

Sandra Misale

List of Publications by Year in descending order

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Version: 2024-02-01

22
papers

4,438
citations

361413

20
h-index

677142

22
g-index

22
all docs

22
docs citations

22
times ranked

7606
citing authors

#	ARTICLE	IF	CITATIONS
1	Expanding the Reach of Precision Oncology by Drugging All <i>KRAS</i> Mutants. <i>Cancer Discovery</i> , 2022, 12, 924-937.	9.4	110
2	Anatomic position determines oncogenic specificity in melanoma. <i>Nature</i> , 2022, 604, 354-361.	27.8	44
3	Resistance is futile with fourth-generation EGFR inhibitors. <i>Nature Cancer</i> , 2022, 3, 381-383.	13.2	6
4	TRK xDFG Mutations Trigger a Sensitivity Switch from Type I to II Kinase Inhibitors. <i>Cancer Discovery</i> , 2021, 11, 126-141.	9.4	34
5	<i>KRAS</i> G12C Mutation Is Associated with Increased Risk of Recurrence in Surgically Resected Lung Adenocarcinoma. <i>Clinical Cancer Research</i> , 2021, 27, 2604-2612.	7.0	20
6	EGFR Blockade Reverts Resistance to KRASG12C Inhibition in Colorectal Cancer. <i>Cancer Discovery</i> , 2020, 10, 1129-1139.	9.4	245
7	HER2-Mediated Internalization of Cytotoxic Agents in <i>ERBB2</i> Amplified or Mutant Lung Cancers. <i>Cancer Discovery</i> , 2020, 10, 674-687.	9.4	149
8	Resistance to TRK inhibition mediated by convergent MAPK pathway activation. <i>Nature Medicine</i> , 2019, 25, 1422-1427.	30.7	144
9	Targeting the CBM complex causes Treg cells to prime tumours for immune checkpoint therapy. <i>Nature</i> , 2019, 570, 112-116.	27.8	147
10	KRAS G12C NSCLC Models Are Sensitive to Direct Targeting of KRAS in Combination with PI3K Inhibition. <i>Clinical Cancer Research</i> , 2019, 25, 796-807.	7.0	175
11	Restoring PUMA induction overcomes KRAS-mediated resistance to anti-EGFR antibodies in colorectal cancer. <i>Oncogene</i> , 2018, 37, 4599-4610.	5.9	30
12	MM-151 overcomes acquired resistance to cetuximab and panitumumab in colorectal cancers harboring EGFR extracellular domain mutations. <i>Science Translational Medicine</i> , 2016, 8, 324ra14.	12.4	81
13	Acquired Resistance to the TRK Inhibitor Entrectinib in Colorectal Cancer. <i>Cancer Discovery</i> , 2016, 6, 36-44.	9.4	258
14	Sensitivity to Entrectinib Associated With a Novel LMNA-NTRK1 Gene Fusion in Metastatic Colorectal Cancer. <i>Journal of the National Cancer Institute</i> , 2016, 108, .	6.3	111
15	Emergence of Multiple <i>EGFR</i> Extracellular Mutations during Cetuximab Treatment in Colorectal Cancer. <i>Clinical Cancer Research</i> , 2015, 21, 2157-2166.	7.0	227
16	Vertical suppression of the EGFR pathway prevents onset of resistance in colorectal cancers. <i>Nature Communications</i> , 2015, 6, 8305.	12.8	97
17	Blockade of EGFR and MEK Intercepts Heterogeneous Mechanisms of Acquired Resistance to Anti-EGFR Therapies in Colorectal Cancer. <i>Science Translational Medicine</i> , 2014, 6, 224ra26.	12.4	228
18	Resistance to Anti-EGFR Therapy in Colorectal Cancer: From Heterogeneity to Convergent Evolution. <i>Cancer Discovery</i> , 2014, 4, 1269-1280.	9.4	415

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19	TGFÎ± and Amphiregulin Paracrine Network Promotes Resistance to EGFR Blockade in Colorectal Cancer Cells. <i>Clinical Cancer Research</i> , 2014, 20, 6429-6438.	7.0	101
20	KRAS gene amplification in colorectal cancer and impact on response to EGFR-targeted therapy. <i>International Journal of Cancer</i> , 2013, 133, 1259-1265.	5.1	154
21	STAT3 can serve as a hit in the process of malignant transformation of primary cells. <i>Cell Death and Differentiation</i> , 2012, 19, 1390-1397.	11.2	57
22	Emergence of KRAS mutations and acquired resistance to anti-EGFR therapy in colorectal cancer. <i>Nature</i> , 2012, 486, 532-536.	27.8	1,605