Sergio Giannattasio

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Mitochondrial Research: Yeast and Human Cells as Models. International Journal of Molecular Sciences, 2022, 23, 6654.	4.1	0
2	Analysis of Mitochondrial Retrograde Signaling in Yeast Model Systems. Methods in Molecular Biology, 2021, 2276, 87-102.	0.9	3
3	RTG Signaling Sustains Mitochondrial Respiratory Capacity in HOG1-Dependent Osmoadaptation. Microorganisms, 2021, 9, 1894.	3.6	4
4	Epigenetic silencing of the ubiquitin ligase subunit FBXL7 impairs c-SRC degradation and promotes epithelial-to-mesenchymal transition and metastasis. Nature Cell Biology, 2020, 22, 1130-1142.	10.3	28
5	6-Thioguanine and Its Analogs Promote Apoptosis of Castration-Resistant Prostate Cancer Cells in a BRCA2-Dependent Manner. Cancers, 2019, 11, 945.	3.7	5
6	Acid Stress Triggers Resistance to Acetic Acid-Induced Regulated Cell Death through <i>Hog1</i> Activation Which Requires <i>RTG2</i> in Yeast. Oxidative Medicine and Cellular Longevity, 2019, 2019, 1-9.	4.0	25
7	SLC25A10 biallelic mutations in intractable epileptic encephalopathy with complex I deficiency. Human Molecular Genetics, 2018, 27, 499-504.	2.9	37
8	Guidelines and recommendations on yeast cell death nomenclature. Microbial Cell, 2018, 5, 4-31.	3.2	158
9	Mitochondria–cytosol–nucleus crosstalk: learning from Saccharomyces cerevisiae. FEMS Yeast Research, 2018, 18, .	2.3	53
10	Editorial: Cell Stress, Metabolic Reprogramming, and Cancer. Frontiers in Oncology, 2018, 8, 236.	2.8	5
11	New perspectives from South-Y-East, not all about death A report of the 12th International Meeting on Yeast Apoptosis in Bari, Italy, May 14th-18th, 2017. Microbial Cell, 2018, 5, 112-115.	3.2	0
12	Heterologous expression of carnation Italian ringspot virus p36 protein enhances necrotic cell death in response to acetic acid in Saccharomyces cerevisiae. Mechanisms of Ageing and Development, 2017, 161, 255-261.	4.6	2
13	Mitochondrial Dysfunction: A Novel Potential Driver of Epithelial-to-Mesenchymal Transition in Cancer. Frontiers in Oncology, 2017, 7, 295.	2.8	96
14	The transcription factors ADR1 or CAT8 are required for RTG pathway activation and evasion from yeast acetic acid-induced programmed cell death in raffinose. Microbial Cell, 2016, 3, 621-631.	3.2	18
15	Silencing of BRCA2 to Identify Novel BRCA2-regulated Biological Functions in Cultured Human Cells. Journal of Visualized Experiments, 2015, , e52849.	0.3	0
16	Differential proteome–metabolome profiling of YCA1-knock-out and wild type cells reveals novel metabolic pathways and cellular processes dependent on the yeast metacaspase. Molecular BioSystems, 2015, 11, 1573-1583.	2.9	9
17	Proteome and metabolome profiling of wild-type and YCA1 -knock-out yeast cells during acetic acid-induced programmed cell death. Journal of Proteomics, 2015, 128, 173-188.	2.4	27
18	Yeast as a Tool to Study Mitochondrial Retrograde Pathway En Route to Cell Stress Response. Methods in Molecular Biology, 2015, 1265, 321-331.	0.9	7

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19	The expanding role of yeast in cancer research and diagnosis: insights into the function of the oncosuppressors p53 and BRCA1/2. FEMS Yeast Research, 2014, 14, 2-16.	2.3	51
20	Mitochondrial dysfunction in cancer chemoresistance. Biochemical Pharmacology, 2014, 92, 62-72.	4.4	73
21	Silencing of BRCA2 decreases anoikis and its heterologous expression sensitizes yeast cells to acetic acid-induced programmed cell death. Apoptosis: an International Journal on Programmed Cell Death, 2014, 19, 1330-1341.	4.9	7
22	Yeast between life and death: a summary of the Ninth International Meeting on Yeast Apoptosis in Rome, Italy, 17–20 September 2012. Cell Death and Differentiation, 2013, 20, 1281-1283.	11.2	0
23	Yeast growth in raffinose results in resistance to acetic-acid induced programmed cell death mostly due to the activation of the mitochondrial retrograde pathway. Biochimica Et Biophysica Acta - Molecular Cell Research, 2013, 1833, 2765-2774.	4.1	39
24	Stressâ€Related Mitochondrial Components and Mitochondrial Genome as Targets of Anticancer Therapy. Chemical Biology and Drug Design, 2013, 81, 102-112.	3.2	17
25	Yeast Stress, Aging, and Death. Oxidative Medicine and Cellular Longevity, 2013, 2013, 1-3.	4.0	6
26	Molecular mechanisms of Saccharomyces cerevisiae stress adaptation and programmed cell death in response to acetic acid. Frontiers in Microbiology, 2013, 4, 33.	3.5	133
27	The role of mitochondria in yeast programmed cell death. Frontiers in Oncology, 2012, 2, 70.	2.8	54
28	The N-Acetylcysteine-Insensitive Acetic Acid-Induced Yeast Programmed Cell Death Occurs Without Macroautophagy. Current Pharmaceutical Biotechnology, 2012, 13, 2705-2711.	1.6	4
29	Yeast as a Tool to Study Signaling Pathways in Mitochondrial Stress Response and Cytoprotection. Scientific World Journal, The, 2012, 2012, 1-10.	2.1	35
30	Molecular Mechanisms of Programmed Cell Death Induced by Acetic Acid in Saccharomyces cerevisiae. Microbiology Monographs, 2012, , 57-75.	0.6	1
31	Cytochrome c Trp65Ser substitution results in inhibition of acetic acid-induced programmed cell death in Saccharomyces cerevisiae. Mitochondrion, 2011, 11, 987-991.	3.4	9
32	Achievements and perspectives in yeast acetic acid-induced programmed cell death pathways. Biochemical Society Transactions, 2011, 39, 1538-1543.	3.4	45
33	Yeast acetic acidâ€induced programmed cell death can occur without cytochrome <i>c</i> release which requires metacaspase YCA1. FEBS Letters, 2010, 584, 224-228.	2.8	52
34	Knockâ€out of metacaspase and/or cytochrome <i>c</i> results in the activation of a ROSâ€independent acetic acidâ€induced programmed cell death pathway in yeast. FEBS Letters, 2010, 584, 3655-3660.	2.8	32
35	Pleiotropic effects of the yeast Sal1 and Aac2 carriers on mitochondrial function via an activity distinct from adenine nucleotide transport. Molecular Genetics and Genomics, 2008, 280, 25-39.	2.1	17
36	Molecular evolution of B6 enzymes: Binding of pyridoxal-5'-phosphate and Lys41Arg substitution turn ribonuclease A into a model B6 protoenzyme. BMC Biochemistry, 2008, 9, 17.	4.4	16

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37	A transient proteasome activation is needed for acetic acid-induced programmed cell death to occur in Saccharomyces cerevisiae. FEMS Yeast Research, 2008, 8, 400-404.	2.3	16
38	Catalase T and Cu, Znâ€superoxide dismutase in the acetic acidâ€induced programmed cell death in <i>Saccharomyces cerevisiae</i> . FEBS Letters, 2008, 582, 210-214.	2.8	44
39	Cytochrome <i>c</i> is released from coupled mitochondria of yeast en route to acetic acidâ€induced programmed cell death and can work as an electron donor and a ROS scavenger. FEBS Letters, 2008, 582, 1519-1525.	2.8	55
40	Hydrogen peroxide and superoxide anion production during acetic acid-induced yeast programmed cell death. Folia Microbiologica, 2007, 52, 237-40.	2.3	37
41	Molecular Basis of Cystic Fibrosis in Lithuania: IncompleteCFTRMutation Detection by PCR-Based Screening Protocols. Genetic Testing and Molecular Biomarkers, 2006, 10, 169-173.	1.7	4
42	YCA1 participates in the acetic acid induced yeast programmed cell death also in a manner unrelated to its caspase-like activity. FEBS Letters, 2006, 580, 6880-6884.	2.8	71
43	Retrograde Response to Mitochondrial Dysfunction Is Separable from TOR1/2 Regulation of Retrograde Gene Expression. Journal of Biological Chemistry, 2005, 280, 42528-42535.	3.4	78
44	Acid stress adaptation protects Saccharomyces cerevisiae from acetic acid-induced programmed cell death. Gene, 2005, 354, 93-98.	2.2	112
45	An increase in the ATP levels occurs in cerebellar granule cells en route to apoptosis in which ATP derives from both oxidative phosphorylation and anaerobic glycolysis. Biochimica Et Biophysica Acta - Bioenergetics, 2005, 1708, 50-62.	1.0	56
46	Non-radioactive detection of five common microsatellite markers for ATP7B gene in Wilson disease patients. Molecular and Cellular Probes, 2003, 17, 271-274.	2.1	4
47	Simultaneous determination of purine nucleotides, their metabolites and β-nicotinamide adenine dinucleotide in cerebellar granule cells by ion-pair high performance liquid chromatography. Brain Research Protocols, 2003, 10, 168-174.	1.6	48
48	Glutamate neurotoxicity, oxidative stress and mitochondria. FEBS Letters, 2001, 497, 1-5.	2.8	306
49	Genetic Heterogeneity in Five Italian Regions: Analysis of PAH Mutations and Minihaplotypes. Human Heredity, 2001, 52, 154-159.	0.8	20
50	Early release and subsequent caspase-mediated degradation of cytochrome c in apoptotic cerebellar granule cells. FEBS Letters, 1999, 457, 126-130.	2.8	65
51	Kinetic properties and thermal stabilities of mutant forms of mitochondrial aspartate aminotransferase. BBA - Proteins and Proteomics, 1998, 1386, 29-38.	2.1	8
52	Active-site Arg → Lys Substitutions Alter Reaction and Substrate Specificity of Aspartate Aminotransferase. Journal of Biological Chemistry, 1997, 272, 21932-21937.	3.4	45
53	Detection of microsatellites by ethidium bromide staining. The analysis of an STR system in the human phenylalanine hydroxylase gene. Molecular and Cellular Probes, 1997, 11, 81-83.	2.1	5
54	The STR252 - IVS10nt546 - VNTR7 phenylalanine hydroxylase minihaplotype in five Mediterranean samples. Human Genetics, 1997, 100, 350-355.	3.8	17

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55	Novel missense mutation in the phenylalanine hydroxylase gene leading to complete loss of enzymatic activity. Human Mutation, 1995, 6, 247-249.	2.5	4
56	Cumulative Effects of Mutations in Newly Synthesized Mitochondrial Aspartate Aminotransferase on Uptake into Mitochondria. Biochemical and Biophysical Research Communications, 1995, 214, 511-517.	2.1	1
57	Use of Protease Sensitivity to Probe the Conformations of Newly Synthesized Mutant Forms of Mitochondrial Aspartate Aminotransferase. Biochemical and Biophysical Research Communications, 1995, 215, 800-807.	2.1	0
58	Molecular screening of genetic defects with RNA–SSCP analysis: the PKU and cystinuria model. Molecular and Cellular Probes, 1995, 9, 201-205.	2.1	3
59	Characterization of mitochondrial DNA in primary cardiomyopathies. Clinica Chimica Acta, 1995, 243, 181-189.	1.1	13
60	The N-Terminal Region of Mature Mitochondrial Aspartate Aminotransferase Can Direct Cytosolic Dihydrofolate Reductase into Mitochondria in Vitro. Biochemical and Biophysical Research Communications, 1994, 201, 1059-1065.	2.1	4
61	Shift in pH-Rate Profile and Enhanced Discrimination between Dicarboxylic and Aromatic Substrates in Mitochondrial Aspartate Aminotransferase Y70H. Biochemistry, 1994, 33, 2757-2760.	2.5	10
62	Import of mutant forms of mitochondrial aspartate aminotransferase into isolated mitochondria. Archives of Biochemistry and Biophysics, 1992, 298, 532-537.	3.0	8
63	The in vitro-synthesized precursor and mature mitochondrial aspartate aminotransferase share the same import pathway in isolated mitochondria. Archives of Biochemistry and Biophysics, 1991, 290, 528-534.	3.0	8
64	Certain N-terminal peptides inhibit uptake of mature aspartate aminotransferase by isolated mitochondria. Biochemical and Biophysical Research Communications, 1990, 170, 609-615.	2.1	3
65	Fumarate permeation in rat liver mitochondria: Fumarate/malate and fumarate/phosphate translocators. Biochemical and Biophysical Research Communications, 1985, 132, 8-18.	2.1	18
66	Mechanisms of peroxidic oxygen transfer to organic substrates. Tetrahedron, 1984, 40, 2763-2771.	1.9	24
67	Yeast as a Model to Unravel New BRCA2 Functions in Cell Metabolism. Frontiers in Oncology, 0, 12, .	2.8	0