

# Brian J Druker

## List of Publications by Citations

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The third column is the impact factor (IF) of the journal, and the fourth column is the number of citations of the article.

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 | Efficacy and safety of a specific inhibitor of the BCR-ABL tyrosine kinase in chronic myeloid leukemia. <i>New England Journal of Medicine</i> , <b>2001</b> , 344, 1031-7   | 59.2 | 4179      |
| 101 | Five-year follow-up of patients receiving imatinib for chronic myeloid leukemia. <i>New England Journal of Medicine</i> , <b>2006</b> , 355, 2408-17   | 59.2 | 2811      |
| 100 | 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> , <b>2001</b> , 344, 1038-42 | 59.2 | 2309      |
| 99  | The development of imatinib as a therapeutic agent for chronic myeloid leukemia. <i>Blood</i> , <b>2005</b> , 105, 2640-53   | 10.1 | 1027      |
| 98  | 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> , <b>2002</b> , 99, 3530-9   | 2.2  | 986       |
| 97  | In vitro activity of Bcr-Abl inhibitors AMN107 and BMS-354825 against clinically relevant imatinib-resistant Abl kinase domain mutants. <i>Cancer Research</i> , <b>2005</b> , 65, 4500-5  | 10.1 | 904       |
| 96  | AP24534, a pan-BCR-ABL inhibitor for chronic myeloid leukemia, potently inhibits the T315I mutant and overcomes mutation-based resistance. <i>Cancer Cell</i> , <b>2009</b> , 16, 401-12   | 24.3 | 852       |
| 95  | Imatinib induces durable hematologic and cytogenetic responses in patients with accelerated phase chronic myeloid leukemia: results of a phase 2 study. <i>Blood</i> , <b>2002</b> , 99, 1928-37   | 2.2  | 850       |
| 94  | Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. <i>New England Journal of Medicine</i> , <b>2017</b> , 376, 917-927   | 59.2 | 618       |
| 93  | Ponatinib in refractory Philadelphia chromosome-positive leukemias. <i>New England Journal of Medicine</i> , <b>2012</b> , 367, 2075-88  | 59.2 | 556       |
| 92  | Human chronic myeloid leukemia stem cells are insensitive to imatinib despite inhibition of BCR-ABL activity. <i>Journal of Clinical Investigation</i> , <b>2011</b> , 121, 396-409  | 15.9 | 555       |
| 91  | Translation of the Philadelphia chromosome into therapy for CML. <i>Blood</i> , <b>2008</b> , 112, 4808-17   | 2.2  | 520       |
| 90  | A phase 2 study of imatinib in patients with relapsed or refractory Philadelphia chromosome-positive acute lymphoid leukemias. <i>Blood</i> , <b>2002</b> , 100, 1965-71   | 2.2  | 480       |
| 89  | 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> , <b>2006</b> , 66, 473-81       | 10.1 | 398       |
| 88  | Functional genomic landscape of acute myeloid leukaemia. <i>Nature</i> , <b>2018</b> , 562, 526-531  | 50.4 | 391       |
| 87  | Oncogenic CSF3R mutations in chronic neutrophilic leukemia and atypical CML. <i>New England Journal of Medicine</i> , <b>2013</b> , 368, 1781-90   | 59.2 | 388       |
| 86  | Practical management of patients with chronic myeloid leukemia receiving imatinib. <i>Journal of Clinical Oncology</i> , <b>2003</b> , 21, 1637-47   | 2.2  | 325       |

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|----|--|------|-----|
| 85 | Several Bcr-Abl kinase domain mutants associated with imatinib mesylate resistance remain sensitive to imatinib. <i>Blood</i> , <b>2003</b> , 101, 4611-4  | 2.2  | 280 |
| 84 | Activating alleles of JAK3 in acute megakaryoblastic leukemia. <i>Cancer Cell</i> , <b>2006</b> , 10, 65-75  | 24.3 | 265 |
| 83 | Specific targeted therapy of chronic myelogenous leukemia with imatinib. <i>Pharmacological Reviews</i> , <b>2003</b> , 55, 401-23   | 22.5 | 257 |
| 82 | BCR-ABL1 compound mutations combining key kinase domain positions confer clinical resistance to ponatinib in Ph chromosome-positive leukemia. <i>Cancer Cell</i> , <b>2014</b> , 26, 428-442                                       | 24.3 | 233 |
| 81 | Perspectives on the development of a molecularly targeted agent. <i>Cancer Cell</i> , <b>2002</b> , 1, 31-6  | 24.3 | 225 |
| 80 | STI571 (Gleevec) as a paradigm for cancer therapy. <i>Trends in Molecular Medicine</i> , <b>2002</b> , 8, S14-8  | 11.5 | 197 |
| 79 | CYT387, a novel JAK2 inhibitor, induces hematologic responses and normalizes inflammatory cytokines in murine myeloproliferative neoplasms. <i>Blood</i> , <b>2010</b> , 115, 5232-40  | 2.2  | 188 |
| 78 | TNF $\alpha$ facilitates clonal expansion of JAK2V617F positive cells in myeloproliferative neoplasms. <i>Blood</i> , <b>2011</b> , 118, 6392-8  | 2.2  | 179 |
| 77 | 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> , <b>2006</b> , 26, 6082-93    | 11.8 | 174 |
| 76 | Characterization of murine JAK2V617F-positive myeloproliferative disease. <i>Cancer Research</i> , <b>2006</b> , 66, 11156-65  | 10.1 | 168 |
| 75 | BCR-ABL1 compound mutations in tyrosine kinase inhibitor-resistant CML: frequency and clonal relationships. <i>Blood</i> , <b>2013</b> , 121, 489-98   | 2.2  | 154 |
| 74 | Identification of driver and passenger mutations of FLT3 by high-throughput DNA sequence analysis and functional assessment of candidate alleles. <i>Cancer Cell</i> , <b>2007</b> , 12, 501-13                                    | 24.3 | 154 |
| 73 | Targeted CML therapy: controlling drug resistance, seeking cure. <i>Current Opinion in Genetics and Development</i> , <b>2006</b> , 16, 92-9   | 4.9  | 153 |
| 72 | Targeting the BCR-ABL signaling pathway in therapy-resistant Philadelphia chromosome-positive leukemia. <i>Clinical Cancer Research</i> , <b>2011</b> , 17, 212-21   | 12.9 | 107 |
| 71 | Kinase pathway dependence in primary human leukemias determined by rapid inhibitor screening. <i>Cancer Research</i> , <b>2013</b> , 73, 285-96  | 10.1 | 106 |
| 70 | 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> , <b>2007</b> , 13, 6136-43 | 12.9 | 102 |
| 69 | 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> , <b>2006</b> , 107, 4250-6                            | 2.2  | 101 |
| 68 | Inhibition of the Bcr-Abl tyrosine kinase as a therapeutic strategy for CML. <i>Oncogene</i> , <b>2002</b> , 21, 8541-6  | 9.2  | 100 |

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|----|--|------|----|
| 67 | The TP53 Apoptotic Network Is a Primary Mediator of Resistance to BCL2 Inhibition in AML Cells. <i>Cancer Discovery</i> , <b>2019</b> , 9, 910-925   | 24.4 | 98 |
| 66 | 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> , <b>2010</b> , 115, 315-25  | 2.2  | 98 |
| 65 | High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. <i>Blood</i> , <b>2009</b> , 113, 1749-55  | 2.2  | 97 |
| 64 | Age-related mutations and chronic myelomonocytic leukemia. <i>Leukemia</i> , <b>2016</b> , 30, 906-13  | 10.7 | 94 |
| 63 | 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> , <b>2009</b> , 106, 8695-700   | 11.5 | 88 |
| 62 | In vitro efficacy of combined treatment depends on the underlying mechanism of resistance in imatinib-resistant Bcr-Abl-positive cell lines. <i>Blood</i> , <b>2004</b> , 103, 208-15  | 2.2  | 88 |
| 61 | Combined Abl inhibitor therapy for minimizing drug resistance in chronic myeloid leukemia: Src/Abl inhibitors are compatible with imatinib. <i>Clinical Cancer Research</i> , <b>2005</b> , 11, 6987-93  | 12.9 | 88 |
| 60 | Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. <i>Cancer Research</i> , <b>2019</b> , 79, 4744-4753   | 10.1 | 87 |
| 59 | Perspectives on the development of imatinib and the future of cancer research. <i>Nature Medicine</i> , <b>2009</b> , 15, 1149-52  | 50.5 | 87 |
| 58 | AMN107: tightening the grip of imatinib. <i>Cancer Cell</i> , <b>2005</b> , 7, 117-9   | 24.3 | 87 |
| 57 | Blockade of JAK2-mediated extrinsic survival signals restores sensitivity of CML cells to ABL inhibitors. <i>Leukemia</i> , <b>2012</b> , 26, 1140-3   | 10.7 | 85 |
| 56 | 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> , <b>2011</b> , 71, 3189-95   | 10.1 | 78 |
| 55 | High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. <i>Blood</i> , <b>2008</b> , 111, 4788-96  | 2.2  | 77 |
| 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. <i>Cancer Research</i> , <b>2005</b> , 65, 9436-44 | 10.1 | 76 |
| 53 | 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> , <b>2015</b> , 112, E5381-90   | 11.5 | 72 |
| 52 | Mutations of the BCR-ABL-kinase domain occur in a minority of patients with stable complete cytogenetic response to imatinib. <i>Leukemia</i> , <b>2007</b> , 21, 489-93   | 10.7 | 69 |
| 51 | MET receptor sequence variants R970C and T992I lack transforming capacity. <i>Cancer Research</i> , <b>2010</b> , 70, 6233-7   | 10.1 | 63 |
| 50 | Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. <i>Nature Communications</i> , <b>2019</b> , 10, 244  | 17.4 | 63 |

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|----|---|------|----|
| 49 | RNAi screening of the tyrosine kinome identifies therapeutic targets in acute myeloid leukemia. <i>Blood</i> , <b>2008</b> , 111, 2238-45   | 2.2  | 62 |
| 48 | 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> , <b>2017</b> , 114, E7554-E7563                    | 11.5 | 58 |
| 47 | 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> , <b>2005</b> , 19, 960-2  | 0.9  | 56 |
| 46 | Detection of ABL kinase domain mutations with denaturing high-performance liquid chromatography. <i>Leukemia</i> , <b>2004</b> , 18, 864-71   | 10.7 | 55 |
| 45 | Phosphoproteomic analysis of AML cell lines identifies leukemic oncogenes. <i>Leukemia Research</i> , <b>2006</b> , 30, 1097-104  | 2.7  | 51 |
| 44 | Zoledronate inhibits proliferation and induces apoptosis of imatinib-resistant chronic myeloid leukaemia cells. <i>Leukemia</i> , <b>2005</b> , 19, 1896-904  | 10.7 | 51 |
| 43 | Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. <i>Leukemia</i> , <b>2019</b> , 33, 1835-1850  | 10.7 | 50 |
| 42 | 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> , <b>2008</b> , 10, 177-80   | 5.1  | 50 |
| 41 | David A. Karnofsky Award lecture. Imatinib as a paradigm of targeted therapies. <i>Journal of Clinical Oncology</i> , <b>2003</b> , 21, 239s-245s   | 2.2  | 47 |
| 40 | 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. <i>British Journal of Haematology</i> , <b>2014</b> , 164, 223-32 | 4.5  | 46 |
| 39 | Molecularly targeted therapy: have the floodgates opened?. <i>Oncologist</i> , <b>2004</b> , 9, 357-60  | 5.7  | 43 |
| 38 | Imatinib mesylate in the treatment of chronic myeloid leukaemia. <i>Expert Opinion on Pharmacotherapy</i> , <b>2003</b> , 4, 963-71   | 4    | 40 |
| 37 | RNAi-induced down-regulation of FLT3 expression in AML cell lines increases sensitivity to MLN518. <i>Blood</i> , <b>2005</b> , 105, 2952-4   | 2.2  | 36 |
| 36 | e8a2 BCR-ABL: more frequent than other atypical BCR-ABL variants?. <i>Leukemia</i> , <b>2005</b> , 19, 681-4  | 10.7 | 34 |
| 35 | Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. <i>Leukemia</i> , <b>2008</b> , 22, 1184-90   | 10.7 | 33 |
| 34 | 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> , <b>2020</b> , 26, 1852-1858   | 50.5 | 32 |
| 33 | SRCircumventing imatinib resistance. <i>Cancer Cell</i> , <b>2004</b> , 6, 108-10   | 24.3 | 31 |
| 32 | Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. <i>Blood</i> , <b>2019</b> , 134, 867-879   | 2.2  | 29 |

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|----|---|------|----|
| 31 | A specific need for CRKL in p210BCR-ABL-induced transformation of mouse hematopoietic progenitors. <i>Cancer Research</i> , <b>2010</b> , 70, 7325-35   | 10.1 | 28 |
| 30 | An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. <i>Leukemia</i> , <b>2008</b> , 22, 2269-72   | 10.7 | 28 |
| 29 | YM155 potently kills acute lymphoblastic leukemia cells through activation of the DNA damage pathway. <i>Journal of Hematology and Oncology</i> , <b>2015</b> , 8, 39   | 22.4 | 25 |
| 28 | 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> , <b>2009</b> , 4, e7439  | 3.7  | 23 |
| 27 | Threshold levels of ABL tyrosine kinase inhibitors retained in chronic myeloid leukemia cells determine their commitment to apoptosis. <i>Cancer Research</i> , <b>2013</b> , 73, 3356-70                             | 10.1 | 22 |
| 26 | Antileukemic activity of lysophosphatidic acid acyltransferase-beta inhibitor CT32228 in chronic myelogenous leukemia sensitive and resistant to imatinib. <i>Clinical Cancer Research</i> , <b>2006</b> , 12, 6540-6 | 12.9 | 20 |
| 25 | Establishment of a murine model of aggressive systemic mastocytosis/mast cell leukemia. <i>Experimental Hematology</i> , <b>2006</b> , 34, 284-8  | 3.1  | 18 |
| 24 | KIT signaling governs differential sensitivity of mature and primitive CML progenitors to tyrosine kinase inhibitors. <i>Cancer Research</i> , <b>2013</b> , 73, 5775-86  | 10.1 | 17 |
| 23 | A single nucleotide polymorphism in the coding region of ABL and its effects on sensitivity to imatinib. <i>Leukemia</i> , <b>2005</b> , 19, 1859-62  | 10.7 | 17 |
| 22 | NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2-positive myeloproliferative neoplasm cells. <i>Signal Transduction and Targeted Therapy</i> , <b>2020</b> , 5, 5                             | 21   | 15 |
| 21 | 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> , <b>2003</b> , 4, 413-9            |      | 15 |
| 20 | High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. <i>Leukemia</i> , <b>2009</b> , 23, 406-9  | 10.7 | 14 |
| 19 | Catalytic domains of tyrosine kinases determine the phosphorylation sites within c-Cbl. <i>FEBS Letters</i> , <b>2004</b> , 577, 555-62   | 3.8  | 14 |
| 18 | c-CBL is not required for leukemia induction by Bcr-Abl in mice. <i>Oncogene</i> , <b>2003</b> , 22, 8852-60  | 9.2  | 13 |
| 17 | Myeloid lineage enhancers drive oncogene synergy in CEBPA/CSF3R mutant acute myeloid leukemia. <i>Nature Communications</i> , <b>2019</b> , 10, 5455  | 17.4 | 11 |
| 16 | The function of the pleckstrin homology domain in BCR-ABL-mediated leukemogenesis. <i>Leukemia</i> , <b>2010</b> , 24, 226-9  | 10.7 | 10 |
| 15 | A novel fusion in pediatric T-cell acute lymphoblastic leukemia. <i>Haematologica</i> , <b>2018</b> , 103, e87-e91  | 6.6  | 8  |
| 14 | Bruton's tyrosine kinase is not essential for Bcr-Abl-mediated transformation of lymphoid or myeloid cells. <i>Leukemia</i> , <b>2008</b> , 22, 1354-60   | 10.7 | 8  |

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|----|---|------|---|
| 13 | Coexistence of phosphotyrosine-dependent and -independent interactions between Cbl and Bcr-Abl. <i>Experimental Hematology</i> , <b>2004</b> , 32, 113-21   | 3.1  | 8 |
| 12 | 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> , <b>2021</b> , 40, 2697-2710  | 9.2  | 8 |
| 11 | Challenges and approaches to implementing master/basket trials in oncology. <i>Blood Advances</i> , <b>2019</b> , 3, 2237-2243  | 7.8  | 8 |
| 10 | Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. <i>Leukemia</i> , <b>2020</b> , 34, 2342-2353   | 10.7 | 7 |
| 9  | Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. <i>Leukemia</i> , <b>2016</b> , 30, 1418-21   | 10.7 | 7 |
| 8  | Clonal chromosomal abnormalities in CD34+/CD38- hematopoietic cells from cytogenetically normal chronic myeloid leukemia patients with a complete cytogenetic response to tyrosine kinase inhibitors. <i>Leukemia</i> , <b>2010</b> , 24, 1525-1528 | 10.7 | 6 |
| 7  | ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. <i>Leukemia</i> , <b>2020</b> , 34, 2798-2804   | 10.7 | 6 |
| 6  | Functional characterization of an activating TEK mutation in acute myeloid leukemia: a cellular context-dependent activating mutation. <i>Leukemia</i> , <b>2009</b> , 23, 1345-8   | 10.7 | 4 |
| 5  | Identification and prioritization of myeloid malignancy germline variants in a large cohort of adult AML patients. <i>Blood</i> , <b>2021</b> ,   | 2.2  | 3 |
| 4  | Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. <i>Cancer Research</i> , <b>2021</b> , 81, 4685-4695   | 10.1 | 2 |
| 3  | Bayesian multi-source regression and monocyte-associated gene expression predict BCL-2 inhibitor resistance in acute myeloid leukemia. <i>Npj Precision Oncology</i> , <b>2021</b> , 5, 71  | 9.8  | 0 |
| 2  | Peter C. Nowell (1928-2016). <i>Proceedings of the National Academy of Sciences of the United States of America</i> , <b>2017</b> , 114, 4569-4570  | 11.5 |   |
| 1  | BCR-ABL+ Chronic Myeloid Leukemia Arising in a Family With Inherited ANKRD26-Related Thrombocytopenia. <i>JCO Precision Oncology</i> , <b>2021</b> , 5,   | 3.6  |   |