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List of Publications by Year in descending order

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59
papers

3,840
citations

136950

32
h-index

133252

59
g-index

61
all docs

61
docs citations

61
times ranked

4732
citing authors

#	ARTICLE	IF	CITATIONS
1	The bioenergetic signature of cancer: a marker of tumor progression. <i>Cancer Research</i> , 2002, 62, 6674-81.	0.9	317
2	Overexpression of Akt converts radial growth melanoma to vertical growth melanoma. <i>Journal of Clinical Investigation</i> , 2007, 117, 719-729.	8.2	246
3	The Mitochondrial ATPase Inhibitory Factor 1 Triggers a ROS-Mediated Retrograde Prosurvival and Proliferative Response. <i>Molecular Cell</i> , 2012, 45, 731-742.	9.7	214
4	Alteration of the bioenergetic phenotype of mitochondria is a hallmark of breast, gastric, lung and oesophageal cancer. <i>Biochemical Journal</i> , 2004, 378, 17-20.	3.7	179
5	Up-regulation of the ATPase Inhibitory Factor 1 (IF1) of the Mitochondrial H ⁺ -ATP Synthase in Human Tumors Mediates the Metabolic Shift of Cancer Cells to a Warburg Phenotype. <i>Journal of Biological Chemistry</i> , 2010, 285, 25308-25313.	3.4	178
6	Loss of the Mitochondrial Bioenergetic Capacity Underlies the Glucose Avidity of Carcinomas. <i>Cancer Research</i> , 2007, 67, 9013-9017.	0.9	162
7	Breast carcinomas fulfill the Warburg hypothesis and provide metabolic markers of cancer prognosis. <i>Carcinogenesis</i> , 2005, 26, 2095-2104.	2.8	155
8	Mitochondrial ROS Production Protects the Intestine from Inflammation through Functional M2 Macrophage Polarization. <i>Cell Reports</i> , 2017, 19, 1202-1213.	6.4	146
9	The bioenergetic signature of lung adenocarcinomas is a molecular marker of cancer diagnosis and prognosis. <i>Carcinogenesis</i> , 2004, 25, 1157-1163.	2.8	131
10	Selection of cancer cells with repressed mitochondria triggers colon cancer progression. <i>Carcinogenesis</i> , 2010, 31, 567-576.	2.8	123
11	The mitochondrial ATP synthase is a shared drug target for aging and dementia. <i>Aging Cell</i> , 2018, 17, e12715.	6.7	109
12	PKA Phosphorylates the ATPase Inhibitory Factor 1 and Inactivates Its Capacity to Bind and Inhibit the Mitochondrial H ⁺ -ATP Synthase. <i>Cell Reports</i> , 2015, 12, 2143-2155.	6.4	104
13	The ATPase Inhibitory Factor 1 (IF1): A master regulator of energy metabolism and of cell survival. <i>Biochimica Et Biophysica Acta - Bioenergetics</i> , 2016, 1857, 1167-1182.	1.0	101
14	In vivo inhibition of the mitochondrial H ⁺ -ATP synthase in neurons promotes metabolic preconditioning. <i>EMBO Journal</i> , 2014, 33, 762-778.	7.8	93
15	Efficient execution of cell death in non-glycolytic cells requires the generation of ROS controlled by the activity of mitochondrial H ⁺ -ATP synthase. <i>Carcinogenesis</i> , 2006, 27, 925-935.	2.8	91
16	The H ⁺ -ATP synthase: A gate to ROS-mediated cell death or cell survival. <i>Biochimica Et Biophysica Acta - Bioenergetics</i> , 2014, 1837, 1099-1112.	1.0	91
17	The tumor suppressor function of mitochondria: Translation into the clinics. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2009, 1792, 1145-1158.	3.8	89
18	Translational regulation of mitochondrial differentiation in neonatal rat liver. Specific increase in the translational efficiency of the nuclear-encoded mitochondrial beta-F1-ATPase mRNA. <i>Journal of Biological Chemistry</i> , 1993, 268, 1868-1875.	3.4	71

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19	Lack of GDAP1 Induces Neuronal Calcium and Mitochondrial Defects in a Knockout Mouse Model of Charcot-Marie-Tooth Neuropathy. <i>PLoS Genetics</i> , 2015, 11, e1005115.	3.5	70
20	A Review of the Inhibition of the Mitochondrial ATP Synthase by IF1 in vivo: Reprogramming Energy Metabolism and Inducing Mitohormesis. <i>Frontiers in Physiology</i> , 2018, 9, 1322.	2.8	66
21	Degradation of IF1 controls energy metabolism during osteogenic differentiation of stem cells. <i>EMBO Reports</i> , 2013, 14, 638-644.	4.5	62
22	Coordinate Î²-adrenergic inhibition of mitochondrial activity and angiogenesis arrest tumor growth. <i>Nature Communications</i> , 2020, 11, 3606.	12.8	62
23	The bioenergetic signature of isogenic colon cancer cells predicts the cell death response to treatment with 3-bromopyruvate, iodoacetate or 5-fluorouracil. <i>Journal of Translational Medicine</i> , 2011, 9, 19.	4.4	61
24	Mitochondria-Mediated Energy Adaption in Cancer: The H ⁺ -ATP Synthase-Geared Switch of Metabolism in Human Tumors. <i>Antioxidants and Redox Signaling</i> , 2013, 19, 285-298.	5.4	59
25	Down-regulation of oxidative phosphorylation in the liver by expression of the ATPase inhibitory factor 1 induces a tumor-promoter metabolic state. <i>Oncotarget</i> , 2016, 7, 490-508.	1.8	59
26	The Role of Mitochondrial H ⁺ -ATP Synthase in Cancer. <i>Frontiers in Oncology</i> , 2018, 8, 53.	2.8	58
27	AMPK and GCN2â€“ATF4 signal the repression of mitochondria in colon cancer cells. <i>Biochemical Journal</i> , 2012, 444, 249-259.	3.7	56
28	Post-transcriptional regulation of the mitochondrial H ⁺ -ATP synthase: A key regulator of the metabolic phenotype in cancer. <i>Biochimica Et Biophysica Acta - Bioenergetics</i> , 2011, 1807, 543-551.	1.0	54
29	Cancer Abolishes the Tissue Type-Specific Differences in the Phenotype of Energetic Metabolism. <i>Translational Oncology</i> , 2009, 2, 138-145.	3.7	53
30	Hif-1Î± Knockdown Reduces Glycolytic Metabolism and Induces Cell Death of Human Synovial Fibroblasts Under Normoxic Conditions. <i>Scientific Reports</i> , 2017, 7, 3644.	3.3	53
31	Regulation of the H ⁺ -ATP synthase by IF1: a role in mitohormesis. <i>Cellular and Molecular Life Sciences</i> , 2017, 74, 2151-2166.	5.4	50
32	Metabolic reprogramming and disease progression in cancer patients. <i>Biochimica Et Biophysica Acta - Molecular Basis of Disease</i> , 2020, 1866, 165721.	3.8	45
33	Dysfunctional oxidative phosphorylation shunts branchedâ€“chain amino acid catabolism onto lipogenesis in skeletal muscle. <i>EMBO Journal</i> , 2020, 39, e103812.	7.8	33
34	Mitochondrial H ⁺ -ATP synthase in human skeletal muscle: contribution to dyslipidaemia and insulin resistance. <i>Diabetologia</i> , 2017, 60, 2052-2065.	6.3	32
35	MYC Induces a Hybrid Energetics Program Early in Cell Reprogramming. <i>Stem Cell Reports</i> , 2018, 11, 1479-1492.	4.8	31
36	Overexpression of Mitochondrial IF1 Prevents Metastatic Disease of Colorectal Cancer by Enhancing Anoikis and Tumor Infiltration of NK Cells. <i>Cancers</i> , 2020, 12, 22.	3.7	31

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37	Reverse phase protein microarrays quantify and validate the bioenergetic signature as biomarker in colorectal cancer. <i>Cancer Letters</i> , 2011, 311, 210-218.	7.2	28
38	Tissue-specific expression and post-transcriptional regulation of the ATPase inhibitory factor 1 (IF1) in human and mouse tissues. <i>FASEB Journal</i> , 2019, 33, 1836-1851.	0.5	23
39	Quantitative analysis of proteins of metabolism by reverse phase protein microarrays identifies potential biomarkers of rare neuromuscular diseases. <i>Journal of Translational Medicine</i> , 2015, 13, 65.	4.4	22
40	Overexpression of the ATPase Inhibitory Factor 1 Favors a Non-metastatic Phenotype in Breast Cancer. <i>Frontiers in Oncology</i> , 2017, 7, 69.	2.8	22
41	Generation of mitochondrial reactive oxygen species is controlled by ATPase inhibitory factor 1 and regulates cognition. <i>PLoS Biology</i> , 2021, 19, e3001252.	5.6	22
42	Short-term exposure of nontumorigenic human bronchial epithelial cells to carcinogenic chromium(VI) compromises their respiratory capacity and alters their bioenergetic signature. <i>FEBS Open Bio</i> , 2014, 4, 594-601.	2.3	19
43	Prognostic implications of markers of the metabolic phenotype in human cutaneous melanoma. <i>British Journal of Dermatology</i> , 2019, 181, 114-127.	1.5	19
44	Pyruvate kinase M2 and the mitochondrial ATPase Inhibitory Factor 1 provide novel biomarkers of dermatomyositis: a metabolic link to oncogenesis. <i>Journal of Translational Medicine</i> , 2017, 15, 29.	4.4	16
45	Plasma metabolome and skin proteins in Charcot-Marie-Tooth 1A patients. <i>PLoS ONE</i> , 2017, 12, e0178376.	2.5	16
46	Reprogramming Oxidative Phosphorylation in Cancer: A Role for RNA-Binding Proteins. <i>Antioxidants and Redox Signaling</i> , 2020, 33, 927-945.	5.4	13
47	Metformin as an Adjuvant to Photodynamic Therapy in Resistant Basal Cell Carcinoma Cells. <i>Cancers</i> , 2020, 12, 668.	3.7	13
48	Critical requirement of SOS1 RAS-GEF function for mitochondrial dynamics, metabolism, and redox homeostasis. <i>Oncogene</i> , 2021, 40, 4538-4551.	5.9	13
49	Changes in the Turnover of the Cellular Proteome during Metabolic Reprogramming: A Role for mtROS in Proteostasis. <i>Journal of Proteome Research</i> , 2019, 18, 3142-3155.	3.7	12
50	Specific Effects of Trabectedin and Lurbinectedin on Human Macrophage Function and Fate—Novel Insights. <i>Cancers</i> , 2020, 12, 3060.	3.7	11
51	Different mitochondrial genetic defects exhibit the same protein signature of metabolism in skeletal muscle of PEO and MELAS patients: A role for oxidative stress. <i>Free Radical Biology and Medicine</i> , 2018, 126, 235-248.	2.9	10
52	Mitochondrial Elongation and OPA1 Play Crucial Roles during the Stemness Acquisition Process in Pancreatic Ductal Adenocarcinoma. <i>Cancers</i> , 2022, 14, 3432.	3.7	8
53	Metformin overcomes metabolic reprogramming-induced resistance of skin squamous cell carcinoma to photodynamic therapy. <i>Molecular Metabolism</i> , 2022, 60, 101496.	6.5	7
54	The ATPase Inhibitory Factor 1 is a Tissue-Specific Physiological Regulator of the Structure and Function of Mitochondrial ATP Synthase: A Closer Look Into Neuronal Function. <i>Frontiers in Physiology</i> , 2022, 13, .	2.8	7

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55	Analysis of the metabolic proteome of lung adenocarcinomas by reverse-phase protein arrays (RPPA) emphasizes mitochondria as targets for therapy. <i>Oncogenesis</i> , 2022, 11, 24.	4.9	7
56	Exploiting the passenger ACO1-deficiency arising from 9p21 deletions to kill T-cell lymphoblastic neoplasia cells. <i>Carcinogenesis</i> , 2020, 41, 1113-1122.	2.8	6
57	Effective therapeutic strategies in a preclinical mouse model of Charcotâ€“Marieâ€“Tooth disease. <i>Human Molecular Genetics</i> , 2021, 30, 2441-2455.	2.9	5
58	Chronic inhibition of the mitochondrial ATP synthase in skeletal muscle triggers sarcoplasmic reticulum distress and tubular aggregates. <i>Cell Death and Disease</i> , 2022, 13, .	6.3	5
59	Sensitivity to anti-Fas is independent of increased cathepsin D activity and adrenodoxin reductase expression occurring in NOS-3 overexpressing HepG2 cells. <i>Biochimica Et Biophysica Acta - Molecular Cell Research</i> , 2015, 1853, 1182-1194.	4.1	1