Kirsten L Bryant

List of Publications by Year
in descending order

21 papers	2,217 citations	687363 13 h-index	713466 21 g-index
22 all docs	22 docs citations	22 times ranked	5124 citing authors

#	Article	IF	Citations
1	Unraveling and targeting RAS-driven metabolic signaling for therapeutic gain. Advances in Cancer Research, 2022, 153, 267-304.	5.0	2
2	Susceptibility to autophagy inhibition is enhanced by dual IGF1R and MAPK/ERK inhibition in pancreatic cancer. Autophagy, 2022, 18, 1737-1739.	9.1	7
3	Concurrent Inhibition of IGF1R and ERK Increases Pancreatic Cancer Sensitivity to Autophagy Inhibitors. Cancer Research, 2022, 82, 586-598.	0.9	27
4	KRAS-dependent cancer cells promote survival by producing exosomes enriched in Survivin. Cancer Letters, 2021, 517, 66-77.	7.2	22
5	CHK1 protects oncogenic KRAS-expressing cells from DNA damage and is a target for pancreatic cancer treatment. Cell Reports, 2021, 37, 110060.	6.4	14
6	Atypical KRASG12R Mutant Is Impaired in PI3K Signaling and Macropinocytosis in Pancreatic Cancer. Cancer Discovery, 2020, 10, 104-123.	9.4	131
7	Genome-wide DNA methylation analysis of KRAS mutant cell lines. Scientific Reports, 2020, 10, 10149.	3.3	7
8	The Sustained Induction of c-MYC Drives Nab-Paclitaxel Resistance in Primary Pancreatic Ductal Carcinoma Cells. Molecular Cancer Research, 2019, 17, 1815-1827.	3.4	40
9	Combination of ERK and autophagy inhibition as a treatment approach for pancreatic cancer. Nature Medicine, 2019, 25, 628-640.	30.7	476
10	Blocking autophagy to starve pancreatic cancer. Nature Reviews Molecular Cell Biology, 2019, 20, 265-265.	37.0	18
11	KRAS Suppression-Induced Degradation of MYC Is Antagonized by a MEK5-ERK5 Compensatory Mechanism. Cancer Cell, 2018, 34, 807-822.e7.	16.8	112
12	Mutant RAS Calms Stressed-Out Cancer Cells. Developmental Cell, 2017, 40, 120-122.	7.0	5
13	KRAS Mutant Pancreatic Cancer: No Lone Path to an Effective Treatment. Cancers, 2016, 8, 45.	3.7	147
14	Long-Term ERK Inhibition in KRAS-Mutant Pancreatic Cancer Is Associated with MYC Degradation and Senescence-like Growth Suppression. Cancer Cell, 2016, 29, 75-89.	16.8	191
15	A novel fluorescence-based biosynthetic trafficking method provides pharmacologic evidence that PI4-kinase IIIÎ± is important for protein trafficking from the endoplasmic reticulum to the plasma membrane. BMC Cell Biology, 2015, 16, 5.	3.0	5
16	Spatially Defined EGF Receptor Activation Reveals an F-Actin-Dependent Phospho-Erk Signaling Complex. Biophysical Journal, 2014, 107, 2639-2651.	0.5	20
17	Polyunsaturated fatty acids inhibit stimulated coupling between the ER Ca2+ sensor STIM1 and the Ca2+ channel protein Orai1 in a process that correlates with inhibition of stimulated STIM1 oligomerization. Biochimica Et Biophysica Acta - Molecular and Cell Biology of Lipids, 2014, 1841, 1210-1216.	2.4	18
18	KRAS: feeding pancreatic cancer proliferation. Trends in Biochemical Sciences, 2014, 39, 91-100.	7.5	546

#	Article	IF	Citations
19	Mutations in the Polybasic Juxtamembrane Sequence of Both Plasma Membrane- and Endoplasmic Reticulum-localized Epidermal Growth Factor Receptors Confer Ligand-independent Cell Transformation. Journal of Biological Chemistry, 2013, 288, 34930-34942.	3.4	9
20	Microâ€patterned arrays of epidermal growth factor (EGF) reveal stimulated association of paxillin, ERK, and Fâ€actin with EGF receptors during cell signaling. FASEB Journal, 2012, 26, 971.5.	0.5	0
21	Cancer cell-derived microvesicles induce transformation by transferring tissue transglutaminase and fibronectin to recipient cells. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 4852-4857.	7.1	415