Almudena Porras

List of Publications by Year in descending order

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59 papers 3,515 citations

257450 24 h-index 54 g-index

60 all docs 60 docs citations

60 times ranked

5068 citing authors

#	Article	IF	CITATIONS
1	p38 MAP kinases: beyond the stress response. Trends in Biochemical Sciences, 2000, 25, 257-260.	7.5	526
2	Essential Role of p38α MAP Kinase in Placental but Not Embryonic Cardiovascular Development. Molecular Cell, 2000, 6, 109-116.	9.7	468
3	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
4	p38α Mitogen-activated Protein Kinase Sensitizes Cells to Apoptosis Induced by Different Stimuli. Molecular Biology of the Cell, 2004, 15, 922-933.	2.1	213
5	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
6	Differentiation of 3T3-L1 fibroblasts to adipocytes induced by transfection of ras oncogenes. Science, 1991, 253, 565-568.	12.6	159
7	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
8	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
9	p38α Mediates Cell Survival in Response to Oxidative Stress via Induction of Antioxidant Genes. Journal of Biological Chemistry, 2012, 287, 2632-2642.	3.4	115
10	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
11	Met acts on Mdm2 via mTOR to signal cell survival during development. Development (Cambridge), 2007, 134, 1443-1451.	2.5	85
12	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
13	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
14	p38 Mitogen-Activated Protein Kinase Mediates Tumor Necrosis Factor-α-Induced Apoptosis in Rat Fetal Brown Adipocytes*. Endocrinology, 2000, 141, 4383-4395.	2.8	59
15	Negative regulation of Akt activity by p38αÂMAP kinase in cardiomyocytes involves membrane localization of PP2A through interaction with caveolin-1. Cellular Signalling, 2007, 19, 62-74.	3.6	57
16	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
17	TNF-α inhibits UCP-1 expression in brown adipocytes via ERKs. FEBS Letters, 2001, 493, 6-11.	2.8	56
18	TNFâ€Î± induces apoptosis in rat fetal brown adipocytes in primary culture. FEBS Letters, 1997, 416, 324-328.	2.8	48

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19	Activation of p38MAPK by TGF- \hat{l}^2 in fetal rat hepatocytes requires radical oxygen production, but is dispensable for cell death. FEBS Letters, 2001, 499, 225-229.	2.8	38
20	Immune Resistance and EGFR Antagonists in Colorectal Cancer. Cancers, 2019, 11, 1089.	3.7	37
21	HGF/c-Met signaling promotes liver progenitor cell migration and invasion by an epithelial–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
22	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
23	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
24	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
25	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
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 forms complexes with Bcr-Abl and p38 \hat{l} ± 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
28	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
29	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
30	C3G knock-down enhances migration and invasion by increasing Rap1-mediated p38 \hat{l} ± activation, while it impairs tumor growth through p38 \hat{l} ±-independent mechanisms. Oncotarget, 2016, 7, 45060-45078.	1.8	23
31	Emerging Insight into MAPK Inhibitors and Immunotherapy in Colorectal Cancer. Current Medicinal Chemistry, 2017, 24, 1383-1402.	2.4	23
32	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
33	TWEAK prevents TNF-α-induced insulin resistance through PP2A activation in human adipocytes. American Journal of Physiology - Endocrinology and Metabolism, 2013, 305, E101-E112.	3.5	22
34	How Rap and its GEFs control liver physiology and cancer development. C3G alterations in human hepatocarcinoma. Hepatic Oncology, 2018, 5, HEP05.	4.2	22
35	p38 MAPK Down-regulates Fibulin 3 Expression through Methylation of Gene Regulatory Sequences. Journal of Biological Chemistry, 2015, 290, 4383-4397.	3.4	21
36	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

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37	Long-Term Treatment with Insulin Induces Apoptosis in Brown Adipocytes: Role of Oxidative Stress. Endocrinology, 2003, 144, 5390-5401.	2.8	19
38	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
39	Differential role of PPARγ in the regulation of UCP-1 and adipogenesis by TNF-α in brown adipocytes. FEBS Letters, 2002, 520, 58-62.	2.8	18
40	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
41	A role for p38α mitogenâ€activated protein kinase in embryonic cardiac differentiation. FEBS Letters, 2008, 582, 1025-1031.	2.8	16
42	C3G, through its GEF activity, induces megakaryocytic differentiation and proplatelet formation. Cell Communication and Signaling, 2018, 16, 101.	6.5	15
43	p38α MAPK can positively or negatively regulate Racâ€1 activity depending on the presence of serum. FEBS Letters, 2007, 581, 3819-3825.	2.8	14
44	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
45	C3G contributes to platelet activation and aggregation by regulating major signaling pathways. Signal Transduction and Targeted Therapy, 2020, 5, 29.	17.1	14
46	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
47	HGK promotes metastatic dissemination in prostate cancer. Scientific Reports, 2021, 11, 12287.	3.3	8
48	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
49	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
50	C3G Is Upregulated in Hepatocarcinoma, Contributing to Tumor Growth and Progression and to HGF/MET Pathway Activation. Cancers, 2020, 12, 2282.	3.7	6
51	Centrosome Dynamics and Its Role in Inflammatory Response and Metastatic Process. Biomolecules, 2021, 11, 629.	4.0	5
52	C3G Protein, a New Player in Glioblastoma. International Journal of Molecular Sciences, 2021, 22, 10018.	4.1	4
53	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
54	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

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55	Characterization of a System to Study the Uncoupling Protein Expression in Brown Adipocyte Primary Cultures., 1990,, 147-152.		1
56	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	O
57	Abstract 1974: C3G down-regulation in glioblastoma induces a pro-invasive and glycolytic phenotype, accompanied by RTKs dysregulation., 2021,,.		O
58	Abstract 2310: Identification of novel essential genes for prostate cancer metastasis by genome scale CRISPR approaches., 2021,,.		0
59	Contribution of C3G and other GEFs to liver cancer development and progression. , 0, , .		O