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List of Publications by Year
in descending order

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64	8,212	94433	123424
papers	citations	37	61
		h-index	g-index

64	64	64	8682
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	Phase II Study of Ponatinib in Advanced Gastrointestinal Stromal Tumors: Efficacy, Safety, and Impact of Liquid Biopsy and Other Biomarkers. Clinical Cancer Research, 2022, 28, 1268-1276.	7.0	7
2	Mobocertinib (TAK-788): A Targeted Inhibitor ofEGFRExon 20 Insertion Mutants in Nonâ€“Small Cell Lung Cancer. Cancer Discovery, 2021, 11, 1672-1687.	9.4	112
3	TargetingHER2Exon 20 Insertionâ€“Mutant Lung Adenocarcinoma with a Novel Tyrosine Kinase Inhibitor Mobocertinib. Cancer Research, 2021, 81, 5311-5324.	0.9	31
4	Ultra-accurate Duplex Sequencing for the assessment of pretreatment ABL1 kinase domain mutations in Ph+ ALL. Blood Cancer Journal, 2020, 10, 61.	6.2	20
5	Ponatinib efficacy and safety in Philadelphia chromosomeâ€“positive leukemia: final 5-year results of the phase 2 PACE trial. Blood, 2018, 132, 393-404.	1.4	392
6	RET fusions observed in lung and colorectal cancers are sensitive to ponatinib. Oncotarget, 2018, 9, 29654-29664.	1.8	20
7	Single-Molecule Sequencing Reveals Patterns of Preexisting Drug Resistance That Suggest Treatment Strategies in Philadelphia-Positive Leukemias. Clinical Cancer Research, 2018, 24, 5321-5334.	7.0	24
8	Ponatinib versus imatinib for newly diagnosed chronic myeloid leukaemia: an international, randomised, open-label, phase 3 trial. Lancet Oncology, The, 2016, 17, 612-621.	10.7	214
9	Discovery of Brigatinib (AP26113), a Phosphine Oxide-Containing, Potent, Orally Active Inhibitor of Anaplastic Lymphoma Kinase. Journal of Medicinal Chemistry, 2016, 59, 4948-4964.	6.4	277
10	Compound mutations in BCR-ABL1 are not major drivers of primary or secondary resistance to ponatinib in CP-CML patients. Blood, 2016, 127, 703-712.	1.4	87
11	The impact of multiple low-level BCR-ABL1 mutations on response to ponatinib. Blood, 2016, 127, 1870-1880.	1.4	58
12	The Potent ALK Inhibitor Brigatinib (AP26113) Overcomes Mechanisms of Resistance to First- and Second-Generation ALK Inhibitors in Preclinical Models. Clinical Cancer Research, 2016, 22, 5527-5538.	7.0	263
13	Brigatinib, an anaplastic lymphoma kinase inhibitor, abrogates activity and growth in ALK-positive neuroblastoma cells,Drosophilaand mice. Oncotarget, 2016, 7, 29011-29022.	1.8	51
14	Acquisition of a single EZH2 D1 domain mutation confers acquired resistance to EZH2-targeted inhibitors. Oncotarget, 2015, 6, 32646-32655.	1.8	65
15	Abstract 2827: Discovery of AP26113, a potent, orally active inhibitor of anaplastic lymphoma kinase and clinically relevant mutants. Cancer Research, 2015, 75, 2827-2827.	0.9	5
16	Abstract 781: The potent ALK inhibitor AP26113 can overcome mechanisms of resistance to first- and second-generation ALK TKIs in preclinical models. , 2015, , .		7
17	Ponatinib Inhibits Polyclonal Drug-Resistant KIT Oncoproteins and Shows Therapeutic Potential in Heavily Pretreated Gastrointestinal Stromal Tumor (GIST) Patients. Clinical Cancer Research, 2014, 20, 5745-5755.	7.0	137
18	Long-Term Follow-up of Ponatinib Efficacy and Safety in the Phase 2 PACE Trial. Blood, 2014, 124, 3135-3135.	1.4	43

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19	Ponatinib Efficacy and Safety in Patients with the T315I Mutation: Long-Term Follow-up of Phase 1 and Phase 2 (PACE) Trials. Blood, 2014, 124, 4552-4552.	1.4	8
20	Combined targeting of FGFR2 and mTOR by ponatinib and ridaforolimus results in synergistic antitumor activity in FGFR2 mutant endometrial cancer models. Cancer Chemotherapy and Pharmacology, 2013, 71, 1315-1323.	2.3	58
21	Comprehensive Analysis Of The In Vitro Potency Of Ponatinib, and All Other Approved BCR-ABL Tyrosine Kinase Inhibitors (TKIs), Against a Panel Of Single and Compound BCR-ABL Mutants. Blood, 2013, 122, 3992-3992.	1.4	11
22	Impact Of Baseline (BL) Mutations, Including Low-Level and Compound Mutations, On Ponatinib Response and End Of Treatment (EOT) Mutation Analysis In Patients (Pts) With Chronic Phase Chronic Myeloid Leukemia (CP-CML). Blood, 2013, 122, 652-652.	1.4	6
23	Phase II Study of the Mammalian Target of Rapamycin Inhibitor Ridaforolimus in Patients With Advanced Bone and Soft Tissue Sarcomas. Journal of Clinical Oncology, 2012, 30, 78-84.	1.6	238
24	Dimerizer-Mediated Regulation of Gene Expression. Cold Spring Harbor Protocols, 2012, 2012, pdb.top070128-pdb.top070128.	0.3	6
25	Synergistic activity of the mTOR inhibitor ridaforolimus and the antiandrogen bicalutamide in prostate cancer models. International Journal of Oncology, 2012, 41, 425-432.	3.3	28
26	Dimerizer-Mediated Regulation of Gene Expression In Vivo. Cold Spring Harbor Protocols, 2012, 2012, pdb.prot070144-pdb.prot070144.	0.3	13
27	Ponatinib in Refractory Philadelphia Chromosomeâ€“Positive Leukemias. New England Journal of Medicine, 2012, 367, 2075-2088.	27.0	668
28	Dimerizer-Mediated Regulation of Gene Expression In Vitro. Cold Spring Harbor Protocols, 2012, 2012, pdb.prot070136.	0.3	5
29	Ponatinib (AP24534), a Multitargeted Pan-FGFR Inhibitor with Activity in Multiple FGFR-Amplified or Mutated Cancer Models. Molecular Cancer Therapeutics, 2012, 11, 690-699.	4.1	289
30	Ridaforolimus for patients with progressive or recurrent malignant glioma: a perisurgical, sequential, ascending-dose trial. Cancer Chemotherapy and Pharmacology, 2012, 69, 849-860.	2.3	18
31	Analysis of the pharmacodynamic activity of the mTOR inhibitor ridaforolimus (AP23573, MK-8669) in a phase 1 clinical trial. Cancer Chemotherapy and Pharmacology, 2012, 69, 1369-1377.	2.3	18
32	Abstract 853: Ponatinib, a potent pan-BCR-ABL inhibitor, retains activity against gatekeeper mutants of FLT3, RET, KIT, PDGFRÎ±/Î² and FGFR1. , 2012, , .		3
33	Multivariate Analyses of the Clinical and Molecular Parameters Associated with Efficacy and Safety in Patients with Chronic Myeloid Leukemia (CML) and Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia (Ph+ ALL) Treated with Ponatinib in the PACE Trial. Blood, 2012, 120, 3747-3747.	1.4	6
34	Therapeutic strategies to overcome crizotinib resistance in non-small cell lung cancers harboring the fusion oncogene EML4-ALK. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 7535-7540.	7.1	515
35	The BCR-ABL35INS insertion/truncation mutant is kinase-inactive and does not contribute to tyrosine kinase inhibitor resistance in chronic myeloid leukemia. Blood, 2011, 118, 5250-5254.	1.4	37
36	Structural Mechanism of the Panâ€BCRâ€ABL Inhibitor Ponatinib (AP24534): Lessons for Overcoming Kinase Inhibitor Resistance. Chemical Biology and Drug Design, 2011, 77, 1-11.	3.2	231

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37	CrizotinibâEResistant Mutants of EML4âEALK Identified Through an Accelerated Mutagenesis Screen. Chemical Biology and Drug Design, 2011, 78, 999-1005.	3.2	127
38	Potent Activity of Ponatinib (AP24534) in Models of FLT3-Driven Acute Myeloid Leukemia and Other Hematologic Malignancies. Molecular Cancer Therapeutics, 2011, 10, 1028-1035.	4.1	135
39	Discovery of 5-(arenethynyl) hetero-monocyclic derivatives as potent inhibitors of BCRâE"ABL including the T315I gatekeeper mutant. Bioorganic and Medicinal Chemistry Letters, 2011, 21, 3743-3748.	2.2	17
40	Ridaforolimus (AP23573; MK-8669), a Potent mTOR Inhibitor, Has Broad Antitumor Activity and Can Be Optimally Administered Using Intermittent Dosing Regimens. Molecular Cancer Therapeutics, 2011, 10, 1059-1071.	4.1	92
41	Assessment of the Safety and Biodistribution of a Regulated AAV2 Gene Transfer Vector after Delivery to Murine Submandibular Glands. Toxicological Sciences, 2011, 123, 247-255.	3.1	4
42	Antitumor Activity of Ridaforolimus and Potential Cell-Cycle Determinants of Sensitivity in Sarcoma and Endometrial Cancer Models. Molecular Cancer Therapeutics, 2011, 10, 1959-1968.	4.1	46
43	Phase IB Study of the mTOR Inhibitor Ridaforolimus With Capecitabine. Journal of Clinical Oncology, 2010, 28, 4554-4561.	1.6	47
44	Discovery of 3-[2-(Imidazo[1,2-b]pyridazin-3-yl)ethynyl]-4-methyl-N-{4-[(4-methylpiperazin-1-yl)methyl]-3-(trifluoromethyl)phenyl}benz (AP24534), a Potent, Orally Active Pan-Inhibitor of Breakpoint Cluster Region-Abelson (BCR-ABL) Kinase Including the T315I Gatekeeper Mutant. Journal of Medicinal Chemistry, 2010, 53, 4701-4719.	6.4	315
45	A Phase 1 Trial of Oral Ponatinib (AP24534) In Patients with Refractory Chronic Myelogenous Leukemia (CML) and Other Hematologic Malignancies: Emerging Safety and Clinical Response Findings. Blood, 2010, 116, 210-210.	1.4	53
46	A Phase I Trial to Determine the Safety, Tolerability, and Maximum Tolerated Dose of Deforolimus in Patients with Advanced Malignancies. Clinical Cancer Research, 2009, 15, 1428-1434.	7.0	89
47	AP24534, a Pan-BCR-ABL Inhibitor for Chronic Myeloid Leukemia, Potently Inhibits the T315I Mutant and Overcomes Mutation-Based Resistance. Cancer Cell, 2009, 16, 401-412.	16.8	1,050
48	A Phase 2 Clinical Trial of Deforolimus (AP23573, MK-8669), a Novel Mammalian Target of Rapamycin Inhibitor, in Patients with Relapsed or Refractory Hematologic Malignancies. Clinical Cancer Research, 2008, 14, 2756-2762.	7.0	233
49	Phase I Trial of the Novel Mammalian Target of Rapamycin Inhibitor Deforolimus (AP23573; MK-8669) Administered Intravenously Daily for 5 Days Every 2 Weeks to Patients With Advanced Malignancies. Journal of Clinical Oncology, 2008, 26, 361-367.	1.6	273
50	Rapamycin-regulated Control of Antiangiogenic Tumor Therapy Following rAAV-mediated Gene Transfer. Molecular Therapy, 2007, 15, 912-920.	8.2	21
51	Dimerizer regulation of AADC expression and behavioral response in AAV-transduced 6-OHDA lesioned rats. Molecular Therapy, 2006, 13, 167-174.	8.2	24
52	Long-term pharmacologically regulated expression of erythropoietin in primates following AAV-mediated gene transfer. Blood, 2005, 105, 1424-1430.	1.4	258
53	Long-Term Inducible Gene Expression in the Eye via Adeno-Associated Virus Gene Transfer in Nonhuman Primates. Human Gene Therapy, 2005, 16, 178-186.	2.7	115
54	Inhibition of wild-type and mutant Bcr-Abl by AP23464, a potent ATP-based oncogenic protein kinase inhibitor: implications for CML. Blood, 2004, 104, 2532-2539.	1.4	181

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55	Regulation of gene expression by synthetic dimerizers with novel specificity. Bioorganic and Medicinal Chemistry Letters, 2003, 13, 3181-3184.	2.2	13
56	Regulated expression of erythropoietin from an AAV vector safely improves the anemia of Î²-thalassemia in a mouse model. Molecular Therapy, 2003, 7, 493-497.	8.2	50
57	A System for Small-Molecule Control of Conditionally Replication-Competent Adenoviral Vectors. Molecular Therapy, 2002, 5, 195-203.	8.2	49
58	Pharmacological Regulation of Protein Expression from Adeno-Associated Viral Vectors in the Eye. Molecular Therapy, 2002, 6, 238-242.	8.2	94
59	Regulated Delivery of Therapeutic Proteins After in Vivo Somatic Cell Gene Transfer. Science, 1999, 283, 88-91.	12.6	313
60	[15] Regulation of gene expression with synthetic dimerizers. Methods in Enzymology, 1999, 306, 263-281.	1.0	12
61	Synthesis and activity of bivalent FKBP12 ligands for the regulated dimerization of proteins. Bioorganic and Medicinal Chemistry, 1998, 6, 1309-1335.	3.0	64
62	Controlling Gene Expression Using Synthetic Ligands. Methods, 1998, 14, 421-429.	3.8	28
63	A humanized system for pharmacologic control of gene expression. Nature Medicine, 1996, 2, 1028-1032.	30.7	538
64	Rapamycin-regulated Control of Antiangiogenic Tumor Therapy Following rAAV-mediated Gene Transfer. Molecular Therapy, 0, , .	8.2	0