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	Caspase-3 triggers early synaptic dysfunction in a mouse model of Alzheimer's disease. Nature Neuroscience, 2011, 14, 69-76.	7.1	479
2	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
3	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
4	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
5	Amyotrophic Lateral Sclerosis: From Current Developments in the Laboratory to Clinical Implications. Antioxidants and Redox Signaling, 2008, 10, 405-444.	2.5	131
6	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
7	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
8	Impairment of mitochondrial calcium handling in a mtSOD1 cell culture model of motoneuron disease. BMC Neuroscience, 2009, 10, 64.	0.8	92
9	SIRT3 and mitochondrial metabolism in neurodegenerative diseases. Neurochemistry International, 2017, 109, 184-192.	1.9	89
10	Mitochondria and ALS: Implications from novel genes and pathways. Molecular and Cellular Neurosciences, 2013, 55, 44-49.	1.0	81
11	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
12	Glutaredoxin 2 prevents aggregation of mutant SOD1 in mitochondria and abolishes its toxicity. Human Molecular Genetics, 2010, 19, 4529-4542.	1.4	79
13	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
14	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
15	What is â€œHyperâ€•in the ALS Hypermetabolism?. Mediators of Inflammation, 2017, 2017, 1-11.	1.4	64
16	Acute focal brain damage alters mitochondrial dynamics and autophagy in axotomized neurons. Cell Death and Disease, 2014, 5, e1545-e1545.	2.7	57
17	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
18	Cell death in amyotrophic lateral sclerosis: interplay between neuronal and glial cells. FASEB Journal, 2004, 18, 1261-1263.	0.2	55

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19	Skeletal-Muscle Metabolic Reprogramming in ALS-SOD1G93A Mice Predates Disease Onset and Is A Promising Therapeutic Target. IScience, 2020, 23, 101087.	1.9	55
20	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
21	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
22	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
23	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
24	Apoptosome inactivation rescues proneural and neural cells from neurodegeneration. Cell Death and Differentiation, 2004, 11, 1179-1191.	5.0	42
25	Superoxide dismutase 1 modulates expression of transferrin receptor. Journal of Biological Inorganic Chemistry, 2006, 11, 489-498.	1.1	41
26	Mitochondrial redox signalling by p66Shc mediates ALS-like disease through Rac1 inactivation. Human Molecular Genetics, 2011, 20, 4196-4208.	1.4	41
27	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
28	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
29	Oxidative modulation of nuclear factor-ÎºB in human cells expressing mutant fALS-typical superoxide dismutases. Journal of Neurochemistry, 2002, 83, 1019-1029.	2.1	35
30	Activity of protein phosphatase calcineurin is decreased in sporadic and familial amyotrophic lateral sclerosispatients. Journal of Neurochemistry, 2004, 90, 1237-1242.	2.1	34
31	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
32	Altered skeletal muscle glucose-fatty acid flux in amyotrophic lateral sclerosis. Brain Communications, 2020, 2, fcaa154.	1.5	32
33	Skeletal Muscle in ALS: An Unappreciated Therapeutic Opportunity?. Cells, 2021, 10, 525.	1.8	32
34	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
35	Protein Aggregation Landscape in Neurodegenerative Diseases: Clinical Relevance and Future Applications. International Journal of Molecular Sciences, 2021, 22, 6016.	1.8	28
36	Histaminergic transmission slows progression of amyotrophic lateral sclerosis. Journal of Cachexia, Sarcopenia and Muscle, 2019, 10, 872-893.	2.9	27

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37	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
38	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
39	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
40	Repurposing of Trimetazidine for amyotrophic lateral sclerosis: A study in SOD1^{G93A}mice. British Journal of Pharmacology, 2022, 179, 1732-1752.	2.7	21
41	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
42	G-actin modified by plasma membrane interaction polymerizes only in the presence of filamentous myosin. FEBS Letters, 1980, 112, 67-69.	1.3	19
43	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
44	Inflammatory cytokines increase mitochondrial damage in motoneuronal cells expressing mutant SOD1. Neurobiology of Disease, 2008, 32, 454-460.	2.1	16
45	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
46	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
47	Overexpression of Neuroglobin Promotes Energy Metabolism and Autophagy Induction in Human Neuroblastoma SH-SY5Y Cells. Cells, 2021, 10, 3394.	1.8	14
48	Copper depletion increases the mitochondrial-associated SOD1 in neuronal cells. BioMetals, 2011, 24, 269-278.	1.8	13
49	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
50	Skeletal Muscle Metabolism: Origin or Prognostic Factor for Amyotrophic Lateral Sclerosis (ALS) Development?. Cells, 2021, 10, 1449.	1.8	11
51	Mechanistic Insights of Mitochondrial Dysfunction in Amyotrophic Lateral Sclerosis: An Update on a Lasting Relationship. Metabolites, 2022, 12, 233.	1.3	11
52	Different polymeric forms of actin detected by the fluorescent probe terbium ion. Biochemistry, 1981, 20, 6362-6366.	1.2	10
53	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
54	CNF1 Enhances Brain Energy Content and Counteracts Spontaneous Epileptiform Phenomena in Aged DBA/2J Mice. PLoS ONE, 2015, 10, e0140495.	1.1	9

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55	Proteome data of neuroblastoma cells overexpressing Neuroglobin. Data in Brief, 2022, 41, 107843.	0.5	8
56	Microglia Morphological Changes in the Motor Cortex of hSOD1G93A Transgenic ALS Mice. Brain Sciences, 2021, 11, 807.	1.1	6
57	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
58	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
59	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