Brian J Druker

List of Publications by Year in Descending Order

102 papers	23,958 citations	56 h-index	108 g-index
108 ext. papers	26,503 ext. citations	12.8 avg, IF	6.42 L-index

#	Paper	IF	Citations
102	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	9.2	8
101	Bayesian multi-source regression and monocyte-associated gene expression predict BCL-2 inhibitor resistance in acute myeloid leukemia. *Npj Precision Oncology*, **2021**, 5, 71	9.8	0
100	BCR-ABL+ Chronic Myeloid Leukemia Arising in a Family With Inherited ANKRD26-Related Thrombocytopenia. *JCO Precision Oncology*, **2021**, 5,	3.6	
99	Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. *Cancer Research*, **2021**, 81, 4685-4695	10.1	2
98	Identification and prioritization of myeloid malignancy germline variants in a large cohort of adult AML patients. *Blood*, **2021**,	2.2	3
97	Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. *Leukemia*, **2020**, 34, 2342-2353	10.7	7
96	NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2-positive myeloproliferative neoplasm cells. *Signal Transduction and Targeted Therapy*, **2020**, 5, 5	21	15
95	ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. *Leukemia*, **2020**, 34, 2798-2804	10.7	6
94	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	50.5	32
93	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. *Leukemia*, **2019**, 33, 1835-1850	10.7	50
92	The TP53 Apoptotic Network Is a Primary Mediator of Resistance to BCL2 Inhibition in AML Cells. *Cancer Discovery*, **2019**, 9, 910-925	24.4	98
91	Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. *Blood*, **2019**, 134, 867-879	2.2	29
90	Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. *Cancer Research*, **2019**, 79, 4744-4753	10.1	87
89	Challenges and approaches to implementing master/basket trials in oncology. *Blood Advances*, **2019**, 3, 2237-2243	7.8	8
88	Myeloid lineage enhancers drive oncogene synergy in CEBPA/CSF3R mutant acute myeloid leukemia. *Nature Communications*, **2019**, 10, 5455	17.4	11
87	Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. *Nature Communications*, **2019**, 10, 244	17.4	63
86	A novel fusion in pediatric T-cell acute lymphoblastic leukemia. *Haematologica*, **2018**, 103, e87-e91	6.6	8

85	Functional genomic landscape of acute myeloid leukaemia. *Nature*, **2018**, 562, 526-531	50.4	391
84	Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. *New England Journal of Medicine*, **2017**, 376, 917-927	59.2	618
83	Peter C. Nowell (1928-2016). *Proceedings of the National Academy of Sciences of the United States of America*, **2017**, 114, 4569-4570	11.5	
82	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	11.5	58
81	Age-related mutations and chronic myelomonocytic leukemia. *Leukemia*, **2016**, 30, 906-13	10.7	94
80	Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. *Leukemia*, **2016**, 30, 1418-21	10.7	7
79	YM155 potently kills acute lymphoblastic leukemia cells through activation of the DNA damage pathway. *Journal of Hematology and Oncology*, **2015**, 8, 39	22.4	25
78	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	11.5	72
77	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	24.3	233
76	Imatinib 800 mg daily induces deeper molecular responses than imatinib 400 mg daily: results of SWOG S0325, an intergroup randomized PHASE II trial in newly diagnosed chronic phase chronic myeloid leukaemia. *British Journal of Haematology*, **2014**, 164, 223-32	4.5	46
75	BCR-ABL1 compound mutations in tyrosine kinase inhibitor-resistant CML: frequency and clonal relationships. *Blood*, **2013**, 121, 489-98	2.2	154
74	Threshold levels of ABL tyrosine kinase inhibitors retained in chronic myeloid leukemia cells determine their commitment to apoptosis. *Cancer Research*, **2013**, 73, 3356-70	10.1	22
73	Oncogenic CSF3R mutations in chronic neutrophilic leukemia and atypical CML. *New England Journal of Medicine*, **2013**, 368, 1781-90	59.2	388
72	KIT signaling governs differential sensitivity of mature and primitive CML progenitors to tyrosine kinase inhibitors. *Cancer Research*, **2013**, 73, 5775-86	10.1	17
71	Kinase pathway dependence in primary human leukemias determined by rapid inhibitor screening. *Cancer Research*, **2013**, 73, 285-96	10.1	106
70	Ponatinib in refractory Philadelphia chromosome-positive leukemias. *New England Journal of Medicine*, **2012**, 367, 2075-88	59.2	556
69	Blockade of JAK2-mediated extrinsic survival signals restores sensitivity of CML cells to ABL inhibitors. *Leukemia*, **2012**, 26, 1140-3	10.7	85
68	TNF facilitates clonal expansion of JAK2V617F positive cells in myeloproliferative neoplasms. *Blood*, **2011**, 118, 6392-8	2.2	179

67	Human chronic myeloid leukemia stem cells are insensitive to imatinib despite inhibition of BCR-ABL activity. *Journal of Clinical Investigation*, **2011**, 121, 396-409	15.9	555
66	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, 3189-95	10.1	78
65	Targeting the BCR-ABL signaling pathway in therapy-resistant Philadelphia chromosome-positive leukemia. *Clinical Cancer Research*, **2011**, 17, 212-21	12.9	107
64	The function of the pleckstrin homology domain in BCR-ABL-mediated leukemogenesis. *Leukemia*, **2010**, 24, 226-9	10.7	10
63	Clonal chromosomal abnormalities in CD34+/CD38- hematopoietic cells from cytogenetically normal chronic myeloid leukemia patients with a complete cytogenetic response to tyrosine kinase inhibitors. *Leukemia*, **2010**, 24, 1525-1528	10.7	6
62	MET receptor sequence variants R970C and T992I lack transforming capacity. *Cancer Research*, **2010**, 70, 6233-7	10.1	63
61	A specific need for CRKL in p210BCR-ABL-induced transformation of mouse hematopoietic progenitors. *Cancer Research*, **2010**, 70, 7325-35	10.1	28
60	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, 315-25	2.2	98
59	CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms. *Blood*, **2010**, 115, 5232-40	2.2	188
58	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	3.7	23
57	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, 8695-700	11.5	88
56	AP24534, a pan-BCR-ABL inhibitor for chronic myeloid leukemia, potently inhibits the T315I mutant and overcomes mutation-based resistance. *Cancer Cell*, **2009**, 16, 401-12	24.3	852
55	High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. *Leukemia*, **2009**, 23, 406-9	10.7	14
54	Functional characterization of an activating TEK mutation in acute myeloid leukemia: a cellular context-dependent activating mutation. *Leukemia*, **2009**, 23, 1345-8	10.7	4
53	Perspectives on the development of imatinib and the future of cancer research. *Nature Medicine*, **2009**, 15, 1149-52	50.5	87
52	High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. *Blood*, **2009**, 113, 1749-55	2.2	97
51	An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. *Leukemia*, **2008**, 22, 2269-72	10.7	28
50	Bruton's tyrosine kinase is not essential for Bcr-Abl-mediated transformation of lymphoid or myeloid cells. *Leukemia*, **2008**, 22, 1354-60	10.7	8

49	Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. *Leukemia*, **2008**, 22, 1184-90	10.7	33
48	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, 177-80	5.1	50
47	Translation of the Philadelphia chromosome into therapy for CML. *Blood*, **2008**, 112, 4808-17	2.2	520
46	RNAi screening of the tyrosine kinome identifies therapeutic targets in acute myeloid leukemia. *Blood*, **2008**, 111, 2238-45	2.2	62
45	High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. *Blood*, **2008**, 111, 4788-96	2.2	77
44	Mutations of the BCR-ABL-kinase domain occur in a minority of patients with stable complete cytogenetic response to imatinib. *Leukemia*, **2007**, 21, 489-93	10.7	69
43	Identification of driver and passenger mutations of FLT3 by high-throughput DNA sequence analysis and functional assessment of candidate alleles. *Cancer Cell*, **2007**, 12, 501-13	24.3	154
42	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, 6136-43	12.9	102
41	Establishment of a murine model of aggressive systemic mastocytosis/mast cell leukemia. *Experimental Hematology*, **2006**, 34, 284-8	3.1	18
40	Activating alleles of JAK3 in acute megakaryoblastic leukemia. *Cancer Cell*, **2006**, 10, 65-75	24.3	265
39	Antileukemic activity of lysophosphatidic acid acyltransferase-beta inhibitor CT32228 in chronic myelogenous leukemia sensitive and resistant to imatinib. *Clinical Cancer Research*, **2006**, 12, 6540-6	12.9	20
38	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 Research*, **2006**, 66, 473-81	10.1	398
37	Characterization of murine JAK2V617F-positive myeloproliferative disease. *Cancer Research*, **2006**, 66, 11156-65	10.1	168
36	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, 6082-93	4.8	174
35	Five-year follow-up of patients receiving imatinib for chronic myeloid leukemia. *New England Journal of Medicine*, **2006**, 355, 2408-17	59.2	2811
34	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, 4250-6	2.2	101
33	Targeted CML therapy: controlling drug resistance, seeking cure. *Current Opinion in Genetics and Development*, **2006**, 16, 92-9	4.9	153
32	Phosphoproteomic analysis of AML cell lines identifies leukemic oncogenes. *Leukemia Research*, **2006**, 30, 1097-104	2.7	51

31	The development of imatinib as a therapeutic agent for chronic myeloid leukemia. *Blood*, **2005**, 105, 2640-53	2.2	1027
30	RNAi-induced down-regulation of FLT3 expression in AML cell lines increases sensitivity to MLN518. *Blood*, **2005**, 105, 2952-4	2.2	36
29	In vitro activity of Bcr-Abl inhibitors AMN107 and BMS-354825 against clinically relevant imatinib-resistant Abl kinase domain mutants. *Cancer Research*, **2005**, 65, 4500-5	10.1	904
28	e8a2 BCR-ABL: more frequent than other atypical BCR-ABL variants?. *Leukemia*, **2005**, 19, 681-4	10.7	34
27	A single nucleotide polymorphism in the coding region of ABL and its effects on sensitivity to imatinib. *Leukemia*, **2005**, 19, 1859-62	10.7	17
26	Zoledronate inhibits proliferation and induces apoptosis of imatinib-resistant chronic myeloid leukaemia cells. *Leukemia*, **2005**, 19, 1896-904	10.7	51
25	AMN107: tightening the grip of imatinib. *Cancer Cell*, **2005**, 7, 117-9	24.3	87
24	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, 9436-44	10.1	76
23	Combined Abl inhibitor therapy for minimizing drug resistance in chronic myeloid leukemia: Src/Abl inhibitors are compatible with imatinib. *Clinical Cancer Research*, **2005**, 11, 6987-93	12.9	88
22	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. *FASEB Journal*, **2005** , 19, 960-2	0.9	56
21	Molecularly targeted therapy: have the floodgates opened?. *Oncologist*, **2004**, 9, 357-60	5.7	43
20	Detection of ABL kinase domain mutations with denaturing high-performance liquid chromatography. *Leukemia*, **2004**, 18, 864-71	10.7	55
19	Coexistence of phosphotyrosine-dependent and -independent interactions between Cbl and Bcr-Abl. *Experimental Hematology*, **2004**, 32, 113-21	3.1	8
18	SRCircumventing imatinib resistance. *Cancer Cell*, **2004**, 6, 108-10	24.3	31
17	Catalytic domains of tyrosine kinases determine the phosphorylation sites within c-Cbl. *FEBS Letters*, **2004**, 577, 555-62	3.8	14
16	In vitro efficacy of combined treatment depends on the underlying mechanism of resistance in imatinib-resistant Bcr-Abl-positive cell lines. *Blood*, **2004**, 103, 208-15	2.2	88
15	Practical management of patients with chronic myeloid leukemia receiving imatinib. *Journal of Clinical Oncology*, **2003**, 21, 1637-47	2.2	325
14	Several Bcr-Abl kinase domain mutants associated with imatinib mesylate resistance remain sensitive to imatinib. *Blood*, **2003**, 101, 4611-4	2.2	280

13	c-CBL is not required for leukemia induction by Bcr-Abl in mice. *Oncogene*, **2003**, 22, 8852-60	9.2	13
12	Specific targeted therapy of chronic myelogenous leukemia with imatinib. *Pharmacological Reviews*, **2003**, 55, 401-23	22.5	257
11	David A. Karnofsky Award lecture. Imatinib as a paradigm of targeted therapies. *Journal of Clinical Oncology*, **2003**, 21, 239s-245s	2.2	47
10	Imatinib mesylate in the treatment of chronic myeloid leukaemia. *Expert Opinion on Pharmacotherapy*, **2003**, 4, 963-71	4	40
9	No correlation between the proliferative status of Bcr-Abl positive cell lines and the proapoptotic activity of imatinib mesylate (Gleevec/Glivec). *The Hematology Journal*, **2003**, 4, 413-9		15
8	Inhibition of the Bcr-Abl tyrosine kinase as a therapeutic strategy for CML. *Oncogene*, **2002**, 21, 8541-6	9.2	100
7	Imatinib induces durable hematologic and cytogenetic responses in patients with accelerated phase chronic myeloid leukemia: results of a phase 2 study. *Blood*, **2002**, 99, 1928-37	2.2	850
6	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, 3530-9	2.2	986
5	A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive acute lymphoid leukemias. *Blood*, **2002**, 100, 1965-71	2.2	480
4	STI571 (Gleevec) as a paradigm for cancer therapy. *Trends in Molecular Medicine*, **2002**, 8, S14-8	11.5	197
3	Perspectives on the development of a molecularly targeted agent. *Cancer Cell*, **2002**, 1, 31-6	24.3	225
2	Efficacy and safety of a specific inhibitor of the BCR-ABL tyrosine kinase in chronic myeloid leukemia. *New England Journal of Medicine*, **2001**, 344, 1031-7	59.2	4179
1	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, 1038-42	59.2	2309