Maria Teresa Carri

List of Publications by Year
in descending order

92 papers	4,754 citations	66343 42 h-index	102487 66 g-index
92 all docs	92 docs citations	92 times ranked	5947 citing authors

#	Article	IF	Citations
1	UsnRNP trafficking is regulated by stress granules and compromised by mutant ALS proteins. Neurobiology of Disease, 2020, 138, 104792.	4.4	15
2	The S100A4 Transcriptional Inhibitor Niclosamide Reduces Pro-Inflammatory and Migratory Phenotypes of Microglia: Implications for Amyotrophic Lateral Sclerosis. Cells, 2019, 8, 1261.	4.1	24
3	nNOS/GSNOR interaction contributes to skeletal muscle differentiation and homeostasis. Cell Death and Disease, 2019, 10, 354.	6.3	9
4	50-Hz magnetic field impairs the expression of iron-related genes in the in vitro SOD1^{G93A} model of amyotrophic lateral sclerosis. International Journal of Radiation Biology, 2019, 95, 368-377.	1.8	9
5	Mitochondria in the nervous system: From health to disease, part II. Neurochemistry International, 2018, 117, 1-4.	3.8	6
6	Neuroinflammation in Amyotrophic Lateral Sclerosis: Role of Redox (dys)Regulation. Antioxidants and Redox Signaling, 2018, 29, 15-36.	5.4	31
7	Differential toxicity of TAR DNAâ€binding protein 43 isoforms depends on their submitochondrial localization in neuronal cells. Journal of Neurochemistry, 2018, 146, 585-597.	3.9	39
8	Epigenetic Changes Associated with the Expression of Amyotrophic Lateral Sclerosis (ALS) Causing Genes. Neuroscience, 2018, 390, 1-11.	2.3	18
9	Oxidative stress and mitochondrial damage in the pathogenesis of ALS: New perspectives. Neuroscience Letters, 2017, 636, 3-8.	2.1	92
10	SIRT3 and mitochondrial metabolism in neurodegenerative diseases. Neurochemistry International, 2017, 109, 184-192.	3.8	89
11	Mitochondria in the nervous system: From health to disease, Part I. Neurochemistry International, 2017, 109, 1-4.	3.8	7
12	Functional interaction between FUS and SMN underlies SMA-like splicing changes in wild-type hFUS mice. Scientific Reports, 2017, 7, 2033.	3.3	27
13	Pathways to mitochondrial dysfunction in ALS pathogenesis. Biochemical and Biophysical Research Communications, 2017, 483, 1187-1193.	2.1	72
14	Cysteine Modifications in the Pathogenesis of ALS. Frontiers in Molecular Neuroscience, 2017, 10, 5.	2.9	22
15	Role of LRRK2 in the regulation of dopamine receptor trafficking. PLoS ONE, 2017, 12, e0179082.	2.5	55
16	Old versus New Mechanisms in the Pathogenesis of ALS. Brain Pathology, 2016, 26, 276-286.	4.1	45
17	Structural insights into the multi-determinant aggregation of TDP-43 in motor neuron-like cells. Neurobiology of Disease, 2016, 94, 63-72.	4.4	29
18	Which TDP-43 aggregates are toxic in ALS?. Oncotarget, 2016, 7, 81973-81974.	1.8	2

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19	Diverse roles of FUS in Amyotrophic Lateral Sclerosis. SpringerPlus, 2015, 4, L54.	1.2	0
20	S-Nitrosoglutathione Reductase Plays Opposite Roles in SH-SY5Y Models of Parkinsonâ€™s Disease and Amyotrophic Lateral Sclerosis. Mediators of Inflammation, 2015, 2015, 1-12.	3.0	12
21	Mitochondrial dynamism and the pathogenesis of Amyotrophic Lateral Sclerosis. Frontiers in Cellular Neuroscience, 2015, 9, 31.	3.7	44
22	Oxidative stress and mitochondrial damage: importance in non-SOD1 ALS. Frontiers in Cellular Neuroscience, 2015, 9, 41.	3.7	98
23	Nuclear accumulation of mRNAs underlies G4C2 repeat-induced translational repression in a cellular model of C9orf72 ALS. Journal of Cell Science, 2015, 128, 1787-99.	2.0	96
24	Cystatin B and SOD1: Proteinâ€“Protein Interaction and Possible Relation to Neurodegeneration. Cellular and Molecular Neurobiology, 2014, 34, 205-213.	3.3	11
25	Mitochondria and ALS: Implications from novel genes and pathways. Molecular and Cellular Neurosciences, 2013, 55, 44-49.	2.2	81
26	Mislocalised FUS mutants stall spliceosomal snRNPs in the cytoplasm. Neurobiology of Disease, 2013, 55, 120-128.	4.4	60
27	The NADPH Oxidase Pathway Is Dysregulated by the P2X7 Receptor in the SOD1-G93A Microglia Model of Amyotrophic Lateral Sclerosis. Journal of Immunology, 2013, 190, 5187-5195.	0.8	103
28	The intriguing case of motor neuron disease: ALS and SMA come closer. Biochemical Society Transactions, 2013, 41, 1593-1597.	3.4	29
29	LRRK2 Affects Vesicle Trafficking, Neurotransmitter Extracellular Level and Membrane Receptor Localization. PLoS ONE, 2013, 8, e77198.	2.5	66
30	Amyotrophic Lateral Sclerosis: New Insights into Underlying Molecular Mechanisms and Opportunities for Therapeutic Intervention. Antioxidants and Redox Signaling, 2012, 17, 1277-1330.	5.4	58
31	Mitochondrial dysfunction in ALS. Progress in Neurobiology, 2012, 97, 54-66.	5.7	197
32	Interaction of Cisplatin with Human Superoxide Dismutase. Journal of the American Chemical Society, 2012, 134, 7009-7014.	13.7	65
33	Astroglial Inhibition of NF-ÎºB Does Not Ameliorate Disease Onset and Progression in a Mouse Model for Amyotrophic Lateral Sclerosis (ALS). PLoS ONE, 2011, 6, e17187.	2.5	55
34	Bcl2-A1 interacts with pro-caspase-3: Implications for amyotrophic lateral sclerosis. Neurobiology of Disease, 2011, 43, 642-650.	4.4	19
35	Mitochondrial redox signalling by p66Shc mediates ALS-like disease through Rac1 inactivation. Human Molecular Genetics, 2011, 20, 4196-4208.	2.9	41
36	SOD1 and mitochondria in ALS: a dangerous liaison. Journal of Bioenergetics and Biomembranes, 2011, 43, 593-599.	2.3	64

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37	Copper depletion increases the mitochondrial-associated SOD1 in neuronal cells. BioMetals, 2011, 24, 269-278.	4.1	13
38	Mutant SOD1 and mitochondrial damage alter expression and splicing of genes controlling neuritogenesis in models of neurodegeneration. Human Mutation, 2011, 32, 168-182.	2.5	33
39	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.	3.9	25
40	Glutaredoxin 2 prevents aggregation of mutant SOD1 in mitochondria and abolishes its toxicity. Human Molecular Genetics, 2010, 19, 4529-4542.	2.9	79
41	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.	3.4	14
42	Abnormal sensitivity of cannabinoid CB1 receptors in the striatum of mice with experimental amyotrophic lateral sclerosis. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders, 2010, 11, 83-90.	2.1	20
43	Amyotrophic Lateral Sclerosis: Mechanisms and Countermeasures. Antioxidants and Redox Signaling, 2009, 11, 1519-1522.	5.4	8
44	The Proinflammatory Action of Microglial P2 Receptors Is Enhanced in SOD1 Models for Amyotrophic Lateral Sclerosis. Journal of Immunology, 2009, 183, 4648-4656.	0.8	105
45	Neuroprotective and neuritogenic activities of novel multimodal ironâ€chelating drugs in motorâ€neuronâ€like NSCâ€34 cells and transgenic mouse model of amyotrophic lateral sclerosis. FASEB Journal, 2009, 23, 3766-3779.	0.5	121
46	Oligomerization of Mutant SOD1 in Mitochondria of Motoneuronal Cells Drives Mitochondrial Damage and Cell Toxicity. Antioxidants and Redox Signaling, 2009, 11, 1547-1558.	5.4	79
47	Impairment of mitochondrial calcium handling in a mtSOD1 cell culture model of motoneuron disease. BMC Neuroscience, 2009, 10, 64.	1.9	92
48	Treatment with lithium carbonate does not improve disease progression in two different strains of SOD1 mutant mice. Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders, 2009, 10, 221-228.	2.1	127
49	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.	3.9	33
50	Minocycline for patients with ALS. Lancet Neurology, The, 2008, 7, 118-119.	10.2	12
51	Inflammatory cytokines increase mitochondrial damage in motoneuronal cells expressing mutant SOD1. Neurobiology of Disease, 2008, 32, 454-460.	4.4	16
52	Amyotrophic Lateral Sclerosis: From Current Developments in the Laboratory to Clinical Implications. Antioxidants and Redox Signaling, 2008, 10, 405-444.	5.4	131
53	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.	3.4	110
54	The Role of Metals and their Effect on Oxidative Stress in Amyotrophic Lateral Sclerosis. , 2007, , 383-398.		0

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55	Apoptotic mechanisms in mutant LRRK2-mediated cell death. Human Molecular Genetics, 2007, 16, 1319-1326.	2.9	175
56	2â€DE and MALDIâ€TOFâ€MS for a comparative analysis of proteins expressed in different cellular models of amyotrophic lateral sclerosis. Electrophoresis, 2007, 28, 4320-4329.	2.4	13
57	Expression of a Cu,Zn superoxide dismutase typical for familial amyotrophic lateral sclerosis increases the vulnerability of neuroblastoma cells to infectious injury. BMC Infectious Diseases, 2007, 7, 131.	2.9	14
58	Mitochondrial damage modulates alternative splicing in neuronal cells: implications for neurodegeneration. Journal of Neurochemistry, 2007, 100, 142-153.	3.9	49
59	Beta-amyloid causes downregulation of calcineurin in neurons through induction of oxidative stress. Neurobiology of Disease, 2007, 26, 342-352.	4.4	52
60	Targets in ALS: designing multidrug therapies. Trends in Pharmacological Sciences, 2006, 27, 267-273.	8.7	60
61	Superoxide dismutase 1 modulates expression of transferrin receptor. Journal of Biological Inorganic Chemistry, 2006, 11, 489-498.	2.6	41
62	Apaf1 mediates apoptosis and mitochondrial damage induced by mutant human SOD1s typical of familial amyotrophic lateral sclerosis. Neurobiology of Disease, 2006, 21, 69-79.	4.4	25
63	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.	7.1	231
64	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.	3.8	29
65	Cell death in amyotrophic lateral sclerosis: interplay between neuronal and glial cells. FASEB Journal, 2004, 18, 1261-1263.	0.5	55
66	Activity of protein phosphatase calcineurin is decreased in sporadic and familial amyotrophic lateral sclerosispatients. Journal of Neurochemistry, 2004, 90, 1237-1242.	3.9	34
67	Lessons from models of SOD1-linked familial ALS. Trends in Molecular Medicine, 2004, 10, 393-400.	6.7	187
68	Overexpression of superoxide dismutase 1 protects against Î²-amyloid peptide toxicity: effect of estrogen and copper chelators. Neurochemistry International, 2004, 44, 25-33.	3.8	53
69	The sinister side of Italian soccer. Lancet Neurology, The, 2003, 2, 656-657.	10.2	46
70	Neurodegeneration in amyotrophic lateral sclerosis: the role of oxidative stress and altered homeostasis of metals. Brain Research Bulletin, 2003, 61, 365-374.	3.0	186
71	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.	4.4	74
72	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.	2.1	10

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73	Calcineurin Activity Is Regulated Both by Redox Compounds and by Mutant Familial Amyotrophic Lateral Sclerosis-Superoxide Dismutase. Journal of Neurochemistry, 2002, 75, 606-613.	3.9	46
74	Oxidative modulation of nuclear factor-ÎºB in human cells expressing mutant fALS-typical superoxide dismutases. Journal of Neurochemistry, 2002, 83, 1019-1029.	3.9	35
75	Differential role of superoxide and glutathione in S-nitrosoglutathione-mediated apoptosis: a rationale for mild forms of familial amyotrophic lateral sclerosis associated with less active Cu,Zn superoxide dismutase mutants. Journal of Neurochemistry, 2001, 77, 1433-1443.	3.9	35
76	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.	3.9	56
77	Neurochemistry of SOD1 and familial amyotrophic lateral sclerosis. Functional Neurology, 2001, 16, 73-82.	1.3	1
78	A Free Cysteine Residue at the Dimer Interface Decreases Conformational Stability of Xenopus laevis Copper,Zinc Superoxide Dismutase. Archives of Biochemistry and Biophysics, 2000, 377, 284-289.	3.0	14
79	Voltage-activated sodium currents in a cell line expressing a Cu,Zn superoxide dismutase typical of familial ALS. NeuroReport, 1998, 9, 3515-3518.	1.2	20
80	Effect of Lysâ†'Arg mutation on the thermal stability of Cu,Zn superoxide dismutase: influence on the monomerâ€“dimer equilibrium. Protein Engineering, Design and Selection, 1996, 9, 323-325.	2.1	19
81	Metal Uptake of Recombinant Cambialistic Superoxide Dismutase from Propionibacterium shermanii Is Affected by Growth Conditions of Host Escherichia coli Cells. Biochemical and Biophysical Research Communications, 1995, 216, 841-847.	2.1	26
82	Role of Zinc-Coordination and of the Glutathione Redox Couple in the Redox Susceptibility of Human Transcription Factor Sp1. Biochemical and Biophysical Research Communications, 1994, 201, 871-877.	2.1	66
83	Crystal structure of the cyanide-inhibitedXenopus laevisCu,Zn superoxide dismutase at 98 K. FEBS Letters, 1994, 349, 93-98.	2.8	35
84	Mutation of Lys-120 and Lys-134 drastically reduces the catalytic rate of Cu,Zn superoxide dismutase. FEBS Letters, 1994, 352, 76-78.	2.8	23
85	Crystallization and Preliminary Crystallographic Analysis of Recombinant Xenopus laevis Cu, Zn Superoxide Dismutase b. Biochemical and Biophysical Research Communications, 1993, 194, 1008-1011.	2.1	10
86	Evidence for co-regulation of Cu,Zn superoxide dismutase and metallothionein gene expression in yeast through transcriptional control by copper via the ACE 1 factor. FEBS Letters, 1991, 278, 263-266.	2.8	89
87	Activation and induction by copper of Cu/Zn superoxide dismutase in Saccharomyces cerevisiae. Presence of an inactive proenzyme in anaerobic yeast. FEBS Journal, 1991, 196, 545-549.	0.2	42
88	Increase of Cu,Zn-superoxide dismutase activity during differentiation of human K562 cells involves activation by copper of a constantly expressed copper-deficient protein.. Journal of Biological Chemistry, 1991, 266, 24580-24587.	3.4	62
89	Xenopus laevisCu,Zn superoxide dismutase B cDNA sequence. Nucleic Acids Research, 1990, 18, 1641-1641.	14.5	9
90	Primary structure from amino acid and cDNA sequences of two Cu,Zn superoxide dismutase variants from Xenopus laevis. Archives of Biochemistry and Biophysics, 1989, 272, 507-515.	3.0	20

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91	The relationship between chromosomal origins of replication and the nuclear matrix during the cell cycle. Experimental Cell Research, 1986, 164, 426-436.	2.6	70
92	An electron microscope study of chromosomal DNA replication in different eukaryotic systems. Experimental Cell Research, 1982, 137, 127-140.	2.6	34