

Rugang Zhang

List of Publications by Year in descending order

Source: <https://exaly.com/author-pdf/5867612/publications.pdf>

Version: 2024-02-01

87
papers

7,516
citations

61984

43
h-index

56724

83
g-index

87
all docs

87
docs citations

87
times ranked

12443
citing authors

#	ARTICLE	IF	CITATIONS
1	Chromatin basis of the senescence-associated secretory phenotype. <i>Trends in Cell Biology</i> , 2022, 32, 513-526.	7.9	29
2	Targeting cellular senescence to combat cancer and ageing. <i>Molecular Oncology</i> , 2022, 16, 3319-3332.	4.6	6
3	KDM5A Inhibits Antitumor Immune Responses Through Downregulation of the Antigen-Presentation Pathway in Ovarian Cancer. <i>Cancer Immunology Research</i> , 2022, 10, 1028-1038.	3.4	6
4	ADAR1 downregulation by autophagy drives senescence independently of RNA editing by enhancing p16INK4a levels. <i>Nature Cell Biology</i> , 2022, 24, 1202-1210.	10.3	19
5	ARID2 Deficiency Correlates with the Response to Immune Checkpoint Blockade in Melanoma. <i>Journal of Investigative Dermatology</i> , 2021, 141, 1564-1572.e4.	0.7	20
6	Sensitization of ovarian tumor to immune checkpoint blockade by boosting senescence-associated secretory phenotype. <i>IScience</i> , 2021, 24, 102016.	4.1	32
7	Targeting glutamine dependence through GLS1 inhibition suppresses ARID1A-inactivated clear cell ovarian carcinoma. <i>Nature Cancer</i> , 2021, 2, 189-200.	13.2	36
8	m6A-independent genome-wide METTL3 and METTL14 redistribution drives the senescence-associated secretory phenotype. <i>Nature Cell Biology</i> , 2021, 23, 355-365.	10.3	71
9	Targeting the IRE1 α /XBP1 Endoplasmic Reticulum Stress Response Pathway in <i>ARID1A</i> -Mutant Ovarian Cancers. <i>Cancer Research</i> , 2021, 81, 5325-5335.	0.9	12
10	Targeting the IRE1 α /XBP1s pathway suppresses CARM1-expressing ovarian cancer. <i>Nature Communications</i> , 2021, 12, 5321.	12.8	17
11	The SETDB1 α -TRIM28 Complex Suppresses Antitumor Immunity. <i>Cancer Immunology Research</i> , 2021, 9, 1413-1424.	3.4	24
12	NAMPT Inhibition Suppresses Cancer Stem-like Cells Associated with Therapy-Induced Senescence in Ovarian Cancer. <i>Cancer Research</i> , 2020, 80, 890-900.	0.9	83
13	CD38-expressing macrophages drive age-related NAD ⁺ decline. <i>Nature Metabolism</i> , 2020, 2, 1186-1187.	11.9	7
14	Reactivation of dormant tumor cells by modified lipids derived from stress-activated neutrophils. <i>Science Translational Medicine</i> , 2020, 12, .	12.4	107
15	Combining PARP with ATR inhibition overcomes PARP inhibitor and platinum resistance in ovarian cancer models. <i>Nature Communications</i> , 2020, 11, 3726.	12.8	169
16	Targeting CARM1 in ovarian cancer with EZH2 and PARP inhibitors. <i>Molecular and Cellular Oncology</i> , 2020, 7, 1760675.	0.7	3
17	ARID1A mutation and genomic stability. <i>Molecular and Cellular Oncology</i> , 2020, 7, 1690923.	0.7	1
18	Integrative comparison of the genomic and transcriptomic landscape between prostate cancer patients of predominantly African or European genetic ancestry. <i>PLoS Genetics</i> , 2020, 16, e1008641.	3.5	78

#	ARTICLE	IF	CITATIONS
19	Topoisomerase 1 cleavage complex enables pattern recognition and inflammation during senescence. <i>Nature Communications</i> , 2020, 11, 908.	12.8	36
20	EZH2 Inhibition Sensitizes CARM1-High, Homologous Recombination Proficient Ovarian Cancers to PARP Inhibition. <i>Cancer Cell</i> , 2020, 37, 157-167.e6.	16.8	79
21	Selective targeting of different populations of myeloid-derived suppressor cells by histone deacetylase inhibitors. <i>Cancer Immunology, Immunotherapy</i> , 2020, 69, 1929-1936.	4.2	39
22	Epigenetic driver mutations in ARID1A shape cancer immune phenotype and immunotherapy. <i>Journal of Clinical Investigation</i> , 2020, 130, 2712-2726.	8.2	112
23	HDAC6 Inhibition Synergizes with Anti-PD-L1 Therapy in ARID1A-Inactivated Ovarian Cancer. <i>Cancer Research</i> , 2019, 79, 5482-5489.	0.9	86
24	ARID1A promotes genomic stability through protecting telomere cohesion. <i>Nature Communications</i> , 2019, 10, 4067.	12.8	40
25	ARID1A spatially partitions interphase chromosomes. <i>Science Advances</i> , 2019, 5, eaaw5294.	10.3	35
26	NAD ⁺ metabolism controls inflammation during senescence. <i>Molecular and Cellular Oncology</i> , 2019, 6, 1605819.	0.7	8
27	N6-Methylation of Adenosine of <i>FZD10</i> mRNA Contributes to PARP Inhibitor Resistance. <i>Cancer Research</i> , 2019, 79, 2812-2820.	0.9	127
28	NAD ⁺ metabolism governs the proinflammatory senescence-associated secretome. <i>Nature Cell Biology</i> , 2019, 21, 397-407.	10.3	232
29	Involvement of condensin in cellular senescence through gene regulation and compartmental reorganization. <i>Nature Communications</i> , 2019, 10, 5688.	12.8	42
30	CARM1-expressing ovarian cancer depends on the histone methyltransferase EZH2 activity. <i>Nature Communications</i> , 2018, 9, 631.	12.8	72
31	Repurposing Pan-HDAC Inhibitors for ARID1A-Mutated Ovarian Cancer. <i>Cell Reports</i> , 2018, 22, 3393-3400.	6.4	77
32	Mouse embryonic stem cells have increased capacity for replication fork restart driven by the specific FliA-Floped protein complex. <i>Cell Research</i> , 2018, 28, 69-89.	12.0	31
33	Epigenetic inhibitors for the precision treatment of ARID1A-mutant ovarian cancers: what are the next steps?. <i>Expert Review of Precision Medicine and Drug Development</i> , 2018, 3, 233-236.	0.7	3
34	Integrated Analysis of Genetic Ancestry and Genomic Alterations across Cancers. <i>Cancer Cell</i> , 2018, 34, 549-560.e9.	16.8	168
35	CLIC1 and CLIC4 complement CA125 as a diagnostic biomarker panel for all subtypes of epithelial ovarian cancer. <i>Scientific Reports</i> , 2018, 8, 14725.	3.3	35
36	SWI/SNF catalytic subunits TM switch drives resistance to EZH2 inhibitors in ARID1A-mutated cells. <i>Nature Communications</i> , 2018, 9, 4116.	12.8	38

#	ARTICLE	IF	CITATIONS
37	The Tumor Suppressor ARID1A Controls Global Transcription via Pausing of RNA Polymerase II. <i>Cell Reports</i> , 2018, 23, 3933-3945.	6.4	83
38	SWI/SNF Complexes in Ovarian Cancer: Mechanistic Insights and Therapeutic Implications. <i>Molecular Cancer Research</i> , 2018, 16, 1819-1825.	3.4	32
39	Tailoring Chemotherapy for the African-Centric S47 Variant of TP53. <i>Cancer Research</i> , 2018, 78, 5694-5705.	0.9	9
40	SATB1 Expression Governs Epigenetic Repression of PD-1 in Tumor-Reactive T Cells. <i>Immunity</i> , 2017, 46, 51-64.	14.3	122
41	Targeting the ATR/CHK1 Axis with PARP Inhibition Results in Tumor Regression in <i>BRCA</i> -Mutant Ovarian Cancer Models. <i>Clinical Cancer Research</i> , 2017, 23, 3097-3108.	7.0	223
42	ARID1A-mutated ovarian cancers depend on HDAC6 activity. <i>Nature Cell Biology</i> , 2017, 19, 962-973.	10.3	173
43	Detection of the Ubiquitinome in Cells Undergoing Oncogene-Induced Senescence. <i>Methods in Molecular Biology</i> , 2017, 1534, 127-137.	0.9	1
44	Tumor Cell-Independent Estrogen Signaling Drives Disease Progression through Mobilization of Myeloid-Derived Suppressor Cells. <i>Cancer Discovery</i> , 2017, 7, 72-85.	9.4	153
45	Harnessing mutual exclusivity between TP53 and ARID1A mutations. <i>Cell Cycle</i> , 2017, 16, 2313-2314.	2.6	7
46	BET Bromodomain Inhibition Synergizes with PARP Inhibitor in Epithelial Ovarian Cancer. <i>Cell Reports</i> , 2017, 21, 3398-3405.	6.4	130
47	Epigenetic Basis of Cellular Senescence and Its Implications in Aging. <i>Genes</i> , 2017, 8, 343.	2.4	42
48	A patient-derived-xenograft platform to study BRCA-deficient ovarian cancers. <i>JCI Insight</i> , 2017, 2, e89760.	5.0	55
49	Arid1a controls tissue regeneration. <i>Stem Cell Investigation</i> , 2016, 3, 35-35.	3.0	2
50	The Primary Effect on the Proteome of ARID1A-mutated Ovarian Clear Cell Carcinoma is Downregulation of the Mevalonate Pathway at the Post-transcriptional Level. <i>Molecular and Cellular Proteomics</i> , 2016, 15, 3348-3360.	3.8	23
51	BET Bromodomain Inhibition Promotes Anti-tumor Immunity by Suppressing PD-L1 Expression. <i>Cell Reports</i> , 2016, 16, 2829-2837.	6.4	331
52	HMGB2 orchestrates the chromatin landscape of senescence-associated secretory phenotype gene loci. <i>Journal of Cell Biology</i> , 2016, 215, 325-334.	5.2	132
53	BET Inhibitors Suppress ALDH Activity by Targeting <i>ALDH1A1</i> Super-Enhancer in Ovarian Cancer. <i>Cancer Research</i> , 2016, 76, 6320-6330.	0.9	115
54	Targeting RRM2 and Mutant BRAF Is a Novel Combinatorial Strategy for Melanoma. <i>Molecular Cancer Research</i> , 2016, 14, 767-775.	3.4	27

#	ARTICLE	IF	CITATIONS
55	Epigenetic synthetic lethality in ovarian clear cell carcinoma: EZH2 and <i>ARID1A</i> mutations. <i>Molecular and Cellular Oncology</i> , 2016, 3, e1032476.	0.7	21
56	SPOP E3 Ubiquitin Ligase Adaptor Promotes Cellular Senescence by Degrading the SENP7 deSUMOylase. <i>Cell Reports</i> , 2015, 13, 1183-1193.	6.4	55
57	Comprehensive analysis of the ubiquitinome during oncogene-induced senescence in human fibroblasts. <i>Cell Cycle</i> , 2015, 14, 1540-1547.	2.6	22
58	A novel role for the condensin II complex in cellular senescence. <i>Cell Cycle</i> , 2015, 14, 2160-2170.	2.6	18
59	ATM Couples Replication Stress and Metabolic Reprogramming during Cellular Senescence. <i>Cell Reports</i> , 2015, 11, 893-901.	6.4	94
60	Synthetic lethality by targeting EZH2 methyltransferase activity in <i>ARID1A</i> -mutated cancers. <i>Nature Medicine</i> , 2015, 21, 231-238.	30.7	530
61	Potential therapeutic targets in <i>ARID1A</i> -mutated cancers. <i>Expert Opinion on Therapeutic Targets</i> , 2015, 19, 1419-1422.	3.4	58
62	PI3K therapy reprograms mitochondrial trafficking to fuel tumor cell invasion. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, 8638-8643.	7.1	174
63	Filia Is an ESC-Specific Regulator of DNA Damage Response and Safeguards Genomic Stability. <i>Cell Stem Cell</i> , 2015, 16, 684-698.	11.1	46
64	Telomeric repeat-containing RNA (TERRA) constitutes a nucleoprotein component of extracellular inflammatory exosomes. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, E6293-300.	7.1	76
65	Microbially Driven TLR5-Dependent Signaling Governs Distal Malignant Progression through Tumor-Promoting Inflammation. <i>Cancer Cell</i> , 2015, 27, 27-40.	16.8	242
66	Nucleotide metabolism, oncogene-induced senescence and cancer. <i>Cancer Letters</i> , 2015, 356, 204-210.	7.2	109
67	ATM in senescence. <i>Oncotarget</i> , 2015, 6, 14729-14730.	1.8	13
68	Identification of ribonucleotide reductase M2 as a potential target for pro-senescence therapy in epithelial ovarian cancer. <i>Cell Cycle</i> , 2014, 13, 199-207.	2.6	36
69	Metabolic alterations accompanying oncogene-induced senescence. <i>Molecular and Cellular Oncology</i> , 2014, 1, e963481.	0.7	26
70	Protein isoform-specific validation defines multiple chloride intracellular channel and tropomyosin isoforms as serological biomarkers of ovarian cancer. <i>Journal of Proteomics</i> , 2013, 89, 165-178.	2.4	69
71	Suppression of Nucleotide Metabolism Underlies the Establishment and Maintenance of Oncogene-Induced Senescence. <i>Cell Reports</i> , 2013, 3, 1252-1265.	6.4	228
72	Identification of Multiple Novel Protein Biomarkers Shed by Human Serous Ovarian Tumors into the Blood of Immunocompromised Mice and Verified in Patient Sera. <i>PLoS ONE</i> , 2013, 8, e60129.	2.5	16

#	ARTICLE	IF	CITATIONS
73	Three-dimensional culture sensitizes epithelial ovarian cancer cells to EZH2 methyltransferase inhibition. <i>Cell Cycle</i> , 2013, 12, 2113-2119.	2.6	74
74	Chromatin remodeling, BRCA1, SAHF and cellular senescence. <i>Cell Cycle</i> , 2013, 12, 1653-1654.	2.6	12
75	BRG1 Is Required for Formation of Senescence-Associated Heterochromatin Foci Induced by Oncogenic RAS or BRCA1 Loss. <i>Molecular and Cellular Biology</i> , 2013, 33, 1819-1829.	2.3	46
76	Role of EZH2 in Epithelial Ovarian Cancer: From Biological Insights to Therapeutic Target. <i>Frontiers in Oncology</i> , 2013, 3, 47.	2.8	24
77	EZH2 knockdown suppresses the growth and invasion of human inflammatory breast cancer cells. <i>Journal of Experimental and Clinical Cancer Research</i> , 2013, 32, 70.	8.6	38
78	RAS, cellular senescence and transformation. <i>Small GTPases</i> , 2012, 3, 163-167.	1.6	7
79	SUZ12 Promotes Human Epithelial Ovarian Cancer by Suppressing Apoptosis via Silencing HRK. <i>Molecular Cancer Research</i> , 2012, 10, 1462-1472.	3.4	66
80	Oncogenic Ras Regulates BRIP1 Expression to Induce Dissociation of BRCA1 from Chromatin, Inhibit DNA Repair, and Promote Senescence. <i>Developmental Cell</i> , 2011, 21, 1077-1091.	7.0	82
81	Wnt5a Suppresses Epithelial Ovarian Cancer by Promoting Cellular Senescence. <i>Cancer Research</i> , 2011, 71, 6184-6194.	0.9	88
82	Activation of the PIK3CA/AKT Pathway Suppresses Senescence Induced by an Activated RAS Oncogene to Promote Tumorigenesis. <i>Molecular Cell</i> , 2011, 42, 36-49.	9.7	179
83	Enhancer of Zeste Homolog 2 Promotes the Proliferation and Invasion of Epithelial Ovarian Cancer Cells. <i>Molecular Cancer Research</i> , 2010, 8, 1610-1618.	3.4	109
84	Molecular Dissection of Formation of Senescence-Associated Heterochromatin Foci. <i>Molecular and Cellular Biology</i> , 2007, 27, 2343-2358.	2.3	361
85	Definition of pRB- and p53-Dependent and -Independent Steps in HIRA/ASF1a-Mediated Formation of Senescence-Associated Heterochromatin Foci. <i>Molecular and Cellular Biology</i> , 2007, 27, 2452-2465.	2.3	145
86	Downregulation of Wnt Signaling Is a Trigger for Formation of Facultative Heterochromatin and Onset of Cell Senescence in Primary Human Cells. <i>Molecular Cell</i> , 2007, 27, 183-196.	9.7	203
87	Formation of MacroH2A-Containing Senescence-Associated Heterochromatin Foci and Senescence Driven by ASF1a and HIRA. <i>Developmental Cell</i> , 2005, 8, 19-30.	7.0	609