

Brian J Druker

List of Publications by Year in descending order

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108
papers

28,572
citations

27035

58
h-index

31191

106
g-index

108
all docs

108
docs citations

108
times ranked

22062
citing authors

#	ARTICLE	IF	CITATIONS
1	Identification and prioritization of myeloid malignancy germline variants in a large cohort of adult patients with AML. <i>Blood</i> , 2022, 139, 1208-1221.	0.6	48
2	MS4A3 promotes differentiation in chronic myeloid leukemia by enhancing common \hat{I}^2 -chain cytokine receptor endocytosis. <i>Blood</i> , 2022, 139, 761-778.	0.6	7
3	Associating drug sensitivity with differentiation status identifies effective combinations for acute myeloid leukemia. <i>Blood Advances</i> , 2022, 6, 3062-3067.	2.5	6
4	Understanding Drug Sensitivity and Tackling Resistance in Cancer. <i>Cancer Research</i> , 2022, 82, 1448-1460.	0.4	24
5	Mutated <i>SETBP1</i> activates transcription of <i>Myc</i> programs to accelerate <i>CSF3R</i> -driven myeloproliferative neoplasms. <i>Blood</i> , 2022, 140, 644-658.	0.6	12
6	Luxetpinib (CG-806) Targets FLT3 and Clusters of Kinases Operative in Acute Myeloid Leukemia. <i>Molecular Cancer Therapeutics</i> , 2022, 21, 1125-1135.	1.9	4
7	PU.1 and MYC transcriptional network defines synergistic drug responses to KIT and LSD1 inhibition in acute myeloid leukemia. <i>Leukemia</i> , 2022, , .	3.3	7
8	BCR-ABL+ Chronic Myeloid Leukemia Arising in a Family With Inherited ANKRD26-Related Thrombocytopenia. <i>JCO Precision Oncology</i> , 2021, 5, 415-417.	1.5	0
9	Proteasome 26S subunit, non-ATPases 1 (PSMD1) and 3 (PSMD3), play an oncogenic role in chronic myeloid leukemia by stabilizing nuclear factor-kappa B. <i>Oncogene</i> , 2021, 40, 2697-2710.	2.6	20
10	Bayesian multi-source regression and monocyte-associated gene expression predict BCL-2 inhibitor resistance in acute myeloid leukemia. <i>Npj Precision Oncology</i> , 2021, 5, 71.	2.3	12
11	Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. <i>Cancer Research</i> , 2021, 81, 4685-4695.	0.4	6
12	ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. <i>Leukemia</i> , 2020, 34, 2798-2804.	3.3	16
13	Precision medicine treatment in acute myeloid leukemia using prospective genomic profiling: feasibility and preliminary efficacy of the Beat AML Master Trial. <i>Nature Medicine</i> , 2020, 26, 1852-1858.	15.2	104
14	Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. <i>Leukemia</i> , 2020, 34, 2342-2353.	3.3	18
15	NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2V617F-positive myeloproliferative neoplasm cells. <i>Signal Transduction and Targeted Therapy</i> , 2020, 5, 5.	7.1	26
16	Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. <i>Blood</i> , 2019, 134, 867-879.	0.6	55
17	Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. <i>Cancer Research</i> , 2019, 79, 4744-4753.	0.4	139
18	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. <i>Leukemia</i> , 2019, 33, 1835-1850.	3.3	97

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19	The TP53 Apoptotic Network Is a Primary Mediator of Resistance to BCL2 Inhibition in AML Cells. <i>Cancer Discovery</i> , 2019, 9, 910-925.	7.7	215
20	Challenges and approaches to implementing master/basket trials in oncology. <i>Blood Advances</i> , 2019, 3, 2237-2243.	2.5	11
21	Myeloid lineage enhancers drive oncogene synergy in CEBPA/CSF3R mutant acute myeloid leukemia. <i>Nature Communications</i> , 2019, 10, 5455.	5.8	22
22	Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. <i>Nature Communications</i> , 2019, 10, 244.	5.8	111
23	A novel <i>AGGF1-PDGFRb</i> fusion in pediatric T-cell acute lymphoblastic leukemia. <i>Haematologica</i> , 2018, 103, e87-e91.	1.7	8
24	Functional genomic landscape of acute myeloid leukaemia. <i>Nature</i> , 2018, 562, 526-531.	13.7	907
25	Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. <i>New England Journal of Medicine</i> , 2017, 376, 917-927.	13.9	926
26	Peter C. Nowell (1928–2016). <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, 4569-4570.	3.3	0
27	Molecularly targeted drug combinations demonstrate selective effectiveness for myeloid- and lymphoid-derived hematologic malignancies. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, E7554-E7563.	3.3	86
28	Age-related mutations and chronic myelomonocytic leukemia. <i>Leukemia</i> , 2016, 30, 906-913.	3.3	119
29	Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. <i>Leukemia</i> , 2016, 30, 1418-1421.	3.3	9
30	YM155 potently kills acute lymphoblastic leukemia cells through activation of the DNA damage pathway. <i>Journal of Hematology and Oncology</i> , 2015, 8, 39.	6.9	32
31	Structural insight into selectivity and resistance profiles of ROS1 tyrosine kinase inhibitors. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, E5381-90.	3.3	93
32	Imatinib 800Âmg daily induces deeper molecular responses than imatinib 400Âmg daily: results of <i>SWOG S0325</i> , an intergroup randomized <i>PHASE II</i> trial in newly diagnosed chronic phase chronic myeloid leukaemia. <i>British Journal of Haematology</i> , 2014, 164, 223-232.	1.2	56
33	BCR-ABL1 Compound Mutations Combining Key Kinase Domain Positions Confer Clinical Resistance to Ponatinib in Ph Chromosome-Positive Leukemia. <i>Cancer Cell</i> , 2014, 26, 428-442.	7.7	292
34	BCR-ABL1 compound mutations in tyrosine kinase inhibitor-resistant CML: frequency and clonal relationships. <i>Blood</i> , 2013, 121, 489-498.	0.6	187
35	Threshold Levels of ABL Tyrosine Kinase Inhibitors Retained in Chronic Myeloid Leukemia Cells Determine Their Commitment to Apoptosis. <i>Cancer Research</i> , 2013, 73, 3356-3370.	0.4	26
36	Oncogenic <i>CSF3R</i> Mutations in Chronic Neutrophilic Leukemia and Atypical CML. <i>New England Journal of Medicine</i> , 2013, 368, 1781-1790.	13.9	499

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37	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. <i>Cancer Research</i> , 2013, 73, 5775-5786.	0.4	22
38	Kinase Pathway Dependence in Primary Human Leukemias Determined by Rapid Inhibitor Screening. <i>Cancer Research</i> , 2013, 73, 285-296.	0.4	134
39	Blockade of JAK2-mediated extrinsic survival signals restores sensitivity of CML cells to ABL inhibitors. <i>Leukemia</i> , 2012, 26, 1140-1143.	3.3	97
40	Ponatinib in Refractory Philadelphia Chromosome-Positive Leukemias. <i>New England Journal of Medicine</i> , 2012, 367, 2075-2088.	13.9	668
41	TNF \pm facilitates clonal expansion of JAK2V617F positive cells in myeloproliferative neoplasms. <i>Blood</i> , 2011, 118, 6392-6398.	0.6	227
42	Human chronic myeloid leukemia stem cells are insensitive to imatinib despite inhibition of BCR-ABL activity. <i>Journal of Clinical Investigation</i> , 2011, 121, 396-409.	3.9	661
43	The ABL Switch Control Inhibitor DCC-2036 Is Active against the Chronic Myeloid Leukemia Mutant BCR-ABL T315I and Exhibits a Narrow Resistance Profile. <i>Cancer Research</i> , 2011, 71, 3189-3195.	0.4	91
44	Targeting the BCR-ABL Signaling Pathway in Therapy-Resistant Philadelphia Chromosome-Positive Leukemia. <i>Clinical Cancer Research</i> , 2011, 17, 212-221.	3.2	127
45	A gene expression signature of CD34+ cells to predict major cytogenetic response in chronic-phase chronic myeloid leukemia patients treated with imatinib. <i>Blood</i> , 2010, 115, 315-325.	0.6	108
46	CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms. <i>Blood</i> , 2010, 115, 5232-5240.	0.6	216
47	The function of the pleckstrin homology domain in BCR-ABL-mediated leukemogenesis. <i>Leukemia</i> , 2010, 24, 226-229.	3.3	12
48	Clonal chromosomal abnormalities in CD34+/CD38 ^{low} hematopoietic cells from cytogenetically normal chronic myeloid leukemia patients with a complete cytogenetic response to tyrosine kinase inhibitors. <i>Leukemia</i> , 2010, 24, 1525-1528.	3.3	6
49	MET Receptor Sequence Variants R970C and T992I Lack Transforming Capacity. <i>Cancer Research</i> , 2010, 70, 6233-6237.	0.4	65
50	A Specific Need for CRKL in p210BCR-ABL-Induced Transformation of Mouse Hematopoietic Progenitors. <i>Cancer Research</i> , 2010, 70, 7325-7335.	0.4	33
51	A BCR-ABL Mutant Lacking Direct Binding Sites for the GRB2, CBL and CRKL Adapter Proteins Fails to Induce Leukemia in Mice. <i>PLoS ONE</i> , 2009, 4, e7439.	1.1	24
52	RNAi screen for rapid therapeutic target identification in leukemia patients. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2009, 106, 8695-8700.	3.3	110
53	AP24534, a Pan-BCR-ABL Inhibitor for Chronic Myeloid Leukemia, Potently Inhibits the T315I Mutant and Overcomes Mutation-Based Resistance. <i>Cancer Cell</i> , 2009, 16, 401-412.	7.7	1,050
54	High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. <i>Leukemia</i> , 2009, 23, 406-409.	3.3	15

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55	Functional characterization of an activating TEK mutation in acute myeloid leukemia: a cellular context-dependent activating mutation. <i>Leukemia</i> , 2009, 23, 1345-1348.	3.3	5
56	Perspectives on the development of imatinib and the future of cancer research. <i>Nature Medicine</i> , 2009, 15, 1149-1152.	15.2	101
57	High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. <i>Blood</i> , 2009, 113, 1749-1755.	0.6	119
58	An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. <i>Leukemia</i> , 2008, 22, 2269-2272.	3.3	32
59	Bruton's tyrosine kinase is not essential for Bcr-Abl-mediated transformation of lymphoid or myeloid cells. <i>Leukemia</i> , 2008, 22, 1354-1360.	3.3	8
60	Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. <i>Leukemia</i> , 2008, 22, 1184-1190.	3.3	38
61	An Intron-Derived Insertion/Truncation Mutation in the BCR-ABL Kinase Domain in Chronic Myeloid Leukemia Patients Undergoing Kinase Inhibitor Therapy. <i>Journal of Molecular Diagnostics</i> , 2008, 10, 177-180.	1.2	55
62	Translation of the Philadelphia chromosome into therapy for CML. <i>Blood</i> , 2008, 112, 4808-4817.	0.6	587
63	RNAi screening of the tyrosine kinome identifies therapeutic targets in acute myeloid leukemia. <i>Blood</i> , 2008, 111, 2238-2245.	0.6	67
64	High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. <i>Blood</i> , 2008, 111, 4788-4796.	0.6	84
65	A Half-Log Increase in BCR-ABL RNA Predicts a Higher Risk of Relapse in Patients with Chronic Myeloid Leukemia with an Imatinib-Induced Complete Cytogenetic Response. <i>Clinical Cancer Research</i> , 2007, 13, 6136-6143.	3.2	115
66	Mutations of the BCR-ABL-kinase domain occur in a minority of patients with stable complete cytogenetic response to imatinib. <i>Leukemia</i> , 2007, 21, 489-493.	3.3	74
67	Identification of Driver and Passenger Mutations of FLT3 by High-Throughput DNA Sequence Analysis and Functional Assessment of Candidate Alleles. <i>Cancer Cell</i> , 2007, 12, 501-513.	7.7	174
68	BCR-ABL mRNA levels at and after the time of a complete cytogenetic response (CCR) predict the duration of CCR in imatinib mesylate-treated patients with CML. <i>Blood</i> , 2006, 107, 4250-4256.	0.6	120
69	Targeted CML therapy: controlling drug resistance, seeking cure. <i>Current Opinion in Genetics and Development</i> , 2006, 16, 92-99.	1.5	166
70	Phosphoproteomic analysis of AML cell lines identifies leukemic oncogenes. <i>Leukemia Research</i> , 2006, 30, 1097-1104.	0.4	53
71	Establishment of a murine model of aggressive systemic mastocytosis/mast cell leukemia. <i>Experimental Hematology</i> , 2006, 34, 284-288.	0.2	22
72	Activating alleles of JAK3 in acute megakaryoblastic leukemia. <i>Cancer Cell</i> , 2006, 10, 65-75.	7.7	295

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73	Antileukemic Activity of Lysophosphatidic Acid Acyltransferase-1 ² Inhibitor CT32228 in Chronic Myelogenous Leukemia Sensitive and Resistant to Imatinib. <i>Clinical Cancer Research</i> , 2006, 12, 6540-6546.	3.2	22
74	Dasatinib (BMS-354825), a Dual SRC/ABL Kinase Inhibitor, Inhibits the Kinase Activity of Wild-Type, Juxtamembrane, and Activation Loop Mutant KIT Isoforms Associated with Human Malignancies. <i>Cancer Research</i> , 2006, 66, 473-481.	0.4	437
75	Characterization of Murine JAK2V617F-Positive Myeloproliferative Disease. <i>Cancer Research</i> , 2006, 66, 11156-11165.	0.4	184
76	Kinase Domain Mutants of Bcr-Abl Exhibit Altered Transformation Potency, Kinase Activity, and Substrate Utilization, Irrespective of Sensitivity to Imatinib. <i>Molecular and Cellular Biology</i> , 2006, 26, 6082-6093.	1.1	192
77	Five-Year Follow-up of Patients Receiving Imatinib for Chronic Myeloid Leukemia. <i>New England Journal of Medicine</i> , 2006, 355, 2408-2417.	13.9	3,212
78	RNAi-induced down-regulation of FLT3 expression in AML cell lines increases sensitivity to MLN518. <i>Blood</i> , 2005, 105, 2952-2954.	0.6	39
79	In vitro Activity of Bcr-Abl Inhibitors AMN107 and BMS-354825 against Clinically Relevant Imatinib-Resistant Abl Kinase Domain Mutants. <i>Cancer Research</i> , 2005, 65, 4500-4505.	0.4	997
80	e8a2 BCR-ABL: more frequent than other atypical BCR-ABL variants?. <i>Leukemia</i> , 2005, 19, 681-684.	3.3	35
81	A single nucleotide polymorphism in the coding region of ABL and its effects on sensitivity to imatinib. <i>Leukemia</i> , 2005, 19, 1859-1862.	3.3	18
82	Zoledronate inhibits proliferation and induces apoptosis of imatinib-resistant chronic myeloid leukaemia cells. <i>Leukemia</i> , 2005, 19, 1896-1904.	3.3	52
83	AMN107: Tightening the grip of imatinib. <i>Cancer Cell</i> , 2005, 7, 117-119.	7.7	93
84	Low-Level Expression of Proapoptotic Bcl-2-Interacting Mediator in Leukemic Cells in Patients with Chronic Myeloid Leukemia: Role of BCR/ABL, Characterization of Underlying Signaling Pathways, and Reexpression by Novel Pharmacologic Compounds. <i>Cancer Research</i> , 2005, 65, 9436-9444.	0.4	80
85	Combined Abl Inhibitor Therapy for Minimizing Drug Resistance in Chronic Myeloid Leukemia: Src/Abl Inhibitors Are Compatible with Imatinib. <i>Clinical Cancer Research</i> , 2005, 11, 6987-6993.	3.2	96
86	Identification of mTOR as a novel bifunctional target in chronic myeloid leukemia: dissection of growth-inhibitory and VEGF-suppressive effects of rapamycin in leukemic cells. <i>FASEB Journal</i> , 2005, 19, 960-962.	0.2	56
87	The development of imatinib as a therapeutic agent for chronic myeloid leukemia. <i>Blood</i> , 2005, 105, 2640-2653.	0.6	1,137
88	Molecularly Targeted Therapy: Have the Floodgates Opened?. <i>Oncologist</i> , 2004, 9, 357-360.	1.9	48
89	Detection of ABL kinase domain mutations with denaturing high-performance liquid chromatography. <i>Leukemia</i> , 2004, 18, 864-871.	3.3	62
90	Coexistence of phosphotyrosine-dependent and -independent interactions between Cbl and Bcr-Abl. <i>Experimental Hematology</i> , 2004, 32, 113-121.	0.2	8

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91	SR Circumventing imatinib resistance. <i>Cancer Cell</i> , 2004, 6, 108-110.	7.7	33
92	Catalytic domains of tyrosine kinases determine the phosphorylation sites within c-Cbl. <i>FEBS Letters</i> , 2004, 577, 555-562.	1.3	15
93	In vitro efficacy of combined treatment depends on the underlying mechanism of resistance in imatinib-resistant Bcr-Abl ⁺ positive cell lines. <i>Blood</i> , 2004, 103, 208-215.	0.6	93
94	c-CBL is not required for leukemia induction by Bcr-Abl in mice. <i>Oncogene</i> , 2003, 22, 8852-8860.	2.6	13
95	Specific Targeted Therapy of Chronic Myelogenous Leukemia with Imatinib. <i>Pharmacological Reviews</i> , 2003, 55, 401-423.	7.1	305
96	Imatinib As a Paradigm of Targeted Therapies. <i>Journal of Clinical Oncology</i> , 2003, 21, 239s-245.	0.8	50
97	Imatinib mesylate in the treatment of chronic myeloid leukaemia. <i>Expert Opinion on Pharmacotherapy</i> , 2003, 4, 963-971.	0.9	45
98	Practical Management of Patients With Chronic Myeloid Leukemia Receiving Imatinib. <i>Journal of Clinical Oncology</i> , 2003, 21, 1637-1647.	0.8	364
99	Several Bcr-Abl kinase domain mutants associated with imatinib mesylate resistance remain sensitive to imatinib. <i>Blood</i> , 2003, 101, 4611-4614.	0.6	304
100	No correlation between the proliferative status of Bcr-Abl positive cell lines and the proapoptotic activity of imatinib mesylate (Gleevec [®] /Glivec [®]). <i>The Hematology Journal</i> , 2003, 4, 413-419.	2.0	16
101	Imatinib induces durable hematologic and cytogenetic responses in patients with accelerated phase chronic myeloid leukemia: results of a phase 2 study. <i>Blood</i> , 2002, 99, 1928-1937.	0.6	943
102	Imatinib induces hematologic and cytogenetic responses in patients with chronic myelogenous leukemia in myeloid blast crisis: results of a phase II study. <i>Blood</i> , 2002, 99, 3530-3539.	0.6	1,096
103	A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive acute lymphoid leukemias. <i>Blood</i> , 2002, 100, 1965-1971.	0.6	534
104	STI571 (Gleevec [®]) as a paradigm for cancer therapy. <i>Trends in Molecular Medicine</i> , 2002, 8, S14-S18.	3.5	227
105	Perspectives on the development of a molecularly targeted agent. <i>Cancer Cell</i> , 2002, 1, 31-36.	7.7	260
106	Inhibition of the Bcr-Abl tyrosine kinase as a therapeutic strategy for CML. <i>Oncogene</i> , 2002, 21, 8541-8546.	2.6	107
107	Efficacy and Safety of a Specific Inhibitor of the BCR-ABL Tyrosine Kinase in Chronic Myeloid Leukemia. <i>New England Journal of Medicine</i> , 2001, 344, 1031-1037.	13.9	4,825
108	Activity of a Specific Inhibitor of the BCR-ABL Tyrosine Kinase in the Blast Crisis of Chronic Myeloid Leukemia and Acute Lymphoblastic Leukemia with the Philadelphia Chromosome. <i>New England Journal of Medicine</i> , 2001, 344, 1038-1042.	13.9	2,593