Miriam Martini

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/4436853/publications.pdf

Version: 2024-02-01

43 papers	6,920 citations	236925 25 h-index	276875 41 g-index
44 all docs	44 docs citations	44 times ranked	10419 citing authors

#	Article	IF	Citations
1	Lysosomal lipid switch sensitises to nutrient deprivation and mTOR targeting in pancreatic cancer. Gut, 2023, 72, 360-371.	12.1	8
2	Phosphoinositide Conversion Inactivates Râ€RAS and Drives Metastases in Breast Cancer. Advanced Science, 2022, 9, e2103249.	11.2	8
3	Defective lipid signalling caused by mutations inPIK3C2Bunderlies focal epilepsy. Brain, 2022, 145, 2313-2331.	7.6	10
4	Iron supplementation is sufficient to rescue skeletal muscle mass and function in cancer cachexia. EMBO Reports, 2022, 23, e53746.	4.5	26
5	Docking Protein p130Cas Regulates Acinar to Ductal Metaplasia During Pancreatic Adenocarcinoma Development and Pancreatitis. Gastroenterology, 2022, 162, 1242-1255.e11.	1.3	4
6	Prospects of targeting PI3K/AKT/mTOR pathway in pancreatic cancer. Critical Reviews in Oncology/Hematology, 2022, 176, 103749.	4.4	37
7	Cholesterol Activates Cyclic AMP Signaling in Metaplastic Acinar Cells. Metabolites, 2021, 11, 141.	2.9	4
8	Protective Role of the M-Secâ€“Tunneling Nanotube System in Podocytes. Journal of the American Society of Nephrology: JASN, 2021, 32, 1114-1130.	6.1	12
9	The impact of COVID-19 on pancreatic cancer research and the path forward. Gastroenterology, 2021, 161, 1758-1763.	1.3	8
10	PI(3,4)P2-mediated cytokinetic abscission prevents early senescence and cataract formation. Science, 2021, 374, eabk0410.	12.6	37
11	A Novel Multiplex qRT-PCR Assay to Detect SARS-CoV-2 Infection: High Sensitivity and Increased Testing Capacity. Microorganisms, 2020, 8, 1064.	3.6	37
12	Phosphoinositides in cell proliferation and metabolism. Advances in Biological Regulation, 2020, 75, 100693.	2.3	14
13	PI(3,4)P2 Signaling in Cancer and Metabolism. Frontiers in Oncology, 2020, 10, 360.	2.8	48
14	Class II PI3K Functions in Cell Biology and Disease. Trends in Cell Biology, 2019, 29, 339-359.	7.9	99
15	Targeting PI3K signaling in cancer: Challenges and advances. Biochimica Et Biophysica Acta: Reviews on Cancer, 2019, 1871, 361-366.	7.4	54
16	The turtle and the rabbit story in a modern (PI3)key. Molecular and Cellular Oncology, 2018, 5, e1405141.	0.7	0
17	Phosphoinositide 3-Kinase Gamma Inhibition Protects From Anthracycline Cardiotoxicity and Reduces Tumor Growth. Circulation, 2018, 138, 696-711.	1.6	145
18	Signaling Pathways Regulating Redox Balance in Cancer Metabolism. Frontiers in Oncology, 2018, 8, 126.	2.8	57

#	Article	IF	Citations
19	The role of metabolic adaptation to nutrient stress in pancreatic cancer. Cell Stress, 2018, 2, 332-339.	3.2	19
20	Cytokinetic Abscission: Phosphoinositides and ESCRT<scp>s</scp> Direct the Final Cut. Journal of Cellular Biochemistry, 2017, 118, 3561-3568.	2.6	15
21	Mitotic Spindle Assembly and Genomic Stability in Breast Cancer Require PI3K-C2Î± Scaffolding Function. Cancer Cell, 2017, 32, 444-459.e7.	16.8	69
22	PI3K Signaling in Tissue Hyper-Proliferation: From Overgrowth Syndromes to Kidney Cysts. Cancers, 2017, 9, 30.	3.7	29
23	Rac signal adaptation controls neutrophil mobilization from the bone marrow. Science Signaling, 2016, 9, ra124.	3.6	14
24	How PI3K-derived lipids control cell division. Frontiers in Cell and Developmental Biology, 2015, 3, 61.	3.7	13
25	Measurement of PIP3 Levels Reveals an Unexpected Role for p110Î² in Early Adaptive Responses to p110Î±-Specific Inhibitors in Luminal Breast Cancer. Cancer Cell, 2015, 27, 97-108.	16.8	165
26	PI3K Class II Î± Controls Spatially Restricted Endosomal PtdIns3P and Rab11 Activation to Promote Primary Cilium Function. Developmental Cell, 2014, 28, 647-658.	7.0	177
27	PI3K in cancerâ€“stroma interactions: bad in seed and ugly in soil. Oncogene, 2014, 33, 3083-3090.	5.9	55
28	PI3K/AKT signaling pathway and cancer: an updated review. Annals of Medicine, 2014, 46, 372-383.	3.8	887
29	Modeling Tumor Progression by the Sequential Introduction of Genetic Alterations into the Genome of Human Normal Cells. Human Mutation, 2013, 34, 330-337.	2.5	6
30	Targeting PI3K in Cancer: Any Good News?. Frontiers in Oncology, 2013, 3, 108.	2.8	87
31	BRAF V600E Is a Determinant of Sensitivity to Proteasome Inhibitors. Molecular Cancer Therapeutics, 2013, 12, 2950-2961.	4.1	18
32	Mixed Lineage Kinase MLK4 Is Activated in Colorectal Cancers Where It Synergistically Cooperates with Activated RAS Signaling in Driving Tumorigenesis. Cancer Research, 2013, 73, 1912-1921.	0.9	15
33	Targeting oncogenic serine/threonine-protein kinase BRAF in cancer cells inhibits angiogenesis and abrogates hypoxia. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, E353-9.	7.1	51
34	PI3K keeps the balance between metabolism and cancer. Advances in Biological Regulation, 2012, 52, 389-405.	2.3	37
35	Targeted therapies: how personal should we go?. Nature Reviews Clinical Oncology, 2012, 9, 87-97.	27.6	94
36	Integrated molecular dissection of the epidermal growth factor receptor (EFGR) oncogenic pathway to predict response to EGFR-targeted monoclonal antibodies in metastatic colorectal cancer. Targeted Oncology, 2010, 5, 19-28.	3.6	27

#	Article	IF	Citations
37	Effects of KRAS, BRAF, NRAS, and PIK3CA mutations on the efficacy of cetuximab plus chemotherapy in chemotherapy-refractory metastatic colorectal cancer: a retrospective consortium analysis. Lancet Oncology, The, 2010, 11, 753-762.	10.7	1,915
38	Multi-Determinants Analysis of Molecular Alterations for Predicting Clinical Benefit to EGFR-Targeted Monoclonal Antibodies in Colorectal Cancer. PLoS ONE, 2009, 4, e7287.	2.5	241
39	PIK3CA Mutations in Colorectal Cancer Are Associated with Clinical Resistance to EGFR-Targeted Monoclonal Antibodies. Cancer Research, 2009, 69, 1851-1857.	0.9	711
40	Abstract B229: Molecular mechanisms of resistance to cetuximab and panitumumab in colorectal cancer. , 2009, , .		0
41	Wild-Type BRAF Is Required for Response to Panitumumab or Cetuximab in Metastatic Colorectal Cancer. Journal of Clinical Oncology, 2008, 26, 5705-5712.	1.6	1,540
42	Replacement of normal with mutant alleles in the genome of normal human cells unveils mutation-specific drug responses. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 20864-20869.	7.1	95
43	Knock-in of Oncogenic Kras Does Not Transform Mouse Somatic Cells But Triggers a Transcriptional Response that Classifies Human Cancers. Cancer Research, 2007, 67, 8468-8476.	0.9	32