## Brian J Druker

List of Publications by Year in descending order

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108 papers 28,572 citations

23565 58 h-index 106 g-index

108 all docs 108 docs citations

108 times ranked 20289 citing authors

#	Article	IF	CITATIONS
1	Efficacy and Safety of a Specific Inhibitor of the BCR-ABL Tyrosine Kinase in Chronic Myeloid Leukemia. New England Journal of Medicine, 2001, 344, 1031-1037.	27.0	4,825
2	Five-Year Follow-up of Patients Receiving Imatinib for Chronic Myeloid Leukemia. New England Journal of Medicine, 2006, 355, 2408-2417.	27.0	3,212
3	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. New England Journal of Medicine, 2001, 344, 1038-1042.	27.0	2,593
4	The development of imatinib as a therapeutic agent for chronic myeloid leukemia. Blood, 2005, 105, 2640-2653.	1.4	1,137
5	Imatinib induces hematologic and cytogenetic responses in patients with chronic myelogenous leukemia in myeloid blast crisis: results of a phase II study. Blood, 2002, 99, 3530-3539.	1.4	1,096
6	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
7	In vitro Activity of Bcr-Abl Inhibitors AMN107 and BMS-354825 against Clinically Relevant Imatinib-Resistant Abl Kinase Domain Mutants. Cancer Research, 2005, 65, 4500-4505.	0.9	997
8	Imatinib induces durable hematologic and cytogenetic responses in patients with accelerated phase chronic myeloid leukemia: results of a phase 2 study. Blood, 2002, 99, 1928-1937.	1.4	943
9	Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. New England Journal of Medicine, 2017, 376, 917-927.	27.0	926
10	Functional genomic landscape of acute myeloid leukaemia. Nature, 2018, 562, 526-531.	27.8	907
11	Ponatinib in Refractory Philadelphia Chromosome–Positive Leukemias. New England Journal of Medicine, 2012, 367, 2075-2088.	27.0	668
12	Human chronic myeloid leukemia stem cells are insensitive to imatinib despite inhibition of BCR-ABL activity. Journal of Clinical Investigation, 2011, 121, 396-409.	8.2	661
12	Human chronic myeloid leukemia stem cells are insensitive to imatinib despite inhibition of BCR-ABL activity. Journal of Clinical Investigation, 2011, 121, 396-409.  Translation of the Philadelphia chromosome into therapy for CML. Blood, 2008, 112, 4808-4817.	8.2	661 587
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13	activity. Journal of Clinical Investigation, 2011, 121, 396-409.  Translation of the Philadelphia chromosome into therapy for CML. Blood, 2008, 112, 4808-4817.  A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive	1.4	587
13	activity. Journal of Clinical Investigation, 2011, 121, 396-409.  Translation of the Philadelphia chromosome into therapy for CML. Blood, 2008, 112, 4808-4817.  A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive acute lymphoid leukemias. Blood, 2002, 100, 1965-1971.  Oncogenic < i>CSF3R < /i> Mutations in Chronic Neutrophilic Leukemia and Atypical CML. New England	1.4	587 534
13 14 15	Translation of the Philadelphia chromosome into therapy for CML. Blood, 2008, 112, 4808-4817.  A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive acute lymphoid leukemias. Blood, 2002, 100, 1965-1971.  Oncogenic < i > CSF3R < / i > Mutations in Chronic Neutrophilic Leukemia and Atypical CML. New England Journal of Medicine, 2013, 368, 1781-1790.  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. Cancer	1.4 1.4 27.0	587 534 499

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19	Several Bcr-Abl kinase domain mutants associated with imatinib mesylate resistance remain sensitive to imatinib. Blood, 2003, 101, 4611-4614.	1.4	304
20	Activating alleles of JAK3 in acute megakaryoblastic leukemia. Cancer Cell, 2006, 10, 65-75.	16.8	295
21	BCR-ABL1 Compound Mutations Combining Key Kinase Domain Positions Confer Clinical Resistance to Ponatinib in Ph Chromosome-Positive Leukemia. Cancer Cell, 2014, 26, 428-442.	16.8	292
22	Perspectives on the development of a molecularly targeted agent. Cancer Cell, 2002, 1, 31-36.	16.8	260
23	STI571 (Gleevecâ,,¢) as a paradigm for cancer therapy. Trends in Molecular Medicine, 2002, 8, S14-S18.	6.7	227
24	TNFÎ $\pm$ facilitates clonal expansion of JAK2V617F positive cells in myeloproliferative neoplasms. Blood, 2011, 118, 6392-6398.	1.4	227
25	CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms. Blood, 2010, 115, 5232-5240.	1.4	216
26	The TP53 Apoptotic Network Is a Primary Mediator of Resistance to BCL2 Inhibition in AML Cells. Cancer Discovery, 2019, 9, 910-925.	9.4	215
27	Kinase Domain Mutants of Bcr-Abl Exhibit Altered Transformation Potency, Kinase Activity, and Substrate Utilization, Irrespective of Sensitivity to Imatinib. Molecular and Cellular Biology, 2006, 26, 6082-6093.	2.3	192
28	BCR-ABL1 compound mutations in tyrosine kinase inhibitor–resistant CML: frequency and clonal relationships. Blood, 2013, 121, 489-498.	1.4	187
29	Characterization of Murine JAK2V617F-Positive Myeloproliferative Disease. Cancer Research, 2006, 66, 11156-11165.	0.9	184
30	Identification of Driver and Passenger Mutations of FLT3 by High-Throughput DNA Sequence Analysis and Functional Assessment of Candidate Alleles. Cancer Cell, 2007, 12, 501-513.	16.8	174
31	Targeted CML therapy: controlling drug resistance, seeking cure. Current Opinion in Genetics and Development, 2006, 16, 92-99.	3.3	166
32	Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. Cancer Research, 2019, 79, 4744-4753.	0.9	139
33	Kinase Pathway Dependence in Primary Human Leukemias Determined by Rapid Inhibitor Screening. Cancer Research, 2013, 73, 285-296.	0.9	134
34	Targeting the BCR-ABL Signaling Pathway in Therapy-Resistant Philadelphia Chromosome-Positive Leukemia. Clinical Cancer Research, 2011, 17, 212-221.	7.0	127
35	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. Blood, 2006, 107, 4250-4256.	1.4	120
36	High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. Blood, 2009, 113, 1749-1755.	1.4	119

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37	Age-related mutations and chronic myelomonocytic leukemia. Leukemia, 2016, 30, 906-913.	7.2	119
38	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. Clinical Cancer Research, 2007, 13, 6136-6143.	7.0	115
39	Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. Nature Communications, 2019, 10, 244.	12.8	111
40	RNAi screen for rapid therapeutic target identification in leukemia patients. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 8695-8700.	7.1	110
41	A gene expression signature of CD34+ cells to predict major cytogenetic response in chronic-phase chronic myeloid leukemia patients treated with imatinib. Blood, 2010, 115, 315-325.	1.4	108
42	Inhibition of the Bcr-Abl tyrosine kinase as a therapeutic strategy for CML. Oncogene, 2002, 21, 8541-8546.	5.9	107
43	Precision medicine treatment in acute myeloid leukemia using prospective genomic profiling: feasibility and preliminary efficacy of the Beat AML Master Trial. Nature Medicine, 2020, 26, 1852-1858.	30.7	104
44	Perspectives on the development of imatinib and the future of cancer research. Nature Medicine, 2009, 15, 1149-1152.	30.7	101
45	Blockade of JAK2-mediated extrinsic survival signals restores sensitivity of CML cells to ABL inhibitors. Leukemia, 2012, 26, 1140-1143.	7.2	97
46	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. Leukemia, 2019, 33, 1835-1850.	7.2	97
47	Combined Abl Inhibitor Therapy for Minimizing Drug Resistance in Chronic Myeloid Leukemia: Src/Abl Inhibitors Are Compatible with Imatinib. Clinical Cancer Research, 2005, 11, 6987-6993.	7.0	96
48	In vitro efficacy of combined treatment depends on the underlying mechanism of resistance in imatinib-resistant Bcr-Abl–positive cell lines. Blood, 2004, 103, 208-215.	1.4	93
49	AMN107: Tightening the grip of imatinib. Cancer Cell, 2005, 7, 117-119.	16.8	93
50	Structural insight into selectivity and resistance profiles of ROS1 tyrosine kinase inhibitors. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E5381-90.	7.1	93
51	The ABL Switch Control Inhibitor DCC-2036 Is Active against the Chronic Myeloid Leukemia Mutant BCR-ABLT315I and Exhibits a Narrow Resistance Profile. Cancer Research, 2011, 71, 3189-3195.	0.9	91
52	Molecularly targeted drug combinations demonstrate selective effectiveness for myeloid- and lymphoid-derived hematologic malignancies. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E7554-E7563.	7.1	86
53	High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. Blood, 2008, 111, 4788-4796.	1.4	84
54	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. Cancer Research, 2005, 65, 9436-9444.	0.9	80

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55	Mutations of the BCR-ABL-kinase domain occur in a minority of patients with stable complete cytogenetic response to imatinib. Leukemia, 2007, 21, 489-493.	7.2	74
56	RNAi screening of the tyrosine kinome identifies therapeutic targets in acute myeloid leukemia. Blood, 2008, 111, 2238-2245.	1.4	67
57	MET Receptor Sequence Variants R970C and T992I Lack Transforming Capacity. Cancer Research, 2010, 70, 6233-6237.	0.9	65
58	Detection of ABL kinase domain mutations with denaturing high-performance liquid chromatography. Leukemia, 2004, 18, 864-871.	7.2	62
59	Identification of mTOR as a novel bifunctional target in chronic myeloid leukemia: dissection of growthâ€inhibitory and VEGFâ€suppressive effects of rapamycin in leukemic cells. FASEB Journal, 2005, 19, 960-962.	0.5	56
60	Imatinib 800Âmg daily induces deeper molecular responses than imatinib 400Âmg daily: results of <scp>SWOG</scp> S0325, an intergroup randomized <scp>PHASE II</scp> trial in newly diagnosed chronic phase chronic myeloid leukaemia. British Journal of Haematology, 2014, 164, 223-232.	2.5	56
61	An Intron-Derived Insertion/Truncation Mutation in the BCR-ABL Kinase Domain in Chronic Myeloid Leukemia Patients Undergoing Kinase Inhibitor Therapy. Journal of Molecular Diagnostics, 2008, 10, 177-180.	2.8	55
62	Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. Blood, 2019, 134, 867-879.	1.4	55
63	Phosphoproteomic analysis of AML cell lines identifies leukemic oncogenes. Leukemia Research, 2006, 30, 1097-1104.	0.8	53
64	Zoledronate inhibits proliferation and induces apoptosis of imatinib-resistant chronic myeloid leukaemia cells. Leukemia, 2005, 19, 1896-1904.	7.2	52
65	Imatinib As a Paradigm of Targeted Therapies. Journal of Clinical Oncology, 2003, 21, 239s-245.	1.6	50
66	Molecularly Targeted Therapy: Have the Floodgates Opened?. Oncologist, 2004, 9, 357-360.	3.7	48
67	Identification and prioritization of myeloid malignancy germline variants in a large cohort of adult patients with AML. Blood, 2022, 139, 1208-1221.	1.4	48
68	Imatinib mesylate in the treatment of chronic myeloid leukaemia. Expert Opinion on Pharmacotherapy, 2003, 4, 963-971.	1.8	45
69	RNAi-induced down-regulation of FLT3 expression in AML cell lines increases sensitivity to MLN518. Blood, 2005, 105, 2952-2954.	1.4	39
70	Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. Leukemia, 2008, 22, 1184-1190.	7.2	38
71	e8a2 BCR–ABL: more frequent than other atypical BCR–ABL variants?. Leukemia, 2005, 19, 681-684.	7.2	35
72	SRCircumventing imatinib resistance. Cancer Cell, 2004, 6, 108-110.	16.8	33

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73	A Specific Need for CRKL in p210BCR-ABL–Induced Transformation of Mouse Hematopoietic Progenitors. Cancer Research, 2010, 70, 7325-7335.	0.9	33
74	An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. Leukemia, 2008, 22, 2269-2272.	7.2	32
75	YM155 potently kills acute lymphoblastic leukemia cells through activation of the DNA damage pathway. Journal of Hematology and Oncology, 2015, 8, 39.	17.0	32
76	Threshold Levels of ABL Tyrosine Kinase Inhibitors Retained in Chronic Myeloid Leukemia Cells Determine Their Commitment to Apoptosis. Cancer Research, 2013, 73, 3356-3370.	0.9	26
77	NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2V617F-positive myeloproliferative neoplasm cells. Signal Transduction and Targeted Therapy, 2020, 5, 5.	17.1	26
78	A BCR-ABL Mutant Lacking Direct Binding Sites for the GRB2, CBL and CRKL Adapter Proteins Fails to Induce Leukemia in Mice. PLoS ONE, 2009, 4, e7439.	2.5	24
79	Understanding Drug Sensitivity and Tackling Resistance in Cancer. Cancer Research, 2022, 82, 1448-1460.	0.9	24
80	Establishment of a murine model of aggressive systemic mastocytosis/mast cell leukemia. Experimental Hematology, 2006, 34, 284-288.	0.4	22
81	Antileukemic Activity of Lysophosphatidic Acid Acyltransferase-β Inhibitor CT32228 in Chronic Myelogenous Leukemia Sensitive and Resistant to Imatinib. Clinical Cancer Research, 2006, 12, 6540-6546.	7.0	22
82	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. Cancer Research, 2013, 73, 5775-5786.	0.9	22
83	Myeloid lineage enhancers drive oncogene synergy in CEBPA/CSF3R mutant acute myeloid leukemia. Nature Communications, 2019, 10, 5455.	12.8	22
84	Proteasome 26S subunit, non-ATPases 1 (PSMD1) and 3 (PSMD3), play an oncogenic role in chronic myeloid leukemia by stabilizing nuclear factor-kappa B. Oncogene, 2021, 40, 2697-2710.	5.9	20
85	A single nucleotide polymorphism in the coding region of ABL and its effects on sensitivity to imatinib. Leukemia, 2005, 19, 1859-1862.	7.2	18
86	Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. Leukemia, 2020, 34, 2342-2353.	7.2	18
87	ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. Leukemia, 2020, 34, 2798-2804.	7.2	16
88	No correlation between the proliferative status of Bcr-Abl positive cell lines and the proapoptotic activity of imatinib mesylate (Gleevecâ,,¢/Glivec®). The Hematology Journal, 2003, 4, 413-419.	1.4	16
89	Catalytic domains of tyrosine kinases determine the phosphorylation sites within c-Cbl. FEBS Letters, 2004, 577, 555-562.	2.8	15
90	High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. Leukemia, 2009, 23, 406-409.	7.2	15

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91	c-CBL is not required for leukemia induction by Bcr-Abl in mice. Oncogene, 2003, 22, 8852-8860.	5.9	13
92	The function of the pleckstrin homology domain in BCR–ABL-mediated leukemogenesis. Leukemia, 2010, 24, 226-229.	7.2	12
93	Bayesian multi-source regression and monocyte-associated gene expression predict BCL-2 inhibitor resistance in acute myeloid leukemia. Npj Precision Oncology, 2021, 5, 71.	5.4	12
94	Mutated <i>SETBP1</i> activates transcription of Myc programs to accelerate <i>CSF3R</i> driven myeloproliferative neoplasms. Blood, 2022, 140, 644-658.	1.4	12
95	Challenges and approaches to implementing master/basket trials in oncology. Blood Advances, 2019, 3, 2237-2243.	5.2	11
96	Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. Leukemia, 2016, 30, 1418-1421.	7.2	9
97	Coexistence of phosphotyrosine-dependent and -independent interactions between Cbl and Bcr-Abl. Experimental Hematology, 2004, 32, 113-121.	0.4	8
98	Bruton's tyrosine kinase is not essential for Bcr-Abl-mediated transformation of lymphoid or myeloid cells. Leukemia, 2008, 22, 1354-1360.	7.2	8
99	A novel <i>AGGF1-PDGFRb</i> fusion in pediatric T-cell acute lymphoblastic leukemia. Haematologica, 2018, 103, e87-e91.	3.5	8
100	MS4A3 promotes differentiation in chronic myeloid leukemia by enhancing common $\hat{l}^2$ -chain cytokine receptor endocytosis. Blood, 2022, 139, 761-778.	1.4	7
101	PU.1 and MYC transcriptional network defines synergistic drug responses to KIT and LSD1 inhibition in acute myeloid leukemia. Leukemia, 2022, , .	7.2	7
102	Clonal chromosomal abnormalities in CD34+/CD38â^' hematopoietic cells from cytogenetically normal chronic myeloid leukemia patients with a complete cytogenetic response to tyrosine kinase inhibitors. Leukemia, 2010, 24, 1525-1528.	7.2	6
103	Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. Cancer Research, 2021, 81, 4685-4695.	0.9	6
104	Associating drug sensitivity with differentiation status identifies effective combinations for acute myeloid leukemia. Blood Advances, 2022, 6, 3062-3067.	5.2	6
105	Functional characterization of an activating TEK mutation in acute myeloid leukemia: a cellular context-dependent activating mutation. Leukemia, 2009, 23, 1345-1348.	7.2	5
106	Luxeptinib (CG-806) Targets FLT3 and Clusters of Kinases Operative in Acute Myeloid Leukemia. Molecular Cancer Therapeutics, 2022, 21, 1125-1135.	4.1	4
107	Peter C. Nowell (1928–2016). Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 4569-4570.	7.1	0
108	BCR-ABL+ Chronic Myeloid Leukemia Arising in a Family With Inherited ANKRD26-Related Thrombocytopenia. JCO Precision Oncology, 2021, 5, 415-417.	3.0	0