase typical of familial amyotrophic lateral sclerosis. Neurochemistry International, 2005, 46, 227-234.	1.9	29
45	Cell death in amyotrophic lateral sclerosis: interplay between neuronal and glial cells. FASEB Journal, 2004, 18, 1261-1263.	0.2	55
46	Apoptosome inactivation rescues proneural and neural cells from neurodegeneration. Cell Death and Differentiation, 2004, 11, 1179-1191.	5.0	42
47	Activity of protein phosphatase calcineurin is decreased in sporadic and familial amyotrophic lateral sclerosispatients. Journal of Neurochemistry, 2004, 90, 1237-1242.	2.1	34
48	Overexpression of superoxide dismutase 1 protects against Î²-amyloid peptide toxicity: effect of estrogen and copper chelators. Neurochemistry International, 2004, 44, 25-33.	1.9	53
49	Neurodegeneration in amyotrophic lateral sclerosis: the role of oxidative stress and altered homeostasis of metals. Brain Research Bulletin, 2003, 61, 365-374.	1.4	186
50	Mitochondrial dysfunction due to mutant copper/zinc superoxide dismutase associated with amyotrophic lateral sclerosis is reversed by N-acetylcysteine. Neurobiology of Disease, 2003, 13, 213-221.	2.1	74
51	Resistance to striatal dopamine depletion induced by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine in mice expressing human mutant Cu,Zn superoxide dismutase. Neuroscience Letters, 2002, 325, 124-128.	1.0	10
52	Calcineurin Activity Is Regulated Both by Redox Compounds and by Mutant Familial Amyotrophic Lateral Sclerosis-Superoxide Dismutase. Journal of Neurochemistry, 2002, 75, 606-613.	2.1	46
53	Oxidative modulation of nuclear factor-Î°B in human cells expressing mutant fALS-typical superoxide dismutases. Journal of Neurochemistry, 2002, 83, 1019-1029.	2.1	35
54	Aberrant Copper Chemistry as a Major Mediator of Oxidative Stress in a Human Cellular Model of Amyotrophic Lateral Sclerosis. Journal of Neurochemistry, 2001, 73, 1175-1180.	2.1	56

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55	Phosphatidylinositol 3-kinase is recruited to a specific site in the activated IL-1 receptor I. FEBS Letters, 1998, 438, 49-54.	1.3	68
56	Voltage-activated sodium currents in a cell line expressing a Cu,Zn superoxide dismutase typical of familial ALS. NeuroReport, 1998, 9, 3515-3518.	0.6	20
57	Expression of a Cu,Zn superoxide dismutase typical of familial amyotrophic lateral sclerosis induces mitochondrial alteration and increase of cytosolic Ca2+concentration in transfected neuroblastoma SH-SY5Y cells. FEBS Letters, 1997, 414, 365-368.	1.3	195
58	Different polymeric forms of actin detected by the fluorescent probe terbium ion. Biochemistry, 1981, 20, 6362-6366.	1.2	10
59	G-actin modified by plasma membrane interaction polymerizes only in the presence of filamentous myosin. FEBS Letters, 1980, 112, 67-69.	1.3	19