

Giulia Siravegna

List of Publications by Year in Descending Order

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Version: 2024-04-10

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

32 papers	9,742 citations	28 h-index	35 g-index
35 ext. papers	11,711 ext. citations	17 avg, IF	5.58 L-index

#	Paper	IF	Citations
32	Cancer-Associated Fibroblasts: Versatile Players in the Tumor Microenvironment. <i>Cancers</i> , 2020 , 12,	6.6	32
31	A Genomic Analysis Workflow for Colorectal Cancer Precision Oncology. <i>Clinical Colorectal Cancer</i> , 2019 , 18, 91-101.e3	3.8	15
30	Whole exome sequencing analysis of urine trans-renal tumour DNA in metastatic colorectal cancer patients. <i>ESMO Open</i> , 2019 , 4,	6	12
29	Parallel Evaluation of Circulating Tumor DNA and Circulating Tumor Cells in Metastatic Colorectal Cancer. <i>Clinical Colorectal Cancer</i> , 2018 , 17, 80-83	3.8	34
28	Mutation-Enrichment Next-Generation Sequencing for Quantitative Detection of Mutations in Urine Cell-Free DNA from Patients with Advanced Cancers. <i>Clinical Cancer Research</i> , 2017 , 23, 3657-3666 ^{12.9}	12.9	44
27	Integrating liquid biopsies into the management of cancer. <i>Nature Reviews Clinical Oncology</i> , 2017 , 14, 531-548	19.4	970
26	Tracking a CAD-ALK gene rearrangement in urine and blood of a colorectal cancer patient treated with an ALK inhibitor. <i>Annals of Oncology</i> , 2017 , 28, 1302-1308	10.3	23
25	Polyclonal Secondary Mutations Drive Acquired Resistance to FGFR Inhibition in Patients with FGFR2 Fusion-Positive Cholangiocarcinoma. <i>Cancer Discovery</i> , 2017 , 7, 252-263	24.4	262
24	Genetic Evolution of Glioblastoma Stem-Like Cells From Primary to Recurrent Tumor. <i>Stem Cells</i> , 2017 , 35, 2218-2228	5.8	30
23	Inactivation of DNA repair triggers neoantigen generation and impairs tumour growth. <i>Nature</i> , 2017 , 552, 116-120	50.4	290
22	Emergence of MET hyper-amplification at progression to MET and BRAF inhibition in colorectal cancer. <i>British Journal of Cancer</i> , 2017 , 117, 347-352	8.7	22
21	Heterogeneity of Acquired Resistance to Anti-EGFR Monoclonal Antibodies in Patients with Metastatic Colorectal Cancer. <i>Clinical Cancer Research</i> , 2017 , 23, 2414-2422	12.9	111
20	MET-Driven Resistance to Dual EGFR and BRAF Blockade May Be Overcome by Switching from EGFR to MET Inhibition in BRAF-Mutated Colorectal Cancer. <i>Cancer Discovery</i> , 2016 , 6, 963-71	24.4	71
19	Molecular Landscape of Acquired Resistance to Targeted Therapy Combinations in BRAF-Mutant Colorectal Cancer. <i>Cancer Research</i> , 2016 , 76, 4504-15	10.1	63
18	Blood circulating tumor DNA for non-invasive genotyping of colon cancer patients. <i>Molecular Oncology</i> , 2016 , 10, 475-80	7.9	43
17	The First-in-class Anti-EGFR Antibody Mixture Sym004 Overcomes Cetuximab Resistance Mediated by EGFR Extracellular Domain Mutations in Colorectal Cancer. <i>Clinical Cancer Research</i> , 2016 , 22, 3260-7 ^{12.9}	12.9	48
16	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	17.5	61

15	Tumor cells can follow distinct evolutionary paths to become resistant to epidermal growth factor receptor inhibition. <i>Nature Medicine</i> , 2016 , 22, 262-9	50.5	533
14	Tumor Heterogeneity and Lesion-Specific Response to Targeted Therapy in Colorectal Cancer. <i>Cancer Discovery</i> , 2016 , 6, 147-153	24.4	255
13	Acquired Resistance to the TRK Inhibitor Entrectinib in Colorectal Cancer. <i>Cancer Discovery</i> , 2016 , 6, 36-44	24.4	200
12	Acquired RAS or EGFR mutations and duration of response to EGFR blockade in colorectal cancer. <i>Nature Communications</i> , 2016 , 7, 13665	17.4	121
11	Dual-targeted therapy with trastuzumab and lapatinib in treatment-refractory, KRAS codon 12/13 wild-type, HER2-positive metastatic colorectal cancer (HERACLES): a proof-of-concept, multicentre, open-label, phase 2 trial. <i>Lancet Oncology, The</i> , 2016 , 17, 738-746	21.7	533
10	Digital PCR quantification of MGMT methylation refines prediction of clinical benefit from alkylating agents in glioblastoma and metastatic colorectal cancer. <i>Annals of Oncology</i> , 2015 , 26, 1994-1999	10.3	93
9	Molecular Heterogeneity and Receptor Coamplification Drive Resistance to Targeted Therapy in MET-Amplified Esophagogastric Cancer. <i>Cancer Discovery</i> , 2015 , 5, 1271-81	24.4	126
8	Clonal evolution and resistance to EGFR blockade in the blood of colorectal cancer patients. <i>Nature Medicine</i> , 2015 , 21, 795-801	50.5	557
7	Emergence of Multiple EGFR Extracellular Mutations during Cetuximab Treatment in Colorectal Cancer. <i>Clinical Cancer Research</i> , 2015 , 21, 2157-66	12.9	173
6	Minimal residual disease in breast cancer: in blood veritas. <i>Clinical Cancer Research</i> , 2014 , 20, 2505-7	12.9	14
5	Genotyping cell-free tumor DNA in the blood to detect residual disease and drug resistance. <i>Genome Biology</i> , 2014 , 15, 449	18.3	63
4	Detection of circulating tumor DNA in early- and late-stage human malignancies. <i>Science Translational Medicine</i> , 2014 , 6, 224ra24	17.5	2741
3	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	17.5	203
2	Amplification of the MET receptor drives resistance to anti-EGFR therapies in colorectal cancer. <i>Cancer Discovery</i> , 2013 , 3, 658-73	24.4	489
1	Emergence of KRAS mutations and acquired resistance to anti-EGFR therapy in colorectal cancer. <i>Nature</i> , 2012 , 486, 532-6	50.4	1327