Almudena Porras

List of Publications by Year
in descending order

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59 papers	3,515 citations	257450 24 h-index	161849 54 g-index
60 all docs	60 docs citations	60 times ranked	5068 citing authors

#	Article	IF	Citations
1	In vivo production of fluorine-18 in a chicken egg tumor model of breast cancer for proton therapy range verification. Scientific Reports, 2022, 12, 7075.	3.3	3
2	Centrosome Dynamics and Its Role in Inflammatory Response and Metastatic Process. Biomolecules, 2021, 11, 629.	4.0	5
3	C3G downregulation induces the acquisition of a mesenchymal phenotype that enhances aggressiveness of glioblastoma cells. Cell Death and Disease, 2021, 12, 348.	6.3	7
4	HGK promotes metastatic dissemination in prostate cancer. Scientific Reports, 2021, 11, 12287.	3.3	8
5	Abstract 1974: C3G down-regulation in glioblastoma induces a pro-invasive and glycolytic phenotype, accompanied by RTKs dysregulation. , 2021, , .		0
6	Abstract 2310: Identification of novel essential genes for prostate cancer metastasis by genome scale CRISPR approaches. , 2021, , .		0
7	C3G Protein, a New Player in Glioblastoma. International Journal of Molecular Sciences, 2021, 22, 10018.	4.1	4
8	C3G Is Upregulated in Hepatocarcinoma, Contributing to Tumor Growth and Progression and to HGF/MET Pathway Activation. Cancers, 2020, 12, 2282.	3.7	6
9	C3G contributes to platelet activation and aggregation by regulating major signaling pathways. Signal Transduction and Targeted Therapy, 2020, 5, 29.	17.1	14
10	Immune Resistance and EGFR Antagonists in Colorectal Cancer. Cancers, 2019, 11, 1089.	3.7	37
11	JAK/Stat5-mediated subtype-specific lymphocyte antigen 6 complex, locus G6D (LY6G6D) expression drives mismatch repair proficient colorectal cancer. Journal of Experimental and Clinical Cancer Research, 2019, 38, 28.	8.6	24
12	How Rap and its GEFs control liver physiology and cancer development. C3G alterations in human hepatocarcinoma. Hepatic Oncology, 2018, 5, HEP05.	4.2	22
13	TWEAK promotes migration and invasion in MEFs through a mechanism dependent on ERKs activation and Fibulin 3 down-regulation. Journal of Cellular Physiology, 2018, 233, 968-978.	4.1	0
14	C3G, through its GEF activity, induces megakaryocytic differentiation and proplatelet formation. Cell Communication and Signaling, 2018, 16, 101.	6.5	15
15	C3G promotes a selective release of angiogenic factors from activated mouse platelets to regulate angiogenesis and tumor metastasis. Oncotarget, 2017, 8, 110994-111011.	1.8	24
16	Emerging Insight into MAPK Inhibitors and Immunotherapy in Colorectal Cancer. Current Medicinal Chemistry, 2017, 24, 1383-1402.	2.4	23
17	C3G knock-down enhances migration and invasion by increasing Rap1-mediated p38Î± activation, while it impairs tumor growth through p38Î±-independent mechanisms. Oncotarget, 2016, 7, 45060-45078.	1.8	23
18	p38 MAPK Down-regulates Fibulin 3 Expression through Methylation of Gene Regulatory Sequences. Journal of Biological Chemistry, 2015, 290, 4383-4397.	3.4	21

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19	HGF/c-Met signaling promotes liver progenitor cell migration and invasion by an epithelialâ€“mesenchymal transition-independent, phosphatidyl inositol-3 kinase-dependent pathway in an in vitro model. Biochimica Et Biophysica Acta - Molecular Cell Research, 2015, 1853, 2453-2463.	4.1	36
20	C3G forms complexes with Bcr-Abl and p38Î± MAPK at the focal adhesions in chronic myeloid leukemia cells: implication in the regulation of leukemic cell adhesion. Cell Communication and Signaling, 2013, 11, 9.	6.5	24
21	Met signaling in cardiomyocytes is required for normal cardiac function in adult mice. Biochimica Et Biophysica Acta - Molecular Basis of Disease, 2013, 1832, 2204-2215.	3.8	29
22	TWEAK prevents TNF-Î±-induced insulin resistance through PP2A activation in human adipocytes. American Journal of Physiology - Endocrinology and Metabolism, 2013, 305, E101-E112.	3.5	22
23	p38Î± Mediates Cell Survival in Response to Oxidative Stress via Induction of Antioxidant Genes. Journal of Biological Chemistry, 2012, 287, 2632-2642.	3.4	115
24	Critical role of hydrogen peroxide signaling in the sequential activation of p38 MAPK and eNOS in laminar shear stress. Free Radical Biology and Medicine, 2012, 52, 1093-1100.	2.9	57
25	C3G transgenic mouse models with specific expression in platelets reveal a new role for C3G in platelet clotting through its GEF activity. Biochimica Et Biophysica Acta - Molecular Cell Research, 2012, 1823, 1366-1377.	4.1	14
26	C3G down-regulates p38 MAPK activity in response to stress by Rap-1 independent mechanisms: Involvement in cell death. Cellular Signalling, 2010, 22, 533-542.	3.6	26
27	C3G silencing enhances STI-571-induced apoptosis in CML cells through p38 MAPK activation, but it antagonizes STI-571 inhibitory effect on survival. Cellular Signalling, 2009, 21, 1229-1235.	3.6	22
28	A role for p38Î± mitogenâ€activated protein kinase in embryonic cardiac differentiation. FEBS Letters, 2008, 582, 1025-1031.	2.8	16
29	Met acts on Mdm2 via mTOR to signal cell survival during development. Development (Cambridge), 2007, 134, 1443-1451.	2.5	85
30	p38Î± MAPK can positively or negatively regulate Racâ€1 activity depending on the presence of serum. FEBS Letters, 2007, 581, 3819-3825.	2.8	14
31	Negative regulation of Akt activity by p38Î±ÂMAP kinase in cardiomyocytes involves membrane localization of PP2A through interaction with caveolin-1. Cellular Signalling, 2007, 19, 62-74.	3.6	57
32	Apoptosis by cisplatin requires p53 mediated p38Î± MAPK activation through ROS generation. Apoptosis: an International Journal on Programmed Cell Death, 2007, 12, 1733-1742.	4.9	338
33	p38Î± Mitogen-activated Protein Kinase Sensitizes Cells to Apoptosis Induced by Different Stimuli. Molecular Biology of the Cell, 2004, 15, 922-933.	2.1	213
34	Correlation between DNA synthesis in the second, third and fourth generations of spermatogonia and the occurrence of apoptosis in both spermatogonia and spermatocytes. Reproduction, 2003, 126, 661-668.	2.6	19
35	Long-Term Treatment with Insulin Induces Apoptosis in Brown Adipocytes: Role of Oxidative Stress. Endocrinology, 2003, 144, 5390-5401.	2.8	19
36	p38 MAPK enhances STAT1-dependent transcription independently of Ser-727 phosphorylation. Proceedings of the National Academy of Sciences of the United States of America, 2002, 99, 12859-12864.	7.1	119

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37	Regulation of Proliferation, Differentiation and Apoptosis of Brown Adipocytes: Signal Transduction Pathways Involved. Cell and Molecular Response To Stress, 2002, , 269-282.	0.4	2
38	Differential role of PPARÎ³ in the regulation of UCP-1 and adipogenesis by TNF-Î± in brown adipocytes. FEBS Letters, 2002, 520, 58-62.	2.8	18
39	TNF-Î± inhibits UCP-1 expression in brown adipocytes via ERKs. FEBS Letters, 2001, 493, 6-11.	2.8	56
40	Activation of p38MAPK by TGF-Î² in fetal rat hepatocytes requires radical oxygen production, but is dispensable for cell death. FEBS Letters, 2001, 499, 225-229.	2.8	38
41	Noradrenaline induces brown adipocytes cell growth via ?-receptors by a mechanism dependent on ERKs but independent of cAMP and PKA. Journal of Cellular Physiology, 2000, 185, 324-330.	4.1	27
42	p38 MAP kinases: beyond the stress response. Trends in Biochemical Sciences, 2000, 25, 257-260.	7.5	526
43	p38 Mitogen-Activated Protein Kinase Mediates Tumor Necrosis Factor-Î±-Induced Apoptosis in Rat Fetal Brown Adipocytes*. Endocrinology, 2000, 141, 4383-4395.	2.8	59
44	Essential Role of p38Î± MAP Kinase in Placental but Not Embryonic Cardiovascular Development. Molecular Cell, 2000, 6, 109-116.	9.7	468
45	p38 Mitogen-Activated Protein Kinase Mediates Tumor Necrosis Factor-Â-Induced Apoptosis in Rat Fetal Brown Adipocytes. Endocrinology, 2000, 141, 4383-4395.	2.8	12
46	p42/p44 Mitogen-Activated Protein Kinases Activation Is Required for the Insulin-Like Growth Factor-I/Insulin Induced Proliferation, but Inhibits Differentiation, in Rat Fetal Brown Adipocytes. Molecular Endocrinology, 1998, 12, 825-834.	3.7	64
47	p42/p44 Mitogen-Activated Protein Kinases Activation Is Required for the Insulin-Like Growth Factor-I/Insulin Induced Proliferation, but Inhibits Differentiation, in Rat Fetal Brown Adipocytes. Molecular Endocrinology, 1998, 12, 825-834.	3.7	18
48	Notch-1 Controls the Expression of Fatty Acid-activated Transcription Factors and Is Required for Adipogenesis. Journal of Biological Chemistry, 1997, 272, 29729-29734.	3.4	139
49	Mitogen-Activated Protein Kinase Activation Is Not Necessary for, but Antagonizes, 3T3-L1 Adipocytic Differentiation. Molecular and Cellular Biology, 1997, 17, 6068-6075.	2.3	171
50	TNFâ€Î± induces apoptosis in rat fetal brown adipocytes in primary culture. FEBS Letters, 1997, 416, 324-328.	2.8	48
51	Ras Proteins Mediate Induction of Uncoupling Protein, IGF-I, and IGF-I Receptor in Rat Fetal Brown Adipocyte Cell Lines. DNA and Cell Biology, 1996, 15, 921-928.	1.9	7
52	Triiodothyronine Induces the Transcription of the Uncoupling Protein Gene and Stabilizes Its mRNA in Fetal Rat Brown Adipocyte Primary Cultures. Journal of Biological Chemistry, 1996, 271, 2076-2081.	3.4	67
53	Establishment of Permanent Brown Adipocyte Cell Lines Achieved by Transfection with SV40 Large T Antigen and ras Genes. Experimental Cell Research, 1993, 209, 248-254.	2.6	26
54	The ras signaling pathway mimics insulin action on glucose transporter translocation.. Proceedings of the National Academy of Sciences of the United States of America, 1993, 90, 4460-4464.	7.1	103

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55	Differentiation of 3T3-L1 fibroblasts to adipocytes induced by transfection of ras oncogenes. Science, 1991, 253, 565-568.	12.6	159
56	Development of the uncoupling protein in the rat brown-adipose tissue during the perinatal period. Its relationship with the mitochondrial GDP-binding and GDP-sensitive ion permeabilities and respiration. FEBS Journal, 1990, 187, 671-675.	0.2	18
57	Characterization of a System to Study the Uncoupling Protein Expression in Brown Adipocyte Primary Cultures. , 1990, , 147-152.		1
58	Adrenergic regulation of the uncoupling protein expression in foetal rat brown adipocytes in primary culture. Biochemical and Biophysical Research Communications, 1989, 163, 541-547.	2.1	27
59	Contribution of C3G and other GEFs to liver cancer development and progression. , 0, , .		0