Aviad Tsherniak

List of Publications by Citations

74 papers	8,686 citations	39 h-index	82 g-index
82 ext. papers	12,789 ext. citations	22.5 avg, IF	5.22 L-index

#	Paper	IF	Citations
74	Next-generation characterization of the Cancer Cell Line Encyclopedia. *Nature*, **2019**, 569, 503-508	50.4	962
73	Defining a Cancer Dependency Map. *Cell*, **2017**, 170, 564-576.e16	56.2	844
72	Computational correction of copy number effect improves specificity of CRISPR-Cas9 essentiality screens in cancer cells. *Nature Genetics*, **2017**, 49, 1779-1784	36.3	740
71	Catenin-driven cancers require a YAP1 transcriptional complex for survival and tumorigenesis. *Cell*, **2012**, 151, 1457-73	56.2	522
70	Integrative genomic analysis of medulloblastoma identifies a molecular subgroup that drives poor clinical outcome. *Journal of Clinical Oncology*, **2011**, 29, 1424-30	2.2	513
69	Genetic and transcriptional evolution alters cancer cell line drug response. *Nature*, **2018**, 560, 325-330	50.4	379
68	Prognostically relevant gene signatures of high-grade serous ovarian carcinoma. *Journal of Clinical Investigation*, **2013**, 123, 517-25	15.9	371
67	Genomic Copy Number Dictates a Gene-Independent Cell Response to CRISPR/Cas9 Targeting. *Cancer Discovery*, **2016**, 6, 914-29	24.4	343
66	Systematic investigation of genetic vulnerabilities across cancer cell lines reveals lineage-specific dependencies in ovarian cancer. *Proceedings of the National Academy of Sciences of the United States of America*, **2011**, 108, 12372-7	11.5	321
65	SWI/SNF-mutant cancers depend on catalytic and non-catalytic activity of EZH2. *Nature Medicine*, **2015**, 21, 1491-6	50.5	252
64	Parallel genome-scale loss of function screens in 216 cancer cell lines for the identification of context-specific genetic dependencies. *Scientific Data*, **2014**, 1, 140035	8.2	251
63	MTAP deletion confers enhanced dependency on the PRMT5 arginine methyltransferase in cancer cells. *Science*, **2016**, 351, 1214-8	33.3	248
62	The landscape of cancer cell line metabolism. *Nature Medicine*, **2019**, 25, 850-860	50.5	188
61	Mutational processes shape the landscape of TP53 mutations in human cancer. *Nature Genetics*, **2018**, 50, 1381-1387	36.3	165
60	Cancer vulnerabilities unveiled by genomic loss. *Cell*, **2012**, 150, 842-54	56.2	163
59	Improved estimation of cancer dependencies from large-scale RNAi screens using model-based normalization and data integration. *Nature Communications*, **2018**, 9, 4610	17.4	155
58	WRN helicase is a synthetic lethal target in microsatellite unstable cancers. *Nature*, **2019**, 568, 551-556	50.4	137

57	Discovering the anti-cancer potential of non-oncology drugs by systematic viability profiling. *Nature Cancer*, **2020**, 1, 235-248	15.4	137
56	High-throughput identification of genotype-specific cancer vulnerabilities in mixtures of barcoded tumor cell lines. *Nature Biotechnology*, **2016**, 34, 419-23	44.5	127
55	SQSTM1 is a pathogenic target of 5q copy number gains in kidney cancer. *Cancer Cell*, **2013**, 24, 738-50	24.3	111
54	Selective gene dependencies in MYCN-amplified neuroblastoma include the core transcriptional regulatory circuitry. *Nature Genetics*, **2018**, 50, 1240-1246	36.3	94
53	ATARiS: computational quantification of gene suppression phenotypes from multisample RNAi screens. *Genome Research*, **2013**, 23, 665-78	9.7	93
52	Extracting Biological Insights from the Project Achilles Genome-Scale CRISPR Screens in Cancer Cell Lines		88
51	Identification of ADAR1 adenosine deaminase dependency in a subset of cancer cells. *Nature Communications*, **2018**, 9, 5450	17.4	83
50	CRISPR-Cas9 screen reveals a MYCN-amplified neuroblastoma dependency on EZH2. *Journal of Clinical Investigation*, **2018**, 128, 446-462	15.9	72
49	Agreement between two large pan-cancer CRISPR-Cas9 gene dependency data sets. *Nature Communications*, **2019**, 10, 5817	17.4	70
48	Somatic Superenhancer Duplications and Hotspot Mutations Lead to Oncogenic Activation of the KLF5 Transcription Factor. *Cancer Discovery*, **2018**, 8, 108-125	24.4	67
47	Complementary information derived from CRISPR Cas9 mediated gene deletion and suppression. *Nature Communications*, **2017**, 8, 15403	17.4	65
46	Interrogation of Mammalian Protein Complex Structure, Function, and Membership Using Genome-Scale Fitness Screens. *Cell Systems*, **2018**, 6, 555-568.e7	10.6	65
45	Pan-cancer single-cell RNA-seq identifies recurring programs of cellular heterogeneity. *Nature Genetics*, **2020**, 52, 1208-1218	36.3	63
44	Mitochondrial metabolism promotes adaptation to proteotoxic stress. *Nature Chemical Biology*, **2019**, 15, 681-689	11.7	62
43	Predicting relapse in patients with medulloblastoma by integrating evidence from clinical and genomic features. *Journal of Clinical Oncology*, **2011**, 29, 1415-23	2.2	58
42	Characterizing genomic alterations in cancer by complementary functional associations. *Nature Biotechnology*, **2016**, 34, 539-46	44.5	57
41	Targetable vulnerabilities in T- and NK-cell lymphomas identified through preclinical models. *Nature Communications*, **2018**, 9, 2024	17.4	54
40	Identification of cancer-cytotoxic modulators of PDE3A by predictive chemogenomics. *Nature Chemical Biology*, **2016**, 12, 102-8	11.7	51

39	Functional, chemical genomic, and super-enhancer screening identify sensitivity to cyclin D1/CDK4 pathway inhibition in Ewing sarcoma. *Oncotarget*, **2015**, 6, 30178-93	3.3	51
38	Copy-number and gene dependency analysis reveals partial copy loss of wild-type SF3B1 as a novel cancer vulnerability. *ELife*, **2017**, 6,	8.9	49
37	Multiplex CRISPR/Cas9-Based Genome Editing in Human Hematopoietic Stem Cells Models Clonal Hematopoiesis and Myeloid Neoplasia. *Cell Stem Cell*, **2017**, 21, 547-555.e8	18	43
36	Genome-scale CRISPR-Cas9 screen identifies druggable dependencies in wild-type Ewing sarcoma. *Journal of Experimental Medicine*, **2018**, 215, 2137-2155	16.6	43
35	Multiplexed single-cell transcriptional response profiling to define cancer vulnerabilities and therapeutic mechanism of action. *Nature Communications*, **2020**, 11, 4296	17.4	37
34	GeNets: a unified web platform for network-based genomic analyses. *Nature Methods*, **2018**, 15, 543-546	21.6	36
33	Genome-scale analysis identifies paralog lethality as a vulnerability of chromosome 1p loss in cancer. *Nature Genetics*, **2018**, 50, 937-943	36.3	35
32	Integrated genetic and pharmacologic interrogation of rare cancers. *Nature Communications*, **2016**, 7, 11987	17.4	32
31	An Embryonic Diapause-like Adaptation with Suppressed Myc Activity Enables Tumor Treatment Persistence. *Cancer Cell*, **2021**, 39, 240-256.e11	24.3	29
30	Decomposing Oncogenic Transcriptional Signatures to Generate Maps of Divergent Cellular States. *Cell Systems*, **2017**, 5, 105-118.e9	10.6	27
29	mutant tumors depend on oxoglutarate dehydrogenase. *Proceedings of the National Academy of Sciences of the United States of America*, **2017**, 114, E3434-E3443	11.5	24
28	MDM2 and MDM4 Are Therapeutic Vulnerabilities in Malignant Rhabdoid Tumors. *Cancer Research*, **2019**, 79, 2404-2414	10.1	24
27	PRMT1-Mediated Translation Regulation Is a Crucial Vulnerability of Cancer. *Cancer Research*, **2017**, 77, 4613-4625	10.1	21
26	A first-generation pediatric cancer dependency map. *Nature Genetics*, **2021**, 53, 529-538	36.3	21
25	Small-Molecule and CRISPR Screening Converge to Reveal Receptor Tyrosine Kinase Dependencies in Pediatric Rhabdoid Tumors. *Cell Reports*, **2019**, 28, 2331-2344.e8	10.6	20
24	Global computational alignment of tumor and cell line transcriptional profiles. *Nature Communications*, **2021**, 12, 22	17.4	19
23	Neuronal differentiation and cell-cycle programs mediate response to BET-bromodomain inhibition in MYC-driven medulloblastoma. *Nature Communications*, **2019**, 10, 2400	17.4	18
22	Genome-Wide Interrogation of Human Cancers Identifies EGLN1 Dependency in Clear Cell Ovarian Cancers. *Cancer Research*, **2019**, 79, 2564-2579	10.1	18

21	Integrated cross-study datasets of genetic dependencies in cancer. *Nature Communications*, **2021**, 12, 1661	17.4	17
20	Identification of an "Exceptional Responder" Cell Line to MEK1 Inhibition: Clinical Implications for MEK-Targeted Therapy. *Molecular Cancer Research*, **2016**, 14, 207-15	6.6	16
19	Functional Genomics Identify Distinct and Overlapping Genes Mediating Resistance to Different Classes of Heterobifunctional Degraders of Oncoproteins. *Cell Reports*, **2021**, 34, 108532	10.6	15
18	A Community Challenge for Inferring Genetic Predictors of Gene Essentialities through Analysis of a Functional Screen of Cancer Cell Lines. *Cell Systems*, **2017**, 5, 485-497.e3	10.6	14
17	Gene expression has more power for predicting in vitro cancer cell vulnerabilities than genomics		12
16	Functional Genomic Characterization of Cancer Genomes. *Cold Spring Harbor Symposia on Quantitative Biology*, **2016**, 81, 237-246	3.9	11
15	Open Community Challenge Reveals Molecular Network Modules with Key Roles in Diseases		10
14	Pan-cancer single cell RNA-seq uncovers recurring programs of cellular heterogeneity		9
13	Genome-scale screens identify factors regulating tumor cell responses to natural killer cells. *Nature Genetics*, **2021**, 53, 1196-1206	36.3	9
12	Chronos: a cell population dynamics model of CRISPR experiments that improves inference of gene fitness effects.. *Genome Biology*, **2021**, 22, 343	18.3	8
11	Synthetic Lethal Interaction between the ESCRT Paralog Enzymes VPS4A and VPS4B in Cancers Harboring Loss of Chromosome 18q or 16q. *Cell Reports*, **2020**, 33, 108493	10.6	7
10	Catenin-Driven Cancers Require a YAP1 Transcriptional Complex for Survival and Tumorigenesis. *Cell*, **2013**, 153, 267-270	56.2	6
9	Non-oncology drugs are a source of previously unappreciated anti-cancer activity		6
8	Multiplexed single-cell profiling of post-perturbation transcriptional responses to define cancer vulnerabilities and therapeutic mechanism of action		3
7	Integrated cross-study datasets of genetic dependencies in cancer		3
6	Improved estimation of cancer dependencies from large-scale RNAi screens using model-based normalization and data integration		2
5	Global computational alignment of tumor and cell line transcriptional profiles		2
4	Agreement between two large pan-cancer CRISPR-Cas9 gene dependency datasets		2

3	Chronos: a CRISPR cell population dynamics model	2
2	Computational correction of copy-number effect improves specificity of CRISPR-Cas9 essentiality screens in cancer cells	1
1	Post-perturbational transcriptional signatures of cancer cell line vulnerabilities	1