Vladimir L Gabai

List of Publications by Year
in descending order

87 papers	5,570 citations	87888 38 h-index	76900 74 g-index
91 all docs	91 docs citations	91 times ranked	6268 citing authors

#	Article	IF	Citations
1	The role of Bag3 in cell signaling. Journal of Cellular Biochemistry, 2022, 123, 43-53.	2.6	15
2	P62/SQSTM1 beyond Autophagy: Physiological Role and Therapeutic Applications in Laboratory and Domestic Animals. Life, 2022, 12, 539.	2.4	6
3	P62/SQSTM1 enhances osteogenesis and attenuates inflammatory signals in bone marrow microenvironment. General and Comparative Endocrinology, 2022, 320, 114009.	1.8	2
4	HSP70s in Breast Cancer: Promoters of Tumorigenesis and Potential Targets/Tools for Therapy. Cells, 2021, 10, 3446.	4.1	21
5	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.	1.8	8
6	p62-DNA-encoding plasmid reverts tumor grade, changes tumor stroma, and enhances anticancer immunity. Aging, 2019, 11, 10711-10722.	3.1	5
7	Cell Death and Survival Assays. Methods in Molecular Biology, 2018, 1709, 107-127.	0.9	86
8	p62 /SQSTM1 coding plasmid prevents age related macular degeneration in a rat model. Aging, 2018, 10, 2136-2147.	3.1	27
9	P62 plasmid can alleviate diet-induced obesity and metabolic dysfunctions. Oncotarget, 2017, 8, 56030-56040.	1.8	13
10	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.	1.8	24
11	Anticancer Effects of Targeting Hsp70 in Tumor Stromal Cells. Cancer Research, 2016, 76, 5926-5932.	0.9	31
12	Hsp70 in cancer: back to the future. Oncogene, 2015, 34, 4153-4161.	5.9	182
13	Plasmid DNA-coding p62 as a bone effective anti-inflammatory/anabolic agent. Oncotarget, 2015, 6, 3590-3599.	1.8	29
14	Proteotoxicity is not the reason for the dependence of cancer cells on the major chaperone Hsp70. Cell Cycle, 2014, 13, 2306-2310.	2.6	6
15	Proteasome Failure Promotes Positioning of Lysosomes around the Aggresome via Local Block of Microtubule-Dependent Transport. Molecular and Cellular Biology, 2014, 34, 1336-1348.	2.3	62
16	Feasibility Analysis of p62 (SQSTM1)â€"Encoding DNA Vaccine as a Novel Cancer Immunotherapy. International Reviews of Immunology, 2014, 33, 375-382.	3.3	30
17	Hsp70â€"Bag3 Interactions Regulate Cancer-Related Signaling Networks. Cancer Research, 2014, 74, 4731-4740.	0.9	141
18	Pilot study of p62 DNA vaccine in dogs with mammary tumors. Oncotarget, 2014, 5, 12803-12810.	1.8	37

#	Article	IF	Citations
19	Broad-spectrum anti-tumor and anti-metastatic DNA vaccine based on p62-encoding vector. Oncotarget, 2013, 4, 1829-1835.	1.8	36
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.	2.3	99
21	The heat shock transcription factor Hsf1 is downregulated in DNA damageâ€“associated senescence, contributing to the maintenance of senescence phenotype. Aging Cell, 2012, 11, 617-627.	6.7	66
22	Determination of Cell Survival or Death. Methods in Molecular Biology, 2011, 787, 231-244.	0.9	13
23	Oncogenes induce senescence with incomplete growth arrest and suppress the DNA damage response in immortalized cells. Aging Cell, 2011, 10, 949-961.	6.7	35
24	Heat shock protein Hsp72 plays an essential role in Her2-induced mammary tumorigenesis. Oncogene, 2011, 30, 2836-2845.	5.9	78
25	Hsp90 inhibitors as promising agents for radiotherapy. Journal of Molecular Medicine, 2010, 88, 241-247.	3.9	52
26	HSP72 depletion suppresses Î³H2AX activation by genotoxic stresses via p53/p21 signaling. Oncogene, 2010, 29, 1952-1962.	5.9	24
27	Mechanisms of Tumor Cell Necrosis. Current Pharmaceutical Design, 2010, 16, 56-68.	1.9	116
28	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.	5.9	126
29	Heat Shock Protein Hsp72 Controls Oncogene-Induced Senescence Pathways in Cancer Cells. Molecular and Cellular Biology, 2009, 29, 559-569.	2.3	105
30	Effective Expression of Recombinant Cytotoxic Protein via Its Attachment to a Polyglutamine Domain. OMICS A Journal of Integrative Biology, 2009, 13, 211-217.	2.0	0
31	The proteosomal degradation of fusion proteins cannot be predicted from the proteosome susceptibility of their individual components. Protein Science, 2008, 17, 1077-1085.	7.6	2
32	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.	3.8	16
33	Triggering Aggresome Formation. Journal of Biological Chemistry, 2008, 283, 27575-27584.	3.4	75
34	Triggering Senescence Programs Suppresses Chk1 Kinase and Sensitizes Cells To Genotoxic Stresses. Cancer Research, 2008, 68, 1834-1842.	0.9	16
35	Inhibition of Influenza M2-Induced Cell Death Alleviates Its Negative Contribution to Vaccination Efficiency. PLoS ONE, 2008, 3, e1417.	2.5	8
36	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.	2.6	25

#	Article	IF	Citations
37	Hsp27 Modulates p53 Signaling and Suppresses Cellular Senescence. Cancer Research, 2007, 67, 11779-11788.	0.9	121
38	High Levels of Heat Shock Protein Hsp72 in Cancer Cells Suppress Default Senescence Pathways. Cancer Research, 2007, 67, 2373-2381.	0.9	107
39	Involvement of Heat Shock Proteins in Protection of Tumor Cells from Genotoxic Stresses. , 2007, , 169-190.		6
40	Molecular chaperones regulate p53 and suppress senescence programs. FEBS Letters, 2007, 581, 3711-3715.	2.8	35
41	Corrigendum to â€œMolecular chaperones regulate p53 and suppress senescence programsâ€•[FEBS Lett. 581 (2007) 3711-3715]. FEBS Letters, 2007, 581, 5732-5732.	2.8	0
42	Multiple Thermometers in Mammalian Cells: Why Do Cells from Homeothermic Organisms Need to Measure Temperature?. Science Signaling, 2006, 2006, pe16-pe16.	3.6	5
43	Targeting Heat Shock Response to Sensitize Cancer Cells to Proteasome and Hsp90 Inhibitors. Cancer Research, 2006, 66, 1783-1791.	0.9	140
44	Distinct hsp70 Domains Mediate Apoptosis-inducing Factor Release and Nuclear Accumulation. Journal of Biological Chemistry, 2006, 281, 7873-7880.	3.4	103
45	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.	5.9	126
46	Immunology of Apoptosis and Necrosis. Biochemistry (Moscow), 2005, 70, 1310-1320.	1.5	18
47	Hsp72 and Cell Signalling. , 2005, , 144-159.		2
48	Necrosis: a specific form of programmed cell death?. Experimental Cell Research, 2003, 283, 1-16.	2.6	605
49	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.	2.3	57
50	Regulation of Necrosis of H9c2 Myogenic Cells upon Transient Energy Deprivation. Journal of Biological Chemistry, 2003, 278, 50483-50496.	3.4	64
51	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.	2.3	158
52	Invited Review: Interplay between molecular chaperones and signaling pathways in survival of heat shock. Journal of Applied Physiology, 2002, 92, 1743-1748.	2.5	157
53	Necrosis is an active and controlled form of programmed cell death. Biochemistry (Moscow), 2002, 67, 387-408.	1.5	69
54	Intracellular Aggregation of Polypeptides with Expanded Polyglutamine Domain Is Stimulated by Stress-Activated Kinase Mekk1. Journal of Cell Biology, 2001, 153, 851-864.	5.2	54

#	Article	IF	Citations
55	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.	3.4	101
56	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.	2.3	154
57	HSP72 can protect cells from heat-induced apoptosis by accelerating the inactivation of stress kinase JNK. Cell Stress and Chaperones, 2000, 5, 139.	2.9	45
58	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.	3.4	71
59	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.	2.8	43
60	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.	2.3	234
61	Role of Hsp70 in regulation of stress-kinase JNK: implications in apoptosis and aging. FEBS Letters, 1998, 438, 1-4.	2.8	215
62	Proteasome Inhibitors Activate Stress Kinases and Induce Hsp72. Journal of Biological Chemistry, 1998, 273, 6373-6379.	3.4	280
63	Hsp70 Prevents Activation of Stress Kinases. Journal of Biological Chemistry, 1997, 272, 18033-18037.	3.4	473
64	Natural hidden antibodies reacting with DNA or cardiolipin bind to thymocytes and evoke their death. FEBS Letters, 1997, 413, 231-235.	2.8	11
65	Heat Shock Proteins and Cytoprotection. , 1997, , .		32
66	Mechanisms of HSP-Mediated Protection from Ischemia-Induced Apoptosis. , 1997, , 205-220.		0
67	Involvement of Heat Shock Proteins in Protection of Various Normal and Tumor Cells from Ischemic Insult. , 1997, , 141-175.		0
68	â€œProteotoxicityâ€•of ATP Depletion: Disruption of the Cytoskeleton, Protein Aggregation and Involvement of Molecular Chaperones. , 1997, , 49-83.		1
69	ATP Depletion as Inducer of Heat Shock Protein Expression. , 1997, , 85-119.		0
70	ATP Homeostasis, Ionic Balance and Cell Viability. , 1997, , 21-47.		0
71	What Are the Mechanisms of Heat Shock Protein-Mediated Cytoprotection Under ATP Deprivation?. , 1997, , 177-204.		0
72	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.	2.8	51

#	Article	IF	Citations
73	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.	4.1	14
74	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.	2.6	58
75	Resistance of Ehrlich tumor cells to apoptosis can be due to accumulation of heat shock proteins. FEBS Letters, 1995, 375, 21-26.	2.8	67
76	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.	2.1	27
77	Heat-shock proteins maintain the viability of ATP-deprived cells: what is the mechanism?. Trends in Cell Biology, 1994, 4, 193-196.	7.9	38
78	Stress-Induced Insolubilization of Certain Proteins in Ascites Tumor Cells. Archives of Biochemistry and Biophysics, 1994, 309, 247-253.	3.0	23
79	Mitochondrial ATP hydrolysis and ATP depletion in thymocytes and Ehrlich ascites carcinoma cells. FEBS Letters, 1994, 337, 56-59.	2.8	6
80	Protein aggregation as primary and characteristic cell reaction to various stresses. Experientia, 1993, 49, 706-710.	1.2	46
81	Inhibition of uncoupled respiration in tumor cells. FEBS Letters, 1993, 329, 67-71.	2.8	9
82	Rise in heat-shock protein level confers tolerance to energy deprivation. FEBS Letters, 1993, 327, 247-250.	2.8	56
83	Tumor cell resistance to energy deprivation and hyperthermia can be determined by the actin skeleton stability. Cancer Letters, 1993, 70, 25-31.	7.2	23
84	Association of blebbing with assembly of cytoskeletal proteins in ATP-depleted EL-4 ascites tumour cells. Tissue and Cell, 1992, 24, 171-177.	2.2	23
85	Glucose decreases respiratory control ratio in EL-4 tumor cells. FEBS Letters, 1992, 313, 126-128.	2.8	3
86	Redox-sensing is the basis of photophobic responses in cyanobacteria. FEMS Microbiology Letters, 1985, 27, 351-356.	1.8	6
87	Involvement of Ca2+and cGMP in bacterial taxis. FEMS Microbiology Letters, 1985, 28, 259-263.	1.8	15