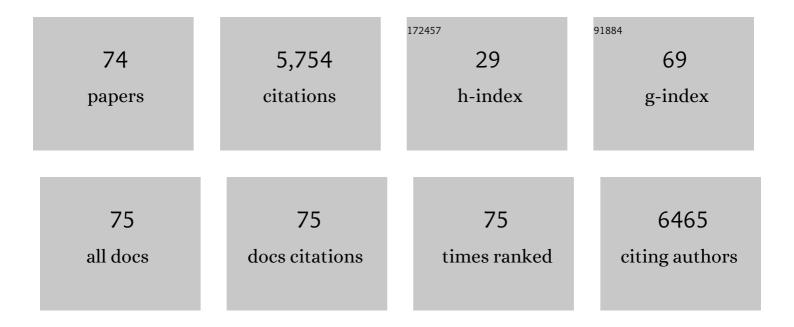
List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Small molecule inhibition of deubiquitinating enzyme JOSD1 as a novel targeted therapy for leukemias with mutant JAK2. Leukemia, 2022, 36, 210-220.	7.2	12
2	Inhibition of theÂdeubiquitinating enzyme USP47 as a novel targeted therapy for hematologic malignancies expressing mutant EZH2. Leukemia, 2022, 36, 1048-1057.	7.2	5
3	BRD9 degraders as chemosensitizers in acute leukemia and multiple myeloma. Blood Cancer Journal, 2022, 12, .	6.2	11
4	Selectively targeting FLT3-ITD mutants over FLT3-wt by a novel inhibitor for acute myeloid leukemia. Haematologica, 2021, 106, 605-609.	3.5	3
5	3D tissue engineered plasma cultures support leukemic proliferation and induces drug resistance. Leukemia and Lymphoma, 2021, 62, 1-9.	1.3	5
6	Essential role of the histone lysine demethylase KDM4A in the biology of malignant pleural mesothelioma (MPM). British Journal of Cancer, 2021, 125, 582-592.	6.4	4
7	Targeting chaperon protein HSP70 as a novel therapeutic strategy for FLT3-ITD-positive acute myeloid leukemia. Signal Transduction and Targeted Therapy, 2021, 6, 334.	17.1	6
8	Inhibitors of the Transcription Factor STAT3 Decrease Growth and Induce Immune Response Genes in Models of Malignant Pleural Mesothelioma (MPM). Cancers, 2021, 13, 7.	3.7	13
9	HSP70 and FLT3-ITD: Targeting chaperone system to overcome drug resistance. Blood Science, 2021, 3, 151-153.	0.9	1
10	Evaluation of ERK as a therapeutic target in acute myelogenous leukemia. Leukemia, 2020, 34, 625-629.	7.2	9
11	Repurposing of Kinase Inhibitors for Treatment of COVID-19. Pharmaceutical Research, 2020, 37, 167.	3.5	102
12	Selective USP7 inhibition elicits cancer cell killing through a p53-dependent mechanism. Scientific Reports, 2020, 10, 5324.	3.3	69
13	Current therapies under investigation for COVID-19: potential COVID-19 treatments. Canadian Journal of Physiology and Pharmacology, 2020, 98, 483-489.	1.4	6
14	Effects of the multiâ€kinase inhibitor midostaurin in combination with chemotherapy in models of acute myeloid leukaemia. Journal of Cellular and Molecular Medicine, 2020, 24, 2968-2980.	3.6	16
15	The combination of FLT3 and SYK kinase inhibitors is toxic to leukaemia cells with CBL mutations. Journal of Cellular and Molecular Medicine, 2020, 24, 2145-2156.	3.6	2
16	Inhibition of the deubiquitinase USP10 induces degradation of SYK. British Journal of Cancer, 2020, 122, 1175-1184.	6.4	19
17	Comparison of effects of midostaurin, crenolanib, quizartinib, gilteritinib, sorafenib and BLUâ€285 on oncogenic mutants of KIT, CBL and FLT3 in haematological malignancies. British Journal of Haematology, 2019, 187, 488-501.	2.5	30
18	A nonâ€covalent inhibitor XMUâ€MPâ€3 overrides ibrutinibâ€resistant <scp><i>Btk</i>^{<i>C481S</i>}</scp> mutation in Bâ€cell malignancies. British Journal of Pharmacology, 2019, 176, 4491-4509.	5.4	17

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19	Discovery of <i>N</i> -(4-(6-Acetamidopyrimidin-4-yloxy)phenyl)-2-(2-(trifluoromethyl)phenyl)acetamide (CHMFL-FLT3-335) as a Potent FMS-like Tyrosine Kinase 3 Internal Tandem Duplication (FLT3-ITD) Mutant Selective Inhibitor for Acute Myeloid Leukemia. Journal of Medicinal Chemistry, 2019, 62, 875-892.	6.4	20
20	Spotlight on midostaurin in the treatment of FLT3-mutated acute myeloid leukemia and systemic mastocytosis: design, development, and potential place in therapy. OncoTargets and Therapy, 2018, Volume 11, 175-182.	2.0	15
21	Midostaurin, a Natural Product-Derived Kinase Inhibitor Recently Approved for the Treatment of Hematological Malignancies. Biochemistry, 2018, 57, 477-478.	2.5	15
22	A Chemoproteomic Approach to Query the Degradable Kinome Using a Multi-kinase Degrader. Cell Chemical Biology, 2018, 25, 88-99.e6.	5.2	313
23	Structure-activity relationship investigation for benzonaphthyridinone derivatives as novel potent Bruton's tyrosine kinase (BTK) irreversible inhibitors. European Journal of Medicinal Chemistry, 2017, 137, 545-557.	5.5	16
24	Structure-guided development of covalent TAK1 inhibitors. Bioorganic and Medicinal Chemistry, 2017, 25, 838-846.	3.0	28
25	Studies of TAK1-centered polypharmacology with novel covalent TAK1 inhibitors. Bioorganic and Medicinal Chemistry, 2017, 25, 1320-1328.	3.0	17
26	Inhibition of USP10 induces degradation of oncogenic FLT3. Nature Chemical Biology, 2017, 13, 1207-1215.	8.0	89
27	Structure-Guided Development of a Potent and Selective Non-covalent Active-Site Inhibitor of USP7. Cell Chemical Biology, 2017, 24, 1490-1500.e11.	5.2	149
28	Acute myeloid leukemia cells require 6-phosphogluconate dehydrogenase for cell growth and NADPH-dependent metabolic reprogramming. Oncotarget, 2017, 8, 67639-67650.	1.8	26
29	Characterization of midostaurin as a dual inhibitor of FLT3 and SYK and potentiation of FLT3 inhibition against FLT3-ITD-driven leukemia harboring activated SYK kinase. Oncotarget, 2017, 8, 52026-52044.	1.8	19
30	Inhibition of SDF-1-induced migration of oncogene-driven myeloid leukemia by the L-RNA aptamer (Spiegelmer), NOX-A12, and potentiation of tyrosine kinase inhibition. Oncotarget, 2017, 8, 109973-109984.	1.8	19
31	Dual inhibition of AKT/FLT3-ITD by A674563 overcomes FLT3 ligand-induced drug resistance in FLT3-ITD positive AML. Oncotarget, 2016, 7, 29131-29142.	1.8	21
32	Discovery of a Highly Potent and Selective Indenoindolone Type 1 Pan-FLT3 Inhibitor. ACS Medicinal Chemistry Letters, 2016, 7, 476-481.	2.8	17
33	Simultaneous inhibition of Vps34 kinase would enhance PI3Kδ inhibitor cytotoxicity in the B-cell malignancies. Oncotarget, 2016, 7, 53515-53525.	1.8	19
34	lbrutinib targets mutant-EGFR kinase with a distinct binding conformation. Oncotarget, 2016, 7, 69760-69769.	1.8	41
35	Characterization of selective and potent PI3Kδ inhibitor (PI3KD-IN-015) for B-Cell malignances. Oncotarget, 2016, 7, 32641-32651.	1.8	7
36	Inhibition of USP10 Induces Degradation of Oncogenic FLT3: A Novel Approach to Therapy of Leukemia. Blood, 2016, 128, 524-524.	1.4	0

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37	Identification of novel therapeutic targets in acute leukemias with NRAS mutations using a pharmacologic approach. Blood, 2015, 125, 3133-3143.	1.4	23
38	Inhibition of Wild-Type p53-Expressing AML by the Novel Small Molecule HDM2 Inhibitor CGM097. Molecular Cancer Therapeutics, 2015, 14, 2249-2259.	4.1	53
39	Identification of ILK as a novel therapeutic target for acute and chronic myeloid leukemia. Leukemia Research, 2015, 39, 1299-1308.	0.8	15
40	Identification of Wee1 as a novel therapeutic target for mutant RAS-driven acute leukemia and other malignancies. Leukemia, 2015, 29, 27-37.	7.2	51
41	Ibrutinib selectively and irreversibly targets EGFR (L858R, Del19) mutant but is moderately resistant to EGFR (T790M) mutant NSCLC Cells. Oncotarget, 2015, 6, 31313-31322.	1.8	38
42	Integrin-Linked Kinase a Novel Therapeutic Target for Acute and Chronic Myeloid Leukemia. Blood, 2015, 126, 3694-3694.	1.4	0
43	Combination therapy with nilotinib for drug-sensitive and drug-resistant BCR-ABL-positive leukemia and other malignancies. Archives of Toxicology, 2014, 88, 2233-2242.	4.2	6
44	Upregulation of IGF1R by Mutant <i>RAS</i> in Leukemia and Potentiation of <i>RAS</i> Signaling Inhibitors by Small-Molecule Inhibition of IGF1R. Clinical Cancer Research, 2014, 20, 5483-5495.	7.0	16
45	Selective Akt Inhibitors Synergize with Tyrosine Kinase Inhibitors and Effectively Override Stroma-Associated Cytoprotection of Mutant FLT3-Positive AML Cells. PLoS ONE, 2013, 8, e56473.	2.5	38
46	Small Molecule Activators Of AMPK Block The Glycogen Production Required For Transformation Of Myeloid Leukemia Cells. Blood, 2013, 122, 1479-1479.	1.4	2
47	An amino-indazole scaffold with spectrum selective kinase inhibition of FLT3, PDGFRα and kit. Bioorganic and Medicinal Chemistry Letters, 2012, 22, 4579-4584.	2.2	12
48	Development of â€~DFG-out' inhibitors of gatekeeper mutant kinases. Bioorganic and Medicinal Chemistry Letters, 2012, 22, 5297-5302.	2.2	10
49	BCR-ABL Transformation Requires Glycogen Synthase 1 (GYS1) Expression for Cell Growth and Increased Glycogen Production. Blood, 2012, 120, 1673-1673.	1.4	0
50	Reversible Resistance Induced by FLT3 Inhibition: A Novel Resistance Mechanism in Mutant FLT3-Expressing Cells. PLoS ONE, 2011, 6, e25351.	2.5	42
51	Potentiation of the Effects of Nilotinib by Combination with Plerixafor in a Mouse Model of BCR-ABL-Positive Residual Disease. Blood, 2011, 118, 2737-2737.	1.4	0
52	Discovery of a small-molecule type II inhibitor of wild-type and gatekeeper mutants of BCR-ABL, PDGFRα, Kit, and Src kinases: novel type II inhibitor of gatekeeper mutants. Blood, 2010, 115, 4206-4216.	1.4	61
53	Discovery and Characterization of Novel Mutant FLT3 Kinase Inhibitors. Molecular Cancer Therapeutics, 2010, 9, 2468-2477.	4.1	15
54	Antileukemic Effects of Novel First- and Second-Generation FLT3 Inhibitors: Structure-Affinity Comparison. Genes and Cancer, 2010, 1, 1021-1032.	1.9	33

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55	FLT3 inhibition and mechanisms of drug resistance in mutant FLT3-positive AML. Drug Resistance Updates, 2009, 12, 81-89.	14.4	95
56	Microenvironment-Dependent Synthetic Lethality: Implications for Tumor Pathophysiology and Anti-Cancer Drug Discovery Blood, 2009, 114, 1722-1722.	1.4	0
57	Stromal-mediated protection of tyrosine kinase inhibitor-treated BCR-ABL-expressing leukemia cells. Molecular Cancer Therapeutics, 2008, 7, 1121-1129.	4.1	65
58	Potentiation of antileukemic therapies by the dual PI3K/PDK-1 inhibitor, BAG956: effects on BCR-ABL– and mutant FLT3-expressing cells. Blood, 2008, 111, 3723-3734.	1.4	81
59	Antileukemic effects of the novel, mutant FLT3 inhibitor NVP-AST487: effects on PKC412-sensitive and -resistant FLT3-expressing cells. Blood, 2008, 112, 5161-5170.	1.4	29
60	Potentiation of antileukemic therapies by Smac mimetic, LBW242: effects on mutant FLT3-expressing cells. Molecular Cancer Therapeutics, 2007, 6, 1951-1961.	4.1	78
61	Beneficial effects of combining nilotinib and imatinib in preclinical models of BCR-ABL+ leukemias. Blood, 2007, 109, 2112-2120.	1.4	98
62	Effects of PKC412, Nilotinib, and Imatinib Against GIST-Associated PDGFRA Mutants With Differential Imatinib Sensitivity. Gastroenterology, 2006, 131, 1734-1742.	1.3	93
63	Characterization of AMN107, a selective inhibitor of native and mutant Bcr-Abl. Cancer Cell, 2005, 7, 129-141.	16.8	1,387
64	Simultaneous Administration of AMN107 and Imatinib in the Treatment of Imatinib-Sensitive and Imatinib-Resistant Chronic Myeloid Leukemia Blood, 2005, 106, 694-694.	1.4	7
65	Novel Hydroxamic Acid-Derived HDAC Inhibitor LBH589 Potently Activates Intrinsic and Extrinsic Apoptotic Pathways, and Induces Tubulin Hyperacetylation in Multiple Myeloma Blood, 2005, 106, 1578-1578.	1.4	1
66	Identifying and characterizing a novel activating mutation of the FLT3 tyrosine kinase in AML. Blood, 2004, 104, 1855-1858.	1.4	80
67	AMD107: Efficacy as a Selective Inhibitor of the Tyrosine Kinase Activity of BCR-ABL in Murine Leukemia Models Blood, 2004, 104, 551-551.	1.4	2
68	PKC412 overcomes resistance to imatinib in a murine model of FIP1L1-PDGFRα-induced myeloproliferative disease. Cancer Cell, 2003, 3, 459-469.	16.8	223
69	Resistance to imatinib (Glivec): update on clinical mechanisms. Drug Resistance Updates, 2003, 6, 231-238.	14.4	84
70	Inhibition of mutant FLT3 receptors in leukemia cells by the small molecule tyrosine kinase inhibitor PKC412. Cancer Cell, 2002, 1, 433-443.	16.8	574
71	ARG tyrosine kinase activity is inhibited by STI571. Blood, 2001, 97, 2440-2448.	1.4	246
72	Mechanism of resistance to the ABL tyrosine kinase inhibitor STI571 in BCR/ABL–transformed hematopoietic cell lines. Blood, 2000, 95, 3498-3505.	1.4	374

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73	Smad4 and FAST-1 in the assembly of activin-responsive factor. Nature, 1997, 389, 85-89.	27.8	534
74	Tensin: A potential link between the cytoskeleton and signal transduction. BioEssays, 1994, 16, 817-823.	2.5	125