

Shaofang Wu

List of Publications by Year in descending order

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Version: 2024-02-01

16
papers

365
citations

759233

12
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1058476

14
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16
docs citations

16
times ranked

649
citing authors

#	ARTICLE	IF	CITATIONS
1	PARP-mediated PARylation of MGMT is critical to promote repair of temozolomide-induced O6-methylguanine DNA damage in glioblastoma. <i>Neuro-Oncology</i> , 2021, 23, 920-931.	1.2	58
2	Integrated analysis of telomerase enzymatic activity unravels an association with cancer stemness and proliferation. <i>Nature Communications</i> , 2021, 12, 139.	12.8	39
3	EGFR Amplification Induces Increased DNA Damage Response and Renders Selective Sensitivity to Talazoparib (PARP Inhibitor) in Glioblastoma. <i>Clinical Cancer Research</i> , 2020, 26, 1395-1407.	7.0	26
4	Tie2-FGFR1 Interaction Induces Adaptive PI3K Inhibitor Resistance by Upregulating Aurora A/PLK1/CDK1 Signaling in Glioblastoma. <i>Cancer Research</i> , 2019, 79, 5088-5101.	0.9	17
5	Wild-type defined gamma-secretase inhibitor sensitivity and synergistic activity with doxorubicin in GSCs. <i>American Journal of Cancer Research</i> , 2019, 9, 1734-1745.	1.4	3
6	BRCA1 identified as a modulator of temozolomide resistance in P53 wild-type GBM using a high-throughput shRNA-based synthetic lethality screening. <i>American Journal of Cancer Research</i> , 2019, 9, 2428-2441.	1.4	1
7	Activation of WEE1 confers resistance to PI3K inhibition in glioblastoma. <i>Neuro-Oncology</i> , 2018, 20, 78-91.	1.2	24
8	DDIS-03. EGFR AMPLIFICATION INDUCED INCREASED DNA DAMAGE RESPONSE AND PREDICTED SELECTIVE SENSITIVITY TO TALAZOPARIB (PARP INHIBITOR) IN GLIOBLASTOMA STEM-LIKE CELLS. <i>Neuro-Oncology</i> , 2018, 20, vi69-vi69.	1.2	0
9	EXTH-11. GLIOBLASTOMA STEM CELL GROWTH DEPENDENCE ON NUTRIENTS: MORE THAN BASAL METABOLIC ACTIVITIES. <i>Neuro-Oncology</i> , 2018, 20, vi87-vi87.	1.2	0
10	The polo-like kinase 1 inhibitor volasertib synergistically increases radiation efficacy in glioma stem cells. <i>Oncotarget</i> , 2018, 9, 10497-10509.	1.8	18
11	Preclinical therapeutic efficacy of a novel blood-brain barrier-penetrant dual PI3K/mTOR inhibitor with preferential response in PI3K/PTEN mutant glioma. <i>Oncotarget</i> , 2017, 8, 21741-21753.	1.8	16
12	APOBEC3G acts as a therapeutic target in mesenchymal gliomas by sensitizing cells to radiation-induced cell death. <i>Oncotarget</i> , 2017, 8, 54285-54296.	1.8	15
13	MSK1-Mediated β -Catenin Phosphorylation Confers Resistance to PI3K/mTOR Inhibitors in Glioblastoma. <i>Molecular Cancer Therapeutics</i> , 2016, 15, 1656-1668.	4.1	25
14	Secondary interaction between MDMX and p53 core domain inhibits p53 DNA binding. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2016, 113, E2558-63.	7.1	38
15	Autoinhibition of MDMX by intramolecular p53 mimicry. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, 4624-4629.	7.1	43
16	Casein Kinase 1 α Regulates an MDMX Intramolecular Interaction To Stimulate p53 Binding. <i>Molecular and Cellular Biology</i> , 2012, 32, 4821-4832.	2.3	42