

Gabriela Paroni

List of Publications by Year in descending order

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36
papers

1,465
citations

430442

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citing authors

#	ARTICLE	IF	CITATIONS
1	A DOCK1 Gene-Derived Circular RNA Is Highly Expressed in Luminal Mammary Tumours and Is Involved in the Epithelial Differentiation, Growth, and Motility of Breast Cancer Cells. <i>Cancers</i> , 2021, 13, 5325.	1.7	6
2	Retinoic Acid Sensitivity of Triple-Negative Breast Cancer Cells Characterized by Constitutive Activation of the notch1 Pathway: The Role of RAR β . <i>Cancers</i> , 2020, 12, 3027.	1.7	10
3	All-Trans Retinoic Acid Stimulates Viral Mimicry, Interferon Responses and Antigen Presentation in Breast-Cancer Cells. <i>Cancers</i> , 2020, 12, 1169.	1.7	15
4	Role of mitochondria and cardiolipins in growth inhibition of breast cancer cells by retinoic acid. <i>Journal of Experimental and Clinical Cancer Research</i> , 2019, 38, 436.	3.5	11
5	HER2-positive breast-cancer cell lines are sensitive to KDM5 inhibition: definition of a gene-expression model for the selection of sensitive cases. <i>Oncogene</i> , 2019, 38, 2675-2689.	2.6	23
6	S100A3 a partner protein regulating the stability/activity of RAR β and PML-RAR β in cellular models of breast/lung cancer and acute myeloid leukemia. <i>Oncogene</i> , 2019, 38, 2482-2500.	2.6	18
7	Network-guided modeling allows tumor-type independent prediction of sensitivity to all-trans-retinoic acid. <i>Annals of Oncology</i> , 2017, 28, 611-621.	0.6	31
8	RAR β 2 and PML-RAR similarities in the control of basal and retinoic acid induced myeloid maturation of acute myeloid leukemia cells. <i>Oncotarget</i> , 2017, 8, 37041-37060.	0.8	8
9	Abstract 2101: A gene-expression fingerprint predicting sensitivity to all-trans-retinoic acid in breast cancer cells is tumor-context independent. , 2016, , .		0
10	Cellular and molecular determinants of all-trans retinoic acid sensitivity in breast cancer: Luminal phenotype and RAR β expression. <i>EMBO Molecular Medicine</i> , 2015, 7, 950-972.	3.3	60
11	All-trans-retinoic Acid Modulates the Plasticity and Inhibits the Motility of Breast Cancer Cells. <i>Journal of Biological Chemistry</i> , 2015, 290, 17690-17709.	1.6	44
12	MicroRNA networks regulated by all-trans retinoic acid and Lapatinib control the growth, survival and motility of breast cancer cells. <i>Oncotarget</i> , 2015, 6, 13176-13200.	0.8	33
13	Retinoids and breast cancer: From basic studies to the clinic and back again. <i>Cancer Treatment Reviews</i> , 2014, 40, 739-749.	3.4	113
14	Abstract 2099: Cellular and molecular determinants of retinoic acid sensitivity in breast cancer. , 2014, , .		0
15	New insights into the molecular mechanisms underlying sensitivity/resistance to the atypical retinoid ST1926 in acute myeloid leukaemia cells: The role of histone H2A.Z, cAMP-dependent protein kinase A and the proteasome. <i>European Journal of Cancer</i> , 2013, 49, 1491-1500.	1.3	14
16	Retinoids and breast cancer: new clues to increase their activity and selectivity. <i>Breast Cancer Research</i> , 2012, 14, 111.	2.2	18
17	Synergistic antitumor activity of lapatinib and retinoids on a novel subtype of breast cancer with coamplification of ERBB2 and RARA. <i>Oncogene</i> , 2012, 31, 3431-3443.	2.6	51
18	p38 β MAPK interacts with and inhibits RAR β : suppression of the kinase enhances the therapeutic activity of retinoids in acute myeloid leukemia cells. <i>Leukemia</i> , 2012, 26, 1850-1861.	3.3	24

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19	Lipofuscin Accumulation and Gene Expression in Different Tissues of mnd Mice. <i>Molecular Neurobiology</i> , 2012, 45, 247-257.	1.9	6
20	Abstract 1836: p38 β MAPK interacts with and inhibits RAR α : Suppression of the kinase enhances the therapeutic activity of retinoids in acute myeloid leukemia cells. , 2012, , .		0
21	Measurement of Caspase Activity: From Cell Populations to Individual Cells. <i>Methods in Molecular Biology</i> , 2011, 740, 65-79.	0.4	1
22	Induction of miR-21 by Retinoic Acid in Estrogen Receptor-positive Breast Carcinoma Cells. <i>Journal of Biological Chemistry</i> , 2011, 286, 4027-4042.	1.6	82
23	Abstract 2287: A sub-population of HER2+breast carcinomas is characterized by co-amplification of the ERBB2 and RARA genes that renders cancer cells sensitive to retinoids and combinations of these agents with lapatinib. , 2011, , .		0
24	Abstract 1717: Combinations of retinoids and lapatinib in the treatment of Her2/Neu-positive breast carcinomas with co-amplification of theERBB2andRARAgenes. , 2010, , .		0
25	PP2A Regulates HDAC4 Nuclear Import. <i>Molecular Biology of the Cell</i> , 2008, 19, 655-667.	0.9	108
26	Atypical retinoids ST1926 and CD437 are S-phase-specific agents causing DNA double-strand breaks: significance for the cytotoxic and antiproliferative activity. <i>Molecular Cancer Therapeutics</i> , 2008, 7, 2941-2954.	1.9	39
27	Dephosphorylation and Caspase Processing Generate Distinct Nuclear Pools of Histone Deacetylase 4. <i>Molecular and Cellular Biology</i> , 2007, 27, 6718-6732.	1.1	35
28	Caspase activation and apoptosis in response to proteasome inhibitors. <i>Cell Death and Differentiation</i> , 2005, 12, 1240-1254.	5.0	52
29	Caspase-dependent Regulation of Histone Deacetylase 4 Nuclear-Cytoplasmic Shuttling Promotes Apoptosis. <i>Molecular Biology of the Cell</i> , 2004, 15, 2804-2818.	0.9	128
30	Role of Caspases, Bid, and p53 in the Apoptotic Response Triggered by Histone Deacetylase Inhibitors Trichostatin-A (TSA) and Suberoylanilide Hydroxamic Acid (SAHA). <i>Journal of Biological Chemistry</i> , 2003, 278, 12579-12589.	1.6	137
31	Caspase-2 Can Trigger Cytochrome c Release and Apoptosis from the Nucleus. <i>Journal of Biological Chemistry</i> , 2002, 277, 15147-15161.	1.6	159
32	The death substrate Gas2 binds m-calpain and increases susceptibility to p53-dependent apoptosis. <i>EMBO Journal</i> , 2001, 20, 2702-2714.	3.5	100
33	Caspase-2-induced Apoptosis Is Dependent on Caspase-9, but Its Processing during UV- or Tumor Necrosis Factor-dependent Cell Death Requires Caspase-3. <i>Journal of Biological Chemistry</i> , 2001, 276, 21907-21915.	1.6	95
34	Reduction of mdr1 Gene Amplification in Human Multidrug-Resistant LoVo DX Cell Line Is Promoted by Triple Helix-Forming Oligonucleotides. <i>Oligonucleotides</i> , 1999, 9, 261-270.	4.4	8
35	Effect of Oligomer Length and Base Substitutions on the Cytotoxic Activity and Specific Nuclear Protein Recognition of GTn Oligonucleotides in the Human Leukemic CCRF-CEM Cell Line. <i>Nucleosides & Nucleotides</i> , 1999, 18, 1711-1716.	0.5	11
36	Lipid-sensors, enigmatic-orphan and orphan nuclear receptors as therapeutic targets in breast-cancer. <i>Oncotarget</i> , 0, 7, 42661-42682.	0.8	24