

Antonis E Koromilas

List of Publications by Year in descending order

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75
papers

4,293
citations

101543

36
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114465

63
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76
all docs

76
docs citations

76
times ranked

5980
citing authors

#	ARTICLE	IF	CITATIONS
1	The integrated stress response in the induction of mutant <i>KRAS</i> lung carcinogenesis: Mechanistic insights and therapeutic implications. <i>BioEssays</i> , 2022, 44, e2200026.	2.5	0
2	The integrated stress response is tumorigenic and constitutes a therapeutic liability in <i>KRAS</i> -driven lung cancer. <i>Nature Communications</i> , 2021, 12, 4651.	12.8	22
3	Translational control of breast cancer plasticity. <i>Nature Communications</i> , 2020, 11, 2498.	12.8	80
4	Detection of Lung Tumor Progression in Mice by Ultrasound Imaging. <i>Journal of Visualized Experiments</i> , 2020, , .	0.3	2
5	An integrated stress response via PKR suppresses HER2+ cancers and improves trastuzumab therapy. <i>Nature Communications</i> , 2019, 10, 2139.	12.8	46
6	M(en)TORship lessons on life and death by the integrated stress response. <i>Biochimica Et Biophysica Acta - General Subjects</i> , 2019, 1863, 644-649.	2.4	19
7	Downregulation of PERK activity and eIF2 γ serine 51 phosphorylation by mTOR complex 1 elicits pro-oxidant and pro-death effects in tuberous sclerosis-deficient cells. <i>Cell Death and Disease</i> , 2018, 9, 254.	6.3	10
8	Defective interplay between mTORC1 activity and endoplasmic reticulum stress-unfolded protein response in uremic vascular calcification. <i>American Journal of Physiology - Renal Physiology</i> , 2018, 314, F1046-F1061.	2.7	32
9	The Shc1 adaptor simultaneously balances Stat1 and Stat3 activity to promote breast cancer immune suppression. <i>Nature Communications</i> , 2017, 8, 14638.	12.8	52
10	Regulation of ULK1 Expression and Autophagy by STAT1. <i>Journal of Biological Chemistry</i> , 2017, 292, 1899-1909.	3.4	24
11	Protein Kinase R Mediates the Inflammatory Response Induced by Hyperosmotic Stress. <i>Molecular and Cellular Biology</i> , 2017, 37, .	2.3	14
12	A Unique ISR Program Determines Cellular Responses to Chronic Stress. <i>Molecular Cell</i> , 2017, 68, 885-900.e6.	9.7	135
13	STAT1 Promotes <i>KRAS</i> Colon Tumor Growth and Susceptibility to Pharmacological Inhibition of Translation Initiation Factor eIF4A. <i>Molecular Cancer Therapeutics</i> , 2016, 15, 3055-3063.	4.1	17
14	mTORC1 and CK2 coordinate ternary and eIF4F complex assembly. <i>Nature Communications</i> , 2016, 7, 11127.	12.8	75
15	STAT1-mediated translational control in tumor suppression and antitumor therapies. <i>Molecular and Cellular Oncology</i> , 2016, 3, e1055049.	0.7	7
16	AMP Kinase Activation Alters Oxidant-Induced Stress Granule Assembly by Modulating Cell Signaling and Microtubule Organization. <i>Molecular Pharmacology</i> , 2016, 90, 460-468.	2.3	27
17	Phosphorylation of eIF2 γ Is a Translational Control Mechanism Regulating Muscle Stem Cell Quiescence and Self-Renewal. <i>Cell Stem Cell</i> , 2016, 18, 79-90.	11.1	206
18	The eIF2 γ serine 51 phosphorylation-ATF4 arm promotes HIPPO signaling and cell death under oxidative stress. <i>Oncotarget</i> , 2016, 7, 51044-51058.	1.8	26

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19	Increased phosphorylation of eIF2 γ in chronic myeloid leukemia cells stimulates secretion of matrix modifying enzymes. <i>Oncotarget</i> , 2016, 7, 79706-79721.	1.8	16
20	[Pemetrexed + Sorafenib] lethality is increased by inhibition of ERBB1/2/3-PI3K-NF κ B compensatory survival signaling. <i>Oncotarget</i> , 2016, 7, 23608-23632.	1.8	27
21	mTORC2 Balances AKT Activation and eIF2 γ Serine 51 Phosphorylation to Promote Survival under Stress. <i>Molecular Cancer Research</i> , 2015, 13, 1377-1388.	3.4	35
22	Stat1 stimulates cap-independent mRNA translation to inhibit cell proliferation and promote survival in response to antitumor drugs. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, E2149-55.	7.1	8
23	Roles of the translation initiation factor eIF2 γ serine 51 phosphorylation in cancer formation and treatment. <i>Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms</i> , 2015, 1849, 871-880.	1.9	102
24	Evidence for eIF2 γ phosphorylation-independent effects of GSK2656157, a novel catalytic inhibitor of PERK with clinical implications. <i>Cell Cycle</i> , 2014, 13, 801-806.	2.6	38
25	Translational Control during Endoplasmic Reticulum Stress beyond Phosphorylation of the Translation Initiation Factor eIF2 γ . <i>Journal of Biological Chemistry</i> , 2014, 289, 12593-12611.	3.4	120
26	Control of oncogenesis by eIF2 γ phosphorylation: implications in PTEN and PI3K \rightarrow Akt signaling and tumor treatment. <i>Future Oncology</i> , 2013, 9, 1005-1015.	2.4	26
27	A Self-defeating Anabolic Program Leads to β -Cell Apoptosis in Endoplasmic Reticulum Stress-induced Diabetes via Regulation of Amino Acid Flux. <i>Journal of Biological Chemistry</i> , 2013, 288, 17202-17213.	3.4	105
28	The tumor suppressor function of STAT1 in breast cancer. <i>Jak-stat</i> , 2013, 2, e23353.	2.2	68
29	eIF2 γ phosphorylation bypasses premature senescence caused by oxidative stress and pro-oxidant antitumor therapies. <i>Aging</i> , 2013, 5, 884-901.	3.1	35
30	The Akt \rightarrow mTOR axis determines cell fate through the regulation of eIF2 α phosphorylation pathway.. <i>FASEB Journal</i> , 2013, 27, 835.9.	0.5	0
31	The PERK-eIF2 γ phosphorylation arm is a pro-survival pathway of BCR-ABL signaling and confers resistance to imatinib treatment in chronic myeloid leukemia cells. <i>Cell Cycle</i> , 2012, 11, 4069-4078.	2.6	58
32	Alternative ferritin mRNA translation via internal initiation. <i>Rna</i> , 2012, 18, 547-556.	3.5	15
33	Protein Tyrosine Phosphatase 1B Deficiency Potentiates PERK/eIF2 γ Signaling in Brown Adipocytes. <i>PLoS ONE</i> , 2012, 7, e34412.	2.5	46
34	Development of transgenic mice expressing a conditionally active form of the eIF2 γ kinase PKR. <i>Genesis</i> , 2011, 49, 743-749.	1.6	2
35	Negative regulation of mTORC2 by glycogen synthase kinase-3 β : an adaptive process to stress with an anticancer therapeutic potential?. <i>Future Oncology</i> , 2011, 7, 845-848.	2.4	4
36	Stat1 is a suppressor of ErbB2/Neu-mediated cellular transformation and mouse mammary gland tumor formation. <i>Cell Cycle</i> , 2011, 10, 794-804.	2.6	60

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37	Akt Determines Cell Fate Through Inhibition of the PERK-eIF2 α Phosphorylation Pathway. <i>Science Signaling</i> , 2011, 4, ra62.	3.6	102
38	eIF2 α Kinase PKR Modulates the Hypoxic Response by Stat3-Dependent Transcriptional Suppression of HIF-1 α . <i>Cancer Research</i> , 2010, 70, 7820-7829.	0.9	44
39	STAT1 Represses <i>Skp2</i> Gene Transcription to Promote p27Kip1 Stabilization in Ras-Transformed Cells. <i>Molecular Cancer Research</i> , 2010, 8, 798-805.	3.4	23
40	Uncovering the PKR pathway's potential for treatment of tumors. <i>Future Oncology</i> , 2010, 6, 643-645.	2.4	9
41	Phosphorylation of eIF2 α at Serine 51 Is an Important Determinant of Cell Survival and Adaptation to Glucose Deficiency. <i>Molecular Biology of the Cell</i> , 2010, 21, 3220-3231.	2.1	100
42	HDAC pharmacological inhibition promotes cell death through the eIF2 α kinases PKR and GCN2. <i>Aging</i> , 2010, 2, 669-677.	3.1	13
43	Tumor Suppression by PTEN Requires the Activation of the PKR-eIF2 α Phosphorylation Pathway. <i>Science Signaling</i> , 2009, 2, ra85.	3.6	72
44	Stat1 is an inhibitor of Ras-MAPK signaling and Rho small GTPase expression with implications in the transcriptional signature of Ras transformed cells. <i>Cell Cycle</i> , 2009, 8, 2070-2079.	2.6	24
45	PKR is not a universal target of tumor suppressor p53 in response to genotoxic stress. <i>Cell Cycle</i> , 2009, 8, 3606-3607.	2.6	5
46	The eIF2 α kinases inhibit vesicular stomatitis virus replication independently of eIF2 phosphorylation. <i>Cell Cycle</i> , 2008, 7, 2346-2351.	2.6	44
47	PERK and PKR: Old kinases learn new tricks. <i>Cell Cycle</i> , 2008, 7, 1146-1150.	2.6	85
48	Modulation of the Eukaryotic Initiation Factor 2 α -Subunit Kinase PERK by Tyrosine Phosphorylation. <i>Journal of Biological Chemistry</i> , 2008, 283, 469-475.	3.4	60
49	PKR and PKR-like Endoplasmic Reticulum Kinase Induce the Proteasome-dependent Degradation of Cyclin D1 via a Mechanism Requiring Eukaryotic Initiation Factor 2 α Phosphorylation. <i>Journal of Biological Chemistry</i> , 2008, 283, 3097-3108.	3.4	82
50	Stat1 Phosphorylation Determines Ras Oncogenicity by Regulating p27Kip1. <i>PLoS ONE</i> , 2008, 3, e3476.	2.5	27
51	The eIF2 α Kinases PERK and PKR Activate Glycogen Synthase Kinase 3 to Promote the Proteasomal Degradation of p53. <i>Journal of Biological Chemistry</i> , 2007, 282, 31675-31687.	3.4	99
52	A Novel Function of eIF2 α Kinases as Inducers of the Phosphoinositide-3 Kinase Signaling Pathway. <i>Molecular Biology of the Cell</i> , 2007, 18, 3635-3644.	2.1	79
53	Interferons induce tyrosine phosphorylation of the eIF2 α kinase PKR through activation of Jak1 and Tyk2. <i>EMBO Reports</i> , 2007, 8, 265-270.	4.5	38
54	Initiation of Protein Synthesis by Hepatitis C Virus Is Refractory to Reduced eIF2 α GTP \cdot Met-tRNAiMet Ternary Complex Availability. <i>Molecular Biology of the Cell</i> , 2006, 17, 4632-4644.	2.1	114

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55	The Catalytic Activity of the Eukaryotic Initiation Factor-2 Kinase PKR Is Required to Negatively Regulate Stat1 and Stat3 via Activation of the T-cell Protein-tyrosine Phosphatase. <i>Journal of Biological Chemistry</i> , 2006, 281, 9439-9449.	3.4	35
56	Tyrosine phosphorylation acts as a molecular switch to full-scale activation of the eIF2 RNA-dependent protein kinase. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2006, 103, 63-68.	7.1	70
57	The eIF2 kinase PKR is a negative regulator of Stat1 and Stat3. <i>FASEB Journal</i> , 2006, 20, A496.	0.5	2
58	Endoplasmic Reticulum Stress Accelerates p53 Degradation by the Cooperative Actions of Hdm2 and Glycogen Synthase Kinase 3. <i>Molecular and Cellular Biology</i> , 2005, 25, 9392-9405.	2.3	99
59	Endoplasmic reticulum stress induces p53 cytoplasmic localization and prevents p53-dependent apoptosis by a pathway involving glycogen synthase kinase-3. <i>Genes and Development</i> , 2004, 18, 261-277.	5.9	232
60	Control of Tumor Suppressor p53 Function by Endoplasmic Reticulum Stress. <i>Cell Cycle</i> , 2004, 3, 565-568.	2.6	18
61	Control of Subunit of Eukaryotic Translation Initiation Factor 2 (eIF2) Phosphorylation by the Human Papillomavirus Type 18 E6 Oncoprotein: Implications for eIF2-Dependent Gene Expression and Cell Death. <i>Molecular and Cellular Biology</i> , 2004, 24, 3415-3429.	2.3	93
62	Resistance to Vesicular Stomatitis Virus Infection Requires a Functional Cross Talk between the Eukaryotic Translation Initiation Factor 2 Kinases PERK and PKR. <i>Journal of Virology</i> , 2004, 78, 12747-12761.	3.4	82
63	Ubiquitination and proteasome degradation of the E6 proteins of human papillomavirus types 11 and 18. <i>Journal of General Virology</i> , 2004, 85, 1419-1426.	2.9	27
64	Control of tumor suppressor p53 function by endoplasmic reticulum stress. <i>Cell Cycle</i> , 2004, 3, 567-70.	2.6	8
65	Functional Characterization of pkr Gene Products Expressed in Cells from Mice with a Targeted Deletion of the N terminus or C terminus Domain of PKR. <i>Journal of Biological Chemistry</i> , 2002, 277, 38364-38372.	3.4	66
66	Activation of the IKK complex by double-stranded RNA-binding defective and catalytic inactive mutants of the interferon-inducible protein kinase PKR. <i>Oncogene</i> , 2001, 20, 1900-1912.	5.9	61
67	Induction of PG G/H Synthase-2 in Bovine Myometrial Cells by Interferon- β , Requires the Activation of the p38 MAPK Pathway. <i>Endocrinology</i> , 2001, 142, 5107-5115.	2.8	12
68	Dominant Negative Function by an Alternatively Spliced Form of the Interferon-inducible Protein Kinase PKR. <i>Journal of Biological Chemistry</i> , 2001, 276, 13881-13890.	3.4	43
69	Enhanced Antiviral and Antiproliferative Properties of a STAT1 Mutant Unable to Interact with the Protein Kinase PKR. <i>Journal of Biological Chemistry</i> , 2001, 276, 13727-13737.	3.4	25
70	A diminished activation capacity of the interferon-inducible protein kinase PKR in human T lymphocytes. <i>FEBS Journal</i> , 2000, 267, 1598-1606.	0.2	16
71	Characterization of Transgenic Mice with Targeted Disruption of the Catalytic Domain of the Double-stranded RNA-dependent Protein Kinase, PKR. <i>Journal of Biological Chemistry</i> , 1999, 274, 5953-5962.	3.4	211
72	The double-stranded RNA activated protein kinase PKR physically associates with the tumor suppressor p53 protein and phosphorylates human p53 on serine 392 in vitro. <i>Oncogene</i> , 1999, 18, 2690-2702.	5.9	207

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73	The human papilloma virus (HPV)-18 E6 oncoprotein physically associates with Tyk2 and impairs Jak-STAT activation by interferon- β . <i>Oncogene</i> , 1999, 18, 5727-5737.	5.9	255
74	Upregulation of STAT1 protein in cells lacking or expressing mutants of the double-stranded RNA-dependent protein kinase PKR. <i>FEBS Journal</i> , 1999, 262, 149-154.	0.2	18
75	Double-Stranded-RNA-Activated Protein Kinase PKR Enhances Transcriptional Activation by Tumor Suppressor p53. <i>Molecular and Cellular Biology</i> , 1999, 19, 2475-2484.	2.3	134