

Francesco Galimi

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/5238612/publications.pdf>

Version: 2024-02-01

19
papers

3,031
citations

430874

18
h-index

752698

20
g-index

21
all docs

21
docs citations

21
times ranked

5775
citing authors

#	ARTICLE	IF	CITATIONS
1	A Molecularly Annotated Platform of Patient-Derived Xenografts (â€œXenopatientsâ€) Identifies HER2 as an Effective Therapeutic Target in Cetuximab-Resistant Colorectal Cancer. <i>Cancer Discovery</i> , 2011, 1, 508-523.	9.4	818
2	Amplification of the <i>MET</i> Receptor Drives Resistance to Anti-EGFR Therapies in Colorectal Cancer. <i>Cancer Discovery</i> , 2013, 3, 658-673.	9.4	585
3	Intrinsic Resistance to MEK Inhibition in KRAS Mutant Lung and Colon Cancer through Transcriptional Induction of ERBB3. <i>Cell Reports</i> , 2014, 7, 86-93.	6.4	266
4	HER2 Activating Mutations Are Targets for Colorectal Cancer Treatment. <i>Cancer Discovery</i> , 2015, 5, 832-841.	9.4	250
5	Selective analysis of cancer-cell intrinsic transcriptional traits defines novel clinically relevant subtypes of colorectal cancer. <i>Nature Communications</i> , 2017, 8, 15107.	12.8	213
6	Inhibition of MEK and PI3K/mTOR Suppresses Tumor Growth but Does Not Cause Tumor Regression in Patient-Derived Xenografts of RAS-Mutant Colorectal Carcinomas. <i>Clinical Cancer Research</i> , 2012, 18, 2515-2525.	7.0	172
7	Conservation of copy number profiles during engraftment and passaging of patient-derived cancer xenografts. <i>Nature Genetics</i> , 2021, 53, 86-99.	21.4	118
8	Genetic and Expression Analysis of MET, MACC1, and HGF in Metastatic Colorectal Cancer: Response to Met Inhibition in Patient Xenografts and Pathologic Correlations. <i>Clinical Cancer Research</i> , 2011, 17, 3146-3156.	7.0	113
9	IGF2 is an actionable target that identifies a distinct subpopulation of colorectal cancer patients with marginal response to anti-EGFR therapies. <i>Science Translational Medicine</i> , 2015, 7, 272ra12.	12.4	100
10	Only a Subset of Met-Activated Pathways Are Required to Sustain Oncogene Addiction. <i>Science Signaling</i> , 2009, 2, ra80.	3.6	84
11	Met signaling regulates growth, repopulating potential and basal cell-fate commitment of mammary luminal progenitors: implications for basal-like breast cancer. <i>Oncogene</i> , 2013, 32, 1428-1440.	5.9	53
12	MACC1 mRNA Levels Predict Cancer Recurrence After Resection of Colorectal Cancer Liver Metastases. <i>Annals of Surgery</i> , 2013, 257, 1089-1095.	4.2	44
13	Colorectal cancer residual disease at maximal response to EGFR blockade displays a druggable Paneth cell-like phenotype. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	40
14	Inhibition of Src Impairs the Growth of Met-Addicted Gastric Tumors. <i>Clinical Cancer Research</i> , 2010, 16, 3933-3943.	7.0	39
15	Met inhibition revokes IFN γ -induction of PD-1 ligands in MET-amplified tumours. <i>British Journal of Cancer</i> , 2019, 120, 527-536.	6.4	34
16	The hepatocyte growth factor and its receptor. <i>Stem Cells</i> , 1993, 11, 22-30.	3.2	33
17	A Correction to the Research Article Titled: "Only a Subset of Met-Activated Pathways Are Required to Sustain Oncogene Addiction" by A. Bertotti, M. F. Burbidge, S. Gastaldi, F. Galimi, D. Torti, E. Medico, S. Giordano, S. Corso, G. Rolland-Valognes, B. P. Lockhart, J. A. Hickman, P. M. Comoglio, L. Trusolino. <i>Science Signaling</i> , 2009, 2, er11.	3.6	23
18	A preclinical algorithm of soluble surrogate biomarkers that correlate with therapeutic inhibition of the MET oncogene in gastric tumors. <i>International Journal of Cancer</i> , 2012, 130, 1357-1366.	5.1	21

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19	Detection of human androgen receptor mRNA in hepatocellular carcinoma by <i>in situ</i> hybridisation. Liver, 1994, 14, 213-219.	0.1	18