Francesco Iorio

List of Publications by Year
in descending order

| 72 papers | 15,311 citations | 101543
36 h-index | 106344
65 g-index |

| 96 all docs | 96 docs citations | 96 times ranked | 27723 citing authors |

#	Article	IF	Citations
1	Drug repurposing: progress, challenges and recommendations. Nature Reviews Drug Discovery, 2019, 18, 41-58.	46.4	2,689
2	Systematic identification of genomic markers of drug sensitivity in cancer cells. Nature, 2012, 483, 570-575.	27.8	2,173
3	Prospective Derivation of a Living Organoid Biobank of Colorectal Cancer Patients. Cell, 2015, 161, 933-945.	28.9	1,710
4	A Landscape of Pharmacogenomic Interactions in Cancer. Cell, 2016, 166, 740-754.	28.9	1,518
5	Prioritization of cancer therapeutic targets using CRISPR–Cas9 screens. Nature, 2019, 568, 511-516.	27.8	886
6	Discovery of drug mode of action and drug repositioning from transcriptional responses. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 14621-14626.	7.1	813
7	Heterogeneity of genomic evolution and mutational profiles in multiple myeloma. Nature Communications, 2014, 5, 2997.	12.8	741
8	A CRISPR Dropout Screen Identifies Genetic Vulnerabilities and Therapeutic Targets in Acute Myeloid Leukemia. Cell Reports, 2016, 17, 1193-1205.	6.4	556
9	Machine Learning Prediction of Cancer Cell Sensitivity to Drugs Based on Genomic and Chemical Properties. PLoS ONE, 2013, 8, e61318.	2.5	406
10	Pharmacogenomic agreement between two cancer cell line data sets. Nature, 2015, 528, 84-87.	27.8	358
11	A Yeast Synthetic Network for In Vivo Assessment of Reverse-Engineering and Modeling Approaches. Cell, 2009, 137, 172-181.	28.9	348
12	Characterizing Mutational Signatures in Human Cancer Cell Lines Reveals Episodic APOBEC Mutagenesis. Cell, 2019, 176, 1282-1294.e20.	28.9	298
13	Transcriptional data: a new gateway to drug repositioning?. Drug Discovery Today, 2013, 18, 350-357.	6.4	209
14	Transcription Factor Activities Enhance Markers of Drug Sensitivity in Cancer. Cancer Research, 2018, 78, 769-780.	0.9	161
15	Agreement between two large pan-cancer CRISPR-Cas9 gene dependency data sets. Nature Communications, 2019, 10, 5817.	12.8	160
16	Integrated cross-study datasets of genetic dependencies in cancer. Nature Communications, 2021, 12, 1661.	12.8	135
17	BRAF inhibitor resistance mediated by the AKT pathway in an oncogenic BRAF mouse melanoma model. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E536-45.	7.1	121
18	CellMinerCDB for Integrative Cross-Database Genomics and Pharmacogenomics Analyses of Cancer Cell Lines. IScience, 2018, 10, 247-264.	4.1	117

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19	Hemopoietic-specific Sf3b1-K700E knock-in mice display the splicing defect seen in human MDS but develop anemia without ring sideroblasts. Leukemia, 2017, 31, 720-727.	7.2	105
20	Transcriptional gene network inference from a massive dataset elucidates transcriptome organization and gene function. Nucleic Acids Research, 2011, 39, 8677-8688.	14.5	102
21	Identifying Network of Drug Mode of Action by Gene Expression Profiling. Journal of Computational Biology, 2009, 16, 241-251.	1.6	94
22	Functional linkage of gene fusions to cancer cell fitness assessed by pharmacological and CRISPR-Cas9 screening. Nature Communications, 2019, 10, 2198.	12.8	92
23	Unsupervised correction of gene-independent cell responses to CRISPR-Cas9 targeting. BMC Genomics, 2018, 19, 604.	2.8	75
24	DvD: An R/Cytoscape pipeline for drug repurposing using public repositories of gene expression data. Bioinformatics, 2013, 29, 132-134.	4.1	64
25	Blood transcriptomics of drug-naïve sporadic Parkinson's disease patients. BMC Genomics, 2015, 16, 876.	2.8	64
26	JACKS: joint analysis of CRISPR/Cas9 knockout screens. Genome Research, 2019, 29, 464-471.	5.5	64
27	Drug mechanism-of-action discovery through the integration of pharmacological and CRISPR screens. Molecular Systems Biology, 2020, 16, e9405.	7.2	63
28	Phosphoproteomics data classify hematological cancer cell lines according to tumor type and sensitivity to kinase inhibitors. Genome Biology, 2013, 14, R37.	9.6	60
29	Combinatorial CRISPR screen identifies fitness effects of gene paralogues. Nature Communications, 2021, 12, 1302.	12.8	59
30	Identification of small molecules enhancing autophagic function from drug network analysis. Autophagy, 2010, 6, 1204-1205.	9.1	58
31	Multilevel models improve precision and speed of IC_{50} estimates. Pharmacogenomics, 2016, 17, 691-700.	1.3	57
32	Cancer research needs a better map. Nature, 2021, 589, 514-516.	27.8	57
33	Project Score database: a resource for investigating cancer cell dependencies and prioritizing therapeutic targets. Nucleic Acids Research, 2021, 49, D1365-D1372.	14.5	54
34	Network based elucidation of drug response: from modulators to targets. BMC Systems Biology, 2013, 7, 139.	3.0	47
35	GDSCTools for mining pharmacogenomic interactions in cancer. Bioinformatics, 2018, 34, 1226-1228.	4.1	45
36	Identification of drug-specific pathways based on gene expression data: application to drug induced lung injury. Integrative Biology (United Kingdom), 2015, 7, 904-920.	1.3	43

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37	Logic models to predict continuous outputs based on binary inputs with an application to personalized cancer therapy. Scientific Reports, 2016, 6, 36812.	3.3	43
38	Minimal genome-wide human CRISPR-Cas9 library. Genome Biology, 2021, 22, 40.	8.8	40
39	A Semi-Supervised Approach for Refining Transcriptional Signatures of Drug Response and Repositioning Predictions. PLoS ONE, 2015, 10, e0139446.	2.5	39
40	Efficient randomization of biological networks while preserving functional characterization of individual nodes. BMC Bioinformatics, 2016, 17, 542.	2.6	38
41	The germline genetic component of drug sensitivity in cancer cell lines. Nature Communications, 2018, 9, 3385.	12.8	38
42	Fast randomization of large genomic datasets while preserving alteration counts. Bioinformatics, 2014, 30, i617-i623.	4.1	36
43	Structural rearrangements generate cell-specific, gene-independent CRISPR-Cas9 loss of fitness effects. Genome Biology, 2019, 20, 27.	8.8	35
44	CELLector: Genomics-Guided Selection of Cancer InÂVitro Models. Cell Systems, 2020, 10, 424-432.e6.	6.2	35
45	Stem cell-like transcriptional reprogramming mediates metastatic resistance to mTOR inhibition. Oncogene, 2017, 36, 2737-2749.	5.9	34
46	Comprehensive Pharmacogenomic Profiling of Malignant Pleural Mesothelioma Identifies a Subgroup Sensitive to FGFR Inhibition. Clinical Cancer Research, 2018, 24, 84-94.	7.0	33
47	Interactive data analysis and clustering of genomic data. Neural Networks, 2008, 21, 368-378.	5.9	31
48	Artificial neural network analysis of circulating tumor cells in metastatic breast cancer patients. Breast Cancer Research and Treatment, 2011, 129, 451-458.	2.5	31
49	Transcriptional response networks for elucidating mechanisms of action of multitargeted agents. Drug Discovery Today, 2016, 21, 1063-1075.	6.4	28
50	Pathway-based dissection of the genomic heterogeneity of cancer hallmarksâ€™ acquisition with SLAPenrich. Scientific Reports, 2018, 8, 6713.	3.3	24
51	Unravelling druggable signalling networks that control F508del-CFTR proteostasis. ELife, 2015, 4, .	6.0	22
52	Genome-wide chemical mutagenesis screens allow unbiased saturation of the cancer genome and identification of drug resistance mutations. Genome Research, 2017, 27, 613-625.	5.5	20
53	Loss of functional BAP1 augments sensitivity to TRAIL in cancer cells. ELife, 2018, 7, .	6.0	20
54	CoRe: a robustly benchmarked R package for identifying core-fitness genes in genome-wide pooled CRISPR-Cas9 screens. BMC Genomics, 2021, 22, 828.	2.8	14

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55	Integrated transcriptomic and proteomic analysis identifies protein kinase CK2 as a key signaling node in an inflammatory cytokine network in ovarian cancer cells. Oncotarget, 2016, 7, 15648-15661.	1.8	13
56	Computational estimation of quality and clinical relevance of cancer cell lines. Molecular Systems Biology, 2022, 18, .	7.2	12
57	Cancer develops, progresses and responds to therapies through restricted perturbation of the protein–protein interaction network. Integrative Biology (United Kingdom), 2012, 4, 1038.	1.3	10
58	Analysis of CRISPR–Cas9 screens identifies genetic dependencies in melanoma. Pigment Cell and Melanoma Research, 2021, 34, 122-131.	3.3	10
59	Functional Impact of Genomic Complexity on the Transcriptome of Multiple Myeloma. Clinical Cancer Research, 2021, 27, 6479-6490.	7.0	9
60	Abstract A44: A landscape of pharmacogenomic interactions in cancer. , 2017, , .		9
61	NIRest: A Tool for Gene Network and Mode of Action Inference. Annals of the New York Academy of Sciences, 2009, 1158, 257-264.	3.8	8
62	Identification of Intrinsic Drug Resistance and Its Biomarkers in High-Throughput Pharmacogenomic and CRISPR Screens. Patterns, 2020, 1, 100065.	5.9	6
63	Exploiting combinatorial patterns in cancer genomic data for personalized therapy and new target discovery. Pharmacogenomics, 2014, 15, 1943-1946.	1.3	5
64	Redefining false discoveries in cancer data analyses. Nature Computational Science, 2021, 1, 22-23.	8.0	1
65	Building Maps of Drugs Mode-of-Action from Gene Expression Data. Lecture Notes in Computer Science, 2009, , 56-65.	1.3	1
66	A Crispr/Cas9 Drop-out Screen Identifies Genome-Wide Genetic Valnerubilies in Acute Myeloid Leukaemia. Blood, 2015, 126, 554-554.	1.4	1
67	Gene ontology fuzzy-enrichment analysis to investigate drug mode-of-action. , 2010, , .		0
68	High-Throughput Analysis of the Drug Mode of Action of PB28, MC18 and MC70, Three Cyclohexylpiperazine Derivative New Molecules. Lecture Notes in Computer Science, 2008, , 1085-1092.	1.3	0
69	Abstract 49: Targeting the TNF network in human ovarian cancer - A systems biology approach. , 2012, , .		0
70	Whole Exome Sequencing Of Multiple Myeloma Reveals An Heterogeneous Clonal Architecture and Genomic Evolution. Blood, 2013, 122, 399-399.	1.4	0
71	Abstract 2218: Genomic characterisation of 1015 cancer cell-lines. , 2014, , .		0
72	CellMinerCDB for Integrative Genomics and Pharmacogenomics Analyses of Cancer Cell Lines. SSRN Electronic Journal, 0, , .	0.4	0