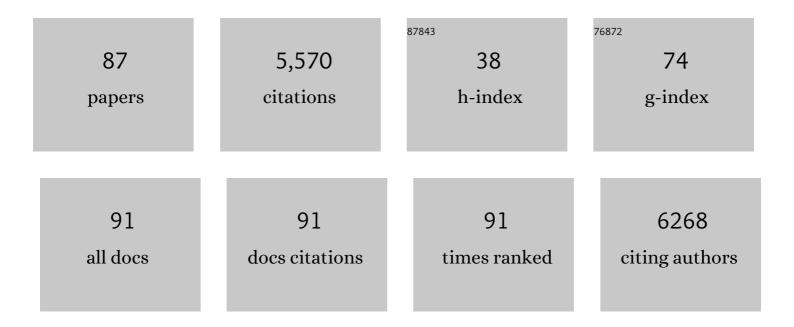
List of Publications by Year in descending order

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VIADIMID | CARAL

#	Article	lF	CITATIONS
1	Necrosis: a specific form of programmed cell death?. Experimental Cell Research, 2003, 283, 1-16.	1.2	605
2	Hsp70 Prevents Activation of Stress Kinases. Journal of Biological Chemistry, 1997, 272, 18033-18037.	1.6	473
3	Proteasome Inhibitors Activate Stress Kinases and Induce Hsp72. Journal of Biological Chemistry, 1998, 273, 6373-6379.	1.6	280
4	Protein-Damaging Stresses Activate c-Jun N-Terminal Kinase via Inhibition of Its Dephosphorylation: a Novel Pathway Controlled by HSP72. Molecular and Cellular Biology, 1999, 19, 2547-2555.	1.1	234
5	Role of Hsp70 in regulation of stress-kinase JNK: implications in apoptosis and aging. FEBS Letters, 1998, 438, 1-4.	1.3	215
6	Hsp70 in cancer: back to the future. Oncogene, 2015, 34, 4153-4161.	2.6	182
7	Hsp72 and Stress Kinase c-jun N-Terminal Kinase Regulate the Bid-Dependent Pathway in Tumor Necrosis Factor-Induced Apoptosis. Molecular and Cellular Biology, 2002, 22, 3415-3424.	1.1	158
8	Invited Review: Interplay between molecular chaperones and signaling pathways in survival of heat shock. Journal of Applied Physiology, 2002, 92, 1743-1748.	1.2	157
9	Hsp72-Mediated Suppression of c-Jun N-Terminal Kinase Is Implicated in Development of Tolerance to Caspase-Independent Cell Death. Molecular and Cellular Biology, 2000, 20, 6826-6836.	1.1	154
10	Hsp70–Bag3 Interactions Regulate Cancer-Related Signaling Networks. Cancer Research, 2014, 74, 4731-4740.	0.4	141
11	Targeting Heat Shock Response to Sensitize Cancer Cells to Proteasome and Hsp90 Inhibitors. Cancer Research, 2006, 66, 1783-1791.	0.4	140
12	Increased expression of the major heat shock protein Hsp72 in human prostate carcinoma cells is dispensable for their viability but confers resistance to a variety of anticancer agents. Oncogene, 2005, 24, 3328-3338.	2.6	126
13	Heat-shock transcription factor HSF1 has a critical role in human epidermal growth factor receptor-2-induced cellular transformation and tumorigenesis. Oncogene, 2010, 29, 5204-5213.	2.6	126
14	Hsp27 Modulates p53 Signaling and Suppresses Cellular Senescence. Cancer Research, 2007, 67, 11779-11788.	0.4	121
15	Mechanisms of Tumor Cell Necrosis. Current Pharmaceutical Design, 2010, 16, 56-68.	0.9	116
16	High Levels of Heat Shock Protein Hsp72 in Cancer Cells Suppress Default Senescence Pathways. Cancer Research, 2007, 67, 2373-2381.	0.4	107
17	Heat Shock Protein Hsp72 Controls Oncogene-Induced Senescence Pathways in Cancer Cells. Molecular and Cellular Biology, 2009, 29, 559-569.	1.1	105
18	Distinct hsp70 Domains Mediate Apoptosis-inducing Factor Release and Nuclear Accumulation. Journal of Biological Chemistry, 2006, 281, 7873-7880.	1.6	103

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19	Suppression of Stress Kinase JNK Is Involved in HSP72-mediated Protection of Myogenic Cells from Transient Energy Deprivation. Journal of Biological Chemistry, 2000, 275, 38088-38094.	1.6	101
20	Heat Shock Transcription Factor Hsf1 Is Involved in Tumor Progression via Regulation of Hypoxia-Inducible Factor 1 and RNA-Binding Protein HuR. Molecular and Cellular Biology, 2012, 32, 929-940.	1,1	99
21	Cell Death and Survival Assays. Methods in Molecular Biology, 2018, 1709, 107-127.	0.4	86
22	Heat shock protein Hsp72 plays an essential role in Her2-induced mammary tumorigenesis. Oncogene, 2011, 30, 2836-2845.	2.6	78
23	Triggering Aggresome Formation. Journal of Biological Chemistry, 2008, 283, 27575-27584.	1.6	75
24	The Function of HSP72 in Suppression of c-Jun N-terminal Kinase Activation Can Be Dissociated from Its Role in Prevention of Protein Damage. Journal of Biological Chemistry, 1999, 274, 20223-20228.	1.6	71
25	Necrosis is an active and controlled form of programmed cell death. Biochemistry (Moscow), 2002, 67, 387-408.	0.7	69
26	Resistance of Ehrlich tumor cells to apoptosis can be due to accumulation of heat shock proteins. FEBS Letters, 1995, 375, 21-26.	1.3	67
27	The heat shock transcription factor Hsf1 is downregulated in DNA damage–associated senescence, contributing to the maintenance of senescence phenotype. Aging Cell, 2012, 11, 617-627.	3.0	66
28	Regulation of Necrosis of H9c2 Myogenic Cells upon Transient Energy Deprivation. Journal of Biological Chemistry, 2003, 278, 50483-50496.	1.6	64
29	Proteasome Failure Promotes Positioning of Lysosomes around the Aggresome via Local Block of Microtubule-Dependent Transport. Molecular and Cellular Biology, 2014, 34, 1336-1348.	1.1	62
30	Heat Shock-Induced Accumulation of 70-kDa Stress Protein (HSP70) Can Protect ATP-Depleted Tumor Cells from Necrosis. Experimental Cell Research, 1995, 217, 15-21.	1.2	58
31	Inactivation of Dual-Specificity Phosphatases Is Involved in the Regulation of Extracellular Signal-Regulated Kinases by Heat Shock and Hsp72. Molecular and Cellular Biology, 2003, 23, 3813-3824.	1.1	57
32	Rise in heat-shock protein level confers tolerance to energy deprivation. FEBS Letters, 1993, 327, 247-250.	1.3	56
33	Intracellular Aggregation of Polypeptides with Expanded Polyglutamine Domain Is Stimulated by Stress-Activated Kinase Mekk1. Journal of Cell Biology, 2001, 153, 851-864.	2.3	54
34	Hsp90 inhibitors as promising agents for radiotherapy. Journal of Molecular Medicine, 2010, 88, 241-247.	1.7	52
35	Distinct effects of heat shock and ATP depletion on distribution and isoform patterns of human Hsp27 in endothelial cells. FEBS Letters, 1996, 392, 100-104.	1.3	51
36	Protein aggregation as primary and characteristic cell reaction to various stresses. Experientia, 1993, 49, 706-710.	1.2	46

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37	HSP72 can protect cells from heat-induced apoptosis by accelerating the inactivation of stress kinase JNK. Cell Stress and Chaperones, 2000, 5, 139.	1.2	45
38	ATPase activity of the heat shock protein Hsp72 is dispensable for its effects on dephosphorylation of stress kinase JNK and on heat-induced apoptosis. FEBS Letters, 1999, 461, 73-76.	1.3	43
39	Heat-shock proteins maintain the viability of ATP-deprived cells: what is the mechanism?. Trends in Cell Biology, 1994, 4, 193-196.	3.6	38
40	Pilot study of p62 DNA vaccine in dogs with mammary tumors. Oncotarget, 2014, 5, 12803-12810.	0.8	37
41	Broad-spectrum anti-tumor and anti-metastatic DNA vaccine based on p62-encoding vector. Oncotarget, 2013, 4, 1829-1835.	0.8	36
42	Molecular chaperones regulate p53 and suppress senescence programs. FEBS Letters, 2007, 581, 3711-3715.	1.3	35
43	Oncogenes induce senescence with incomplete growth arrest and suppress the DNA damage response in immortalized cells. Aging Cell, 2011, 10, 949-961.	3.0	35
44	Heat Shock Proteins and Cytoprotection. , 1997, , .		32
45	Anticancer Effects of Targeting Hsp70 in Tumor Stromal Cells. Cancer Research, 2016, 76, 5926-5932.	0.4	31
46	Feasibility Analysis of p62 (SQSTM1)—Encoding DNA Vaccine as a Novel Cancer Immunotherapy. International Reviews of Immunology, 2014, 33, 375-382.	1.5	30
47	Plasmid DNA-coding p62 as a bone effective anti-inflammatory/anabolic agent. Oncotarget, 2015, 6, 3590-3599.	0.8	29
48	Induction of Heat-Shock Protein Synthesis and Thermotolerance in EL-4 Ascites Tumor Cells by Transient ATP Depletion after Ischemic Stress. Experimental and Molecular Pathology, 1994, 60, 88-99.	0.9	27
49	p62 /SQSTM1 coding plasmid prevents age related macular degeneration in a rat model. Aging, 2018, 10, 2136-2147.	1.4	27
50	Toxicity of Influenza A Virus Matrix Protein 2 for Mammalian Cells is Associated with its Intrinsic Proton-Channeling Activity. Cell Cycle, 2007, 6, 2043-2047.	1.3	25
51	HSP72 depletion suppresses γH2AX activation by genotoxic stresses via p53/p21 signaling. Oncogene, 2010, 29, 1952-1962.	2.6	24
52	Safety and efficacy of p62 DNA vaccine ELENAGEN in a first-in-human trial in patients with advanced solid tumors. Oncotarget, 2017, 8, 53730-53739.	0.8	24
53	Association of blebbing with assembly of cytoskeletal proteins in ATP-depleted EL-4 ascites tumour cells. Tissue and Cell, 1992, 24, 171-177.	1.0	23
54	Tumor cell resistance to energy deprivation and hyperthermia can be determined by the actin skeleton stability. Cancer Letters, 1993, 70, 25-31.	3.2	23

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55	Stress-Induced Insolubilization of Certain Proteins in Ascites Tumor Cells. Archives of Biochemistry and Biophysics, 1994, 309, 247-253.	1.4	23
56	HSP70s in Breast Cancer: Promoters of Tumorigenesis and Potential Targets/Tools for Therapy. Cells, 2021, 10, 3446.	1.8	21
57	Immunology of Apoptosis and Necrosis. Biochemistry (Moscow), 2005, 70, 1310-1320.	0.7	18
58	Prime-boost vaccination with a combination of proteosome-degradable and wild-type forms of two influenza proteins leads to augmented CTL response. Vaccine, 2008, 26, 2177-2185.	1.7	16
59	Triggering Senescence Programs Suppresses Chk1 Kinase and Sensitizes Cells To Genotoxic Stresses. Cancer Research, 2008, 68, 1834-1842.	0.4	16
60	Involvement of Ca2+and cGMP in bacterial taxis. FEMS Microbiology Letters, 1985, 28, 259-263.	0.7	15
61	The role of Bag3 in cell signaling. Journal of Cellular Biochemistry, 2022, 123, 43-53.	1.2	15
62	Adaptation of ehrlich ascites carcinoma cells to energy deprivation in vivo can be associated with heat shock protein accumulation. Journal of Cellular Physiology, 1995, 165, 1-6.	2.0	14
63	Determination of Cell Survival or Death. Methods in Molecular Biology, 2011, 787, 231-244.	0.4	13
64	P62 plasmid can alleviate diet-induced obesity and metabolic dysfunctions. Oncotarget, 2017, 8, 56030-56040.	0.8	13
65	Natural hidden antibodies reacting with DNA or cardiolipin bind to thymocytes and evoke their death. FEBS Letters, 1997, 413, 231-235.	1.3	11
66	Inhibition of uncoupled respiration in tumor cells. FEBS Letters, 1993, 329, 67-71.	1.3	9
67	Inhibition of Influenza M2-Induced Cell Death Alleviates Its Negative Contribution to Vaccination Efficiency. PLoS ONE, 2008, 3, e1417.	1.1	8
68	Response of a chemo-resistant triple-negative breast cancer patient to a combination of p62-encoding plasmid, Elenagen, and CMF chemotherapy. Oncotarget, 2020, 11, 294-299.	0.8	8
69	Redox-sensing is the basis of photophobic responses in cyanobacteria. FEMS Microbiology Letters, 1985, 27, 351-356.	0.7	6
70	Mitochondrial ATP hydrolysis and ATP depletion in thymocytes and Ehrlich ascites carcinoma cells. FEBS Letters, 1994, 337, 56-59.	1.3	6
71	Involvement of Heat Shock Proteins in Protection of Tumor Cells from Genotoxic Stresses. , 2007, , 169-190.		6
72	Proteotoxicity is not the reason for the dependence of cancer cells on the major chaperone Hsp70. Cell Cycle, 2014, 13, 2306-2310.	1.3	6

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73	P62/SQSTM1 beyond Autophagy: Physiological Role and Therapeutic Applications in Laboratory and Domestic Animals. Life, 2022, 12, 539.	1.1	6
74	Multiple Thermometers in Mammalian Cells: Why Do Cells from Homeothermic Organisms Need to Measure Temperature?. Science Signaling, 2006, 2006, pe16-pe16.	1.6	5
75	p62-DNA-encoding plasmid reverts tumor grade, changes tumor stroma, and enhances anticancer immunity. Aging, 2019, 11, 10711-10722.	1.4	5
76	Glucose decreases respiratory control ratio in EL-4 tumor cells. FEBS Letters, 1992, 313, 126-128.	1.3	3
77	Hsp72 and Cell Signalling. , 2005, , 144-159.		2
78	The proteosomal degradation of fusion proteins cannot be predicted from the proteosome susceptibility of their individual components. Protein Science, 2008, 17, 1077-1085.	3.1	2
79	P62/SQSTM1 enhances osteogenesis and attenuates inflammatory signals in bone marrow microenvironment. General and Comparative Endocrinology, 2022, 320, 114009.	0.8	2
80	"Proteotoxicity―of ATP Depletion: Disruption of the Cytoskeleton, Protein Aggregation and Involvement of Molecular Chaperones. , 1997, , 49-83.		1
81	Corrigendum to "Molecular chaperones regulate p53 and suppress senescence programs―[FEBS Lett. 581 (2007) 3711-3715]. FEBS Letters, 2007, 581, 5732-5732.	1.3	0
82	Effective Expression of Recombinant Cytotoxic Protein via Its Attachment to a Polyglutamine Domain. OMICS A Journal of Integrative Biology, 2009, 13, 211-217.	1.0	0
83	Mechanisms of HSP-Mediated Protection from Ischemia-Induced Apoptosis. , 1997, , 205-220.		0
84	Involvement of Heat Shock Proteins in Protection of Various Normal and Tumor Cells from Ischemic Insult. , 1997, , 141-175.		0
85	ATP Depletion as Inducer of Heat Shock Protein Expression. , 1997, , 85-119.		0
86	ATP Homeostasis, Ionic Balance and Cell Viability. , 1997, , 21-47.		0
87	What Are the Mechanisms of Heat Shock Protein-Mediated Cytoprotection Under ATP Deprivation?. , 1997, , 177-204.		0