Ahmet Cingoz

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Identification of SERPINE1 as a Regulator of Glioblastoma Cell Dispersal with Transcriptome Profiling. Cancers, 2019, 11, 1651.	3.7	43
2	Quinacrine Mediated Sensitization of Glioblastoma (GBM) Cells to TRAIL through MMP-Sensitive PEG Hydrogel Carriers. Macromolecular Bioscience, 2017, 17, 1600267.	4.1	28
3	Systematic characterization of chromatin modifying enzymes identifies KDM3B as a critical regulator in castration resistant prostate cancer. Oncogene, 2020, 39, 2187-2201.	5.9	28
4	Identification of Mitoxantrone as a TRAIL-sensitizing agent for Glioblastoma Multiforme. Cancer Biology and Therapy, 2016, 17, 546-557.	3.4	27
5	KDM2B, an H3K36-specific demethylase, regulates apoptotic response of GBM cells to TRAIL. Cell Death and Disease, 2017, 8, e2897-e2897.	6.3	26
6	The pro-apoptotic Bcl-2 family member Harakiri (HRK) induces cell death in glioblastoma multiforme. Cell Death Discovery, 2019, 5, 64.	4.7	26
7	An exploration of plastic deformation dependence of cell viability and adhesion in metallic implant materials. Journal of the Mechanical Behavior of Biomedical Materials, 2016, 60, 177-186.	3.1	23
8	The fungal metabolite chaetocin is a sensitizer for pro-apoptotic therapies in glioblastoma. Cell Death and Disease, 2019, 10, 894.	6.3	21
9	Parameters Influencing Gene Delivery Efficiency of PEGylated Chitosan Nanoparticles: Experimental and Modeling Approach. Advanced NanoBiomed Research, 2022, 2, 2100033.	3.6	12
10	TRAIL-conjugated silver nanoparticles sensitize glioblastoma cells to TRAIL by regulating CHK1 in the DNA repair pathway. Neurological Research, 2020, 42, 1061-1069.	1.3	10
11	Generation of TRAIL-resistant cell line models reveals distinct adaptive mechanisms for acquired resistance and re-sensitization. Oncogene, 2021, 40, 3201-3216.	5.9	5
12	Macromol. Biosci. 2/2017. Macromolecular Bioscience, 2017, 17, .	4.1	1
13	Abstract 4164: TRAIL resistance of glioblastoma cells is associated with DNA damage signalling network. , 2017, , .		1
14	Drug Repositioning Screen on a New Primary Cell Line Identifies Potent Therapeutics for Glioblastoma. Frontiers in Neuroscience, 2020, 14, 578316.	2.8	1
15	EPIG-01THE FUNCTION OF CHROMATIN-MODIFYING ENZYMES IN GBM CELL APOPTOSIS. Neuro-Oncology, 2015, 17, v86.1-v86.	1.2	0
16	CBIO-08IGFBP2 IS A NOVEL MOLECULAR DETERMINANT IN TRAIL-RESISTANT SUBPOPULATIONS OF GBM CELL LINES. Neuro-Oncology, 2015, 17, v56.3-v56.	1.2	0
17	DRES-12. PROFILING OF DIFFERENT GBM CELL POPULATIONS WITH VARYING APOPTOTIC THRESHOLDS IDENTIFIES IGFBP-2 AS AÂNOVEL MEDIATOR OF TRAIL RESISTANCE. Neuro-Oncology, 2016, 18, vi54-vi54.	1.2	0
18	DDIS-04. MITOXANTRONE POTENTIATES TRAIL-INDUCED APOPTOSIS IN GLIOBLASTOMA MULTIFORME. Neuro-Oncology, 2016, 18, vi48-vi48.	1.2	0

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19	Mitoxantrone as a TRAIL-sensitizing agent for glioblastoma multiforme. European Journal of Cancer, 2016, 69, S81.	2.8	Ο
20	PO-306 Identification of chromatin modifiers regulating temozolomide resistance in glioblastoma multiforme. ESMO Open, 2018, 3, A140.	4.5	0
21	Abstract B73: Screen among 1200 FDA-approved drug library reveals mitoxantrone as a TRAIL-sensitizing agent for glioblastoma multiforme. , 2015, , .		Ο
22	EXTH-10. COMBINATION OF EPIGENETIC ENZYME INHIBITORS, GSK-J4 AND BELINOSTAT, REVEALS HIGH EFFICACY IN IDH1 MUTANT GLIOMAS. Neuro-Oncology, 2020, 22, ii88-ii89.	1.2	0