David A Foster

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/7612595/publications.pdf

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38 papers 2,375 citations

236925 25 h-index 315739 38 g-index

41 all docs

41 docs citations

times ranked

41

3587 citing authors

#	Article	IF	CITATIONS
1	Cancer cells with defective RB and CDKN2A are resistant to the apoptotic effects of rapamycin. Cancer Letters, 2021, 522, 164-170.	7.2	7
2	Phosphatidic acid drives mTORC1 lysosomal translocation in the absence of amino acids. Journal of Biological Chemistry, 2020, 295, 263-274.	3.4	19
3	Albumin promotes the progression of fibroblasts through late G $<$ sub $>$ 1 $<$ /sub $>$ into S-phase in the absence of growth factors. Cell Cycle, 2020, 19, 2158-2167.	2.6	3
4	(-)-Oleocanthal and (-)-oleocanthal-rich olive oils induce lysosomal membrane permeabilization in cancer cells. PLoS ONE, 2019, 14, e0216024.	2.5	16
5	Glutamine as an Essential Amino Acid for KRas-Driven Cancer Cells. Trends in Endocrinology and Metabolism, 2019, 30, 357-368.	7.1	52
6	Elevated phospholipase D activity in androgen-insensitive prostate cancer cells promotes both survival and metastatic phenotypes. Cancer Letters, 2018, 423, 28-35.	7.2	13
7	Phospholipase D–dependent mTOR complex 1 (mTORC1) activation by glutamine. Journal of Biological Chemistry, 2018, 293, 16390-16401.	3.4	41
8	Lipid sensing by mTOR complexes via de novo synthesis of phosphatidic acid. Journal of Biological Chemistry, 2017, 292, 6303-6311.	3.4	99
9	A Late G1 Lipid Checkpoint That Is Dysregulated in Clear Cell Renal Carcinoma Cells. Journal of Biological Chemistry, 2017, 292, 936-944.	3.4	17
10	Aspartate Rescues S-phase Arrest Caused by Suppression of Glutamine Utilization in KRas-driven Cancer Cells. Journal of Biological Chemistry, 2016, 291, 9322-9329.	3.4	59
11	The Enigma of Rapamycin Dosage. Molecular Cancer Therapeutics, 2016, 15, 347-353.	4.1	80
12	Apoptotic effects of high-dose rapamycin occur in S-phase of the cell cycle. Cell Cycle, 2015, 14, 2285-2292.	2.6	26
13	(-)-Oleocanthal rapidly and selectively induces cancer cell death via lysosomal membrane permeabilization. Molecular and Cellular Oncology, 2015, 2, e1006077.	0.7	53
14	Rapamycin-induced G1 cell cycle arrest employs both TGF- \hat{l}^2 and Rb pathways. Cancer Letters, 2015, 360, 134-140.	7.2	54
15	5-Aminoimidazole-4-carboxamide- $1-\hat{l}^2$ -4-ribofuranoside (AlCAR) enhances the efficacy of rapamycin in human cancer cells. Cell Cycle, 2015, 14, 3331-3339.	2.6	40
16	Reciprocal Regulation of AMP-activated Protein Kinase and Phospholipase D. Journal of Biological Chemistry, 2015, 290, 6986-6993.	3.4	52
17	Synthetic lethality in KRas-driven cancer cells created by glutamine deprivation. Oncoscience, 2015, 2, 807-808.	2.2	43
18	Metabolic vulnerability of KRAS-driven cancer cells. Molecular and Cellular Oncology, 2014, 1, e963445.	0.7	5

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19	Mutant Ras Elevates Dependence on Serum Lipids and Creates a Synthetic Lethality for Rapamycin. Molecular Cancer Therapeutics, 2014, 13, 733-741.	4.1	26
20	Inhibition of fatty acid synthase induces pro-survival Akt and ERK signaling in K-Ras-driven cancer cells. Cancer Letters, 2014, 353, 258-263.	7.2	11
21	Phospholipase D and the Maintenance of Phosphatidic Acid Levels for Regulation of Mammalian Target of Rapamycin (mTOR). Journal of Biological Chemistry, 2014, 289, 22583-22588.	3.4	108
22	Inhibition of S6 kinase suppresses the apoptotic effect of elF4E ablation by inducing TGF- \hat{l}^2 -dependent G1 cell cycle arrest. Cancer Letters, 2013, 333, 239-243.	7.2	16
23	Phosphatidic acid and lipid-sensing by mTOR. Trends in Endocrinology and Metabolism, 2013, 24, 272-278.	7.1	89
24	Amino Acids and mTOR Mediate Distinct Metabolic Checkpoints in Mammalian G1 Cell Cycle. PLoS ONE, 2013, 8, e74157.	2.5	58
25	Reduced mortality and moderate alcohol consumption: The phospholipase D-mTOR connection. Cell Cycle, 2010, 9, 1291-1294.	2.6	9
26	Targeting mTOR with rapamycin: One dose does not fit all. Cell Cycle, 2009, 8, 1026-1029.	2.6	119
27	Hidesaburo Hanafusa (1929–2009). Nature, 2009, 458, 718-718.	27.8	0
28	Phosphatidic acid signaling to mTOR: Signals for the survival of human cancer cells. Biochimica Et Biophysica Acta - Molecular and Cell Biology of Lipids, 2009, 1791, 949-955.	2.4	182
29	Regulation of mTOR by Phosphatidic Acid?: Figure 1 Cancer Research, 2007, 67, 1-4.	0.9	181
30	Myc stabilization in response to estrogen and phospholipase D in MCF-7 breast cancer cells. FEBS Letters, 2006, 580, 5647-5652.	2.8	26
31	Targeting mTOR-mediated survival signals in anticancer therapeutic strategies. Expert Review of Anticancer Therapy, 2004, 4, 691-701.	2.4	31
32	The enigmatic protein kinase CÎ: complex roles in cell proliferation and survival. FASEB Journal, 2004, 18, 627-636.	0.5	188
33	Phospholipase D prevents apoptosis in v-Src-transformed rat fibroblasts and MDA-MB-231 breast cancer cells. Biochemical and Biophysical Research Communications, 2003, 302, 615-619.	2.1	85
34	Phospholipase D in cell proliferation and cancer. Molecular Cancer Research, 2003, 1, 789-800.	3.4	233
35	Phospholipase D2 stimulates cell protrusion in v-Src-transformed cells. Biochemical and Biophysical Research Communications, 2002, 293, 201-206.	2.1	25
36	Elevated phospholipase D activity induces apoptosis in normal rat fibroblasts. Biochemical and Biophysical Research Communications, 2002, 298, 474-477.	2.1	12

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37	Alcohol Consumption Raises HDL Cholesterol Levels by Increasing the Transport Rate of Apolipoproteins A-I and A-II. Circulation, 2000, 102, 2347-2352.	1.6	264
38	Regulation of Phosphatidic Acid Phosphohydrolase by Epidermal Growth Factor. Journal of Biological Chemistry, 1996, 271, 29529-29532.	3.4	26