

David E Root

List of Publications by Year in descending order

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Version: 2024-02-01

51
papers

23,392
citations

101384

36
h-index

182168

51
g-index

55
all docs

55
docs citations

55
times ranked

36837
citing authors

#	ARTICLE	IF	CITATIONS
1	Genome-Scale CRISPR-Cas9 Knockout Screening in Human Cells. <i>Science</i> , 2014, 343, 84-87.	6.0	4,210
2	Optimized sgRNA design to maximize activity and minimize off-target effects of CRISPR-Cas9. <i>Nature Biotechnology</i> , 2016, 34, 184-191.	9.4	3,168
3	A Next Generation Connectivity Map: L1000 Platform and the First 1,000,000 Profiles. <i>Cell</i> , 2017, 171, 1437-1452.e17.	13.5	2,281
4	Defining a Cancer Dependency Map. <i>Cell</i> , 2017, 170, 564-576.e16.	13.5	1,794
5	Computational correction of copy number effect improves specificity of CRISPR-Cas9 essentiality screens in cancer cells. <i>Nature Genetics</i> , 2017, 49, 1779-1784.	9.4	1,436
6	Rational design of highly active sgRNAs for CRISPR-Cas9-mediated gene inactivation. <i>Nature Biotechnology</i> , 2014, 32, 1262-1267.	9.4	1,351
7	COT drives resistance to RAF inhibition through MAP kinase pathway reactivation. <i>Nature</i> , 2010, 468, 968-972.	13.7	1,325
8	Identification of RPS14 as a 5q- syndrome gene by RNA interference screen. <i>Nature</i> , 2008, 451, 335-339.	13.7	850
9	KRAS and YAP1 Converge to Regulate EMT and Tumor Survival. <i>Cell</i> , 2014, 158, 171-184.	13.5	608
10	A public genome-scale lentiviral expression library of human ORFs. <i>Nature Methods</i> , 2011, 8, 659-661.	9.0	477
11	A melanocyte lineage program confers resistance to MAP kinase pathway inhibition. <i>Nature</i> , 2013, 504, 138-142.	13.7	401
12	<i>MTAP</i> deletion confers enhanced dependency on the PRMT5 arginine methyltransferase in cancer cells. <i>Science</i> , 2016, 351, 1214-1218.	6.0	396
13	Systematic investigation of genetic vulnerabilities across cancer cell lines reveals lineage-specific dependencies in ovarian cancer. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2011, 108, 12372-12377.	3.3	383
14	The landscape of cancer cell line metabolism. <i>Nature Medicine</i> , 2019, 25, 850-860.	15.2	350
15	Mutational processes shape the landscape of TP53 mutations in human cancer. <i>Nature Genetics</i> , 2018, 50, 1381-1387.	9.4	334
16	Parallel genome-scale loss of function screens in 216 cancer cell lines for the identification of context-specific genetic dependencies. <i>Scientific Data</i> , 2014, 1, 140035.	2.4	328
17	A Genome-Scale RNA Interference Screen Implicates NF1 Loss in Resistance to RAF Inhibition. <i>Cancer Discovery</i> , 2013, 3, 350-362.	7.7	299
18	Improved estimation of cancer dependencies from large-scale RNAi screens using model-based normalization and data integration. <i>Nature Communications</i> , 2018, 9, 4610.	5.8	290

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19	A dominant-negative effect drives selection of TP53 missense mutations in myeloid malignancies. <i>Science</i> , 2019, 365, 599-604.	6.0	265
20	WRN helicase is a synthetic lethal target in microsatellite unstable cancers. <i>Nature</i> , 2019, 568, 551-556.	13.7	253
21	A Genome-wide CRISPR Death Screen Identifies Genes Essential for Oxidative Phosphorylation. <i>Cell Metabolism</i> , 2016, 24, 875-885.	7.2	244
22	Orthologous CRISPR-Cas9 enzymes for combinatorial genetic screens. <i>Nature Biotechnology</i> , 2018, 36, 179-189.	9.4	216
23	Selective gene dependencies in MYCN-amplified neuroblastoma include the core transcriptional regulatory circuitry. <i>Nature Genetics</i> , 2018, 50, 1240-1246.	9.4	199
24	High-throughput Phenotyping of Lung Cancer Somatic Mutations. <i>Cancer Cell</i> , 2016, 30, 214-228.	7.7	171
25	Agreement between two large pan-cancer CRISPR-Cas9 gene dependency data sets. <i>Nature Communications</i> , 2019, 10, 5817.	5.8	160
26	A Functional Landscape of Resistance to ALK Inhibition in Lung Cancer. <i>Cancer Cell</i> , 2015, 27, 397-408.	7.7	150
27	Phenotypic Characterization of a Comprehensive Set of MAPK1 /ERK2 Missense Mutants. <i>Cell Reports</i> , 2016, 17, 1171-1183.	2.9	119
28	Cells Lacking the RB1 Tumor Suppressor Gene Are Hyperdependent on Aurora B Kinase for Survival. <i>Cancer Discovery</i> , 2019, 9, 230-247.	7.7	119
29	CRISPR-Cas9 screen reveals a MYCN-amplified neuroblastoma dependency on EZH2. <i>Journal of Clinical Investigation</i> , 2017, 128, 446-462.	3.9	117
30	Complementary information derived from CRISPR Cas9 mediated gene deletion and suppression. <i>Nature Communications</i> , 2017, 8, 15403.	5.8	93
31	Acquired FGFR and FGF Alterations Confer Resistance to Estrogen Receptor (ER) Targeted Therapy in ER+ Metastatic Breast Cancer. <i>Clinical Cancer Research</i> , 2020, 26, 5974-5989.	3.2	87
32	Noncanonical open reading frames encode functional proteins essential for cancer cell survival. <i>Nature Biotechnology</i> , 2021, 39, 697-704.	9.4	85
33	Csnk1a1 inhibition has p53-dependent therapeutic efficacy in acute myeloid leukemia. <i>Journal of Experimental Medicine</i> , 2014, 211, 605-612.	4.2	79
34	The Canonical Wnt Pathway Drives Macropinocytosis in Cancer. <i>Cancer Research</i> , 2018, 78, 4658-4670.	0.4	75
35	Paralog knockout profiling identifies DUSP4 and DUSP6 as a digenic dependence in MAPK pathway-driven cancers. <i>Nature Genetics</i> , 2021, 53, 1664-1672.	9.4	61
36	Genetic modifiers of EGFR dependence in non-small cell lung cancer. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2014, 111, 18661-18666.	3.3	46

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37	Neuronal differentiation and cell-cycle programs mediate response to BET-bromodomain inhibition in MYC-driven medulloblastoma. <i>Nature Communications</i> , 2019, 10, 2400.	5.8	37
38	A Functional Landscape of Resistance to MEK1/2 and CDK4/6 Inhibition in NRAS-Mutant Melanoma. <i>Cancer Research</i> , 2019, 79, 2352-2366.	0.4	34
39	Defining the landscape of ATP-competitive inhibitor resistance residues in protein kinases. <i>Nature Structural and Molecular Biology</i> , 2020, 27, 92-104.	3.6	30
40	LKB1/STK11 Is a Tumor Suppressor in the Progression of Myeloproliferative Neoplasms. <i>Cancer Discovery</i> , 2021, 11, 1398-1410.	7.7	29
41	Structure-function analysis of the SHOC2-MRAS-PP1C holophosphatase complex. <i>Nature</i> , 2022, 609, 408-415.	13.7	28
42	Progression signature underlies clonal evolution and dissemination of multiple myeloma. <i>Blood</i> , 2021, 137, 2360-2372.	0.6	26
43	PPM1D mutations are oncogenic drivers of de novo diffuse midline glioma formation. <i>Nature Communications</i> , 2022, 13, 604.	5.8	22
44	Selective Modulation of a Pan-Essential Protein as a Therapeutic Strategy in Cancer. <i>Cancer Discovery</i> , 2021, 11, 2282-2299.	7.7	21
45	Phosphate dysregulation via the XPR1-KIDINS220 protein complex is a therapeutic vulnerability in ovarian cancer. <i>Nature Cancer</i> , 2022, 3, 681-695.	5.7	21
46	Pooled Lentiviral Delivery Genetic Screens. <i>Current Protocols in Molecular Biology</i> , 2018, 121, 32.1.1-32.1.21.	2.9	20
47	Allosteric inhibition of PPM1D serine/threonine phosphatase via an altered conformational state. <i>Nature Communications</i> , 2022, 13, .	5.8	15
48	A Genome-scale CRISPR Screen Identifies the ERBB and mTOR Signaling Networks as Key Determinants of Response to PI3K Inhibition in Pancreatic Cancer. <i>Molecular Cancer Therapeutics</i> , 2020, 19, 1423-1435.	1.9	14
49	Systematic identification of biomarker-driven drug combinations to overcome resistance. <i>Nature Chemical Biology</i> , 2022, 18, 615-624.	3.9	14
50	A genome-wide gain-of-function screen identifies CDKN2C as a HBV host factor. <i>Nature Communications</i> , 2020, 11, 2707.	5.8	11
51	Genetic barcoding systematically compares genes in del(5q) MDS and reveals a central role for CSNK1A1 in clonal expansion. <i>Blood Advances</i> , 2022, 6, 1780-1796.	2.5	7