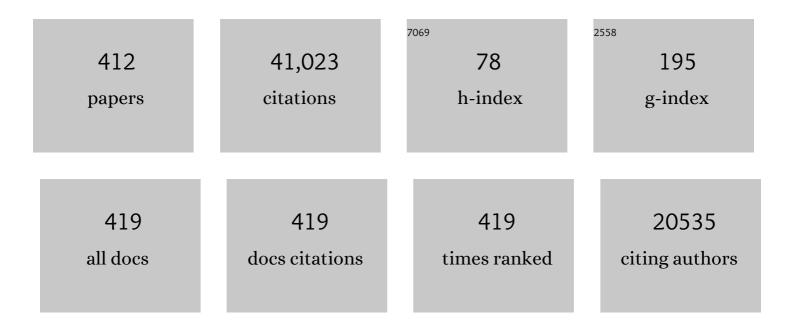
Timothy P Hughes

List of Publications by Year in descending order

Source: https://exaly.com/author-pdf/2830831/publications.pdf Version: 2024-02-01



#	Article	IF	CITATIONS
1	Imatinib Compared with Interferon and Low-Dose Cytarabine for Newly Diagnosed Chronic-Phase Chronic Myeloid Leukemia. New England Journal of Medicine, 2003, 348, 994-1004.	13.9	3,227
2	Five-Year Follow-up of Patients Receiving Imatinib for Chronic Myeloid Leukemia. New England Journal of Medicine, 2006, 355, 2408-2417.	13.9	3,212
3	European LeukemiaNet recommendations for the management of chronic myeloid leukemia: 2013. Blood, 2013, 122, 872-884.	0.6	1,743
4	Nilotinib versus Imatinib for Newly Diagnosed Chronic Myeloid Leukemia. New England Journal of Medicine, 2010, 362, 2251-2259.	13.9	1,497
5	Chronic Myeloid Leukemia: An Update of Concepts and Management Recommendations of European LeukemiaNet. Journal of Clinical Oncology, 2009, 27, 6041-6051.	0.8	1,188
6	Evolving concepts in the management of chronic myeloid leukemia: recommendations from an expert panel on behalf of the European LeukemiaNet. Blood, 2006, 108, 1809-1820.	0.6	1,184
7	Targetable Kinase-Activating Lesions in Ph-like Acute Lymphoblastic Leukemia. New England Journal of Medicine, 2014, 371, 1005-1015.	13.9	1,161
8	Monitoring CML patients responding to treatment with tyrosine kinase inhibitors: review and recommendations for harmonizing current methodology for detecting BCR-ABL transcripts and kinase domain mutations and for expressing results. Blood, 2006, 108, 28-37.	0.6	1,117
9	BCR–ABL1 lymphoblastic leukaemia is characterized by the deletion of Ikaros. Nature, 2008, 453, 110-114.	13.7	955
10	A Phase 2 Trial of Ponatinib in Philadelphia Chromosome–Positive Leukemias. New England Journal of Medicine, 2013, 369, 1783-1796.	13.9	944
11	Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. New England Journal of Medicine, 2017, 376, 917-927.	13.9	926
12	Six-year follow-up of patients receiving imatinib for the first-line treatment of chronic myeloid leukemia. Leukemia, 2009, 23, 1054-1061.	3.3	808
13	Dynamics of chronic myeloid leukaemia. Nature, 2005, 435, 1267-1270.	13.7	795
14	Lin28 promotes transformation and is associated with advanced human malignancies. Nature Genetics, 2009, 41, 843-848.	9.4	742
15	Long-term benefits and risks of frontline nilotinib vs imatinib for chronic myeloid leukemia in chronic phase: 5-year update of the randomized ENESTnd trial. Leukemia, 2016, 30, 1044-1054.	3.3	685
16	Safety and efficacy of imatinib cessation for CML patients with stable undetectable minimal residual disease: results from the TWISTER study. Blood, 2013, 122, 515-522.	0.6	641
17	High frequency of point mutations clustered within the adenosine triphosphate–binding region of BCR/ABL in patients with chronic myeloid leukemia or Ph-positive acute lymphoblastic leukemia who develop imatinib (STI571) resistance. Blood, 2002, 99, 3472-3475.	0.6	629
18	Dasatinib induces notable hematologic and cytogenetic responses in chronic-phase chronic myeloid leukemia after failure of imatinib therapy. Blood, 2007, 109, 2303-2309.	0.6	563

#	Article	IF	CITATIONS
19	Intermittent Target Inhibition With Dasatinib 100 mg Once Daily Preserves Efficacy and Improves Tolerability in Imatinib-Resistant and -Intolerant Chronic-Phase Chronic Myeloid Leukemia. Journal of Clinical Oncology, 2008, 26, 3204-3212.	0.8	458
20	Nilotinib versus imatinib for the treatment of patients with newly diagnosed chronic phase, Philadelphia chromosome-positive, chronic myeloid leukaemia: 24-month minimum follow-up of the phase 3 randomised ENESTnd trial. Lancet Oncology, The, 2011, 12, 841-851.	5.1	444
21	Long-term prognostic significance of early molecular response to imatinib in newly diagnosed chronic myeloid leukemia: an analysis from the International Randomized Study of Interferon and STI571 (IRIS). Blood, 2010, 116, 3758-3765.	0.6	440
22	OCT-1–mediated influx is a key determinant of the intracellular uptake of imatinib but not nilotinib (AMN107): reduced OCT-1 activity is the cause of low in vitro sensitivity to imatinib. Blood, 2006, 108, 697-704.	0.6	413
23	Nilotinib vs imatinib in patients with newly diagnosed Philadelphia chromosome-positive chronic myeloid leukemia in chronic phase: ENESTnd 3-year follow-up. Leukemia, 2012, 26, 2197-2203.	3.3	395
24	Ponatinib efficacy and safety in Philadelphia chromosome–positive leukemia: final 5-year results of the phase 2 PACE trial. Blood, 2018, 132, 393-404.	0.6	392
25	The allosteric inhibitor ABL001 enables dual targeting of BCR–ABL1. Nature, 2017, 543, 733-737.	13.7	389
26	Early molecular and cytogenetic response is predictive for long-term progression-free and overall survival in chronic myeloid leukemia (CML). Leukemia, 2012, 26, 2096-2102.	3.3	383
27	International Randomized Study of Interferon Vs STI571 (IRIS) 8-Year Follow up: Sustained Survival and Low Risk for Progression or Events in Patients with Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Treated with Imatinib Blood, 2009, 114, 1126-1126.	0.6	358
28	Sequential ABL kinase inhibitor therapy selects for compound drug-resistant BCR-ABL mutations with altered oncogenic potency. Journal of Clinical Investigation, 2007, 117, 2562-2569.	3.9	357
29	Dasatinib or high-dose imatinib for chronic-phase chronic myeloid leukemia after failure of first-line imatinib: a randomized phase 2 trial. Blood, 2007, 109, 5143-5150.	0.6	356
30	Desirable performance characteristics for BCR-ABL measurement on an international reporting scale to allow consistent interpretation of individual patient response and comparison of response rates between clinical trials. Blood, 2008, 112, 3330-3338.	0.6	350
31	Nilotinib is effective in patients with chronic myeloid leukemia in chronic phase after imatinib resistance or intolerance: 24-month follow-up results. Blood, 2011, 117, 1141-1145.	0.6	344
32	Dasatinib induces durable cytogenetic responses in patients with chronic myelogenous leukemia in chronic phase with resistance or intolerance to imatinib. Leukemia, 2008, 22, 1200-1206.	3.3	341
33	Most CML patients who have a suboptimal response to imatinib have low OCT-1 activity: higher doses of imatinib may overcome the negative impact of low OCT-1 activity. Blood, 2007, 110, 4064-4072.	0.6	309
34	Impact of Baseline <i>BCR-ABL</i> Mutations on Response to Nilotinib in Patients With Chronic Myeloid Leukemia in Chronic Phase. Journal of Clinical Oncology, 2009, 27, 4204-4210.	0.8	292
35	Nilotinib (formerly AMN107), a highly selective BCR-ABL tyrosine kinase inhibitor, is active in patients with imatinib-resistant or -intolerant accelerated-phase chronic myelogenous leukemia. Blood, 2008, 111, 1834-1839.	0.6	284
36	Moving treatment-free remission into mainstream clinical practice in CML. Blood, 2016, 128, 17-23.	0.6	278

#	Article	IF	CITATIONS
37	Dasatinib treatment of chronic-phase chronic myeloid leukemia: analysis of responses according to preexisting BCR-ABL mutations. Blood, 2009, 114, 4944-4953.	0.6	271
38	Macrophage colony-stimulating factor receptor c-fms is a novel target of imatinib. Blood, 2005, 105, 3127-3132.	0.6	266
39	Phase III, Randomized, Open-Label Study of Daily Imatinib Mesylate 400 mg Versus 800 mg in Patients With Newly Diagnosed, Previously Untreated Chronic Myeloid Leukemia in Chronic Phase Using Molecular End Points: Tyrosine Kinase Inhibitor Optimization and Selectivity Study. Journal of Clinical Oncology, 2010, 28, 424-430.	0.8	265
40	Asciminib in Chronic Myeloid Leukemia after ABL Kinase Inhibitor Failure. New England Journal of Medicine, 2019, 381, 2315-2326.	13.9	257
41	Patients with chronic myeloid leukemia who maintain a complete molecular response after stopping imatinib treatment have evidence of persistent leukemia by DNA PCR. Leukemia, 2010, 24, 1719-1724.	3.3	247
42	Early molecular response predicts outcomes in patients with chronic myeloid leukemia in chronic phase treated with frontline nilotinib or imatinib. Blood, 2014, 123, 1353-1360.	0.6	231
43	Ponatinib versus imatinib for newly diagnosed chronic myeloid leukaemia: an international, randomised, open-label, phase 3 trial. Lancet Oncology, The, 2016, 17, 612-621.	5.1	214
44	Nilotinib in imatinib-resistant or imatinib-intolerant patients with chronic myeloid leukemia in chronic phase: 48-month follow-up results of a phase II study. Leukemia, 2013, 27, 107-112.	3.3	212
45	Dasatinib or highâ€dose imatinib for chronicâ€phase chronic myeloid leukemia resistant to imatinib at a dose of 400 to 600 milligrams daily. Cancer, 2009, 115, 4136-4147.	2.0	195
46	Prognosis for patients with CML and >10% BCR-ABL1 after 3 months of imatinib depends on the rate of BCR-ABL1 decline. Blood, 2014, 124, 511-518.	0.6	182
47	Dasatinib in the Treatment of Chronic Myeloid Leukemia in Accelerated Phase After Imatinib Failure: The START A Trial. Journal of Clinical Oncology, 2009, 27, 3472-3479.	0.8	181
48	Selecting optimal second-line tyrosine kinase inhibitor therapy for chronic myeloid leukemia patients after imatinib failure: does the BCR-ABL mutation status really matter?. Blood, 2009, 114, 5426-5435.	0.6	178
49	Dasatinib Cellular Uptake and Efflux in Chronic Myeloid Leukemia Cells: Therapeutic Implications. Clinical Cancer Research, 2008, 14, 3881-3888.	3.2	169
50	Functional Activity of the OCT-1 Protein Is Predictive of Long-Term Outcome in Patients With Chronic-Phase Chronic Myeloid Leukemia Treated With Imatinib. Journal of Clinical Oncology, 2010, 28, 2761-2767.	0.8	167
51	Impact of early dose intensity on cytogenetic and molecular responses in chronic- phase CML patients receiving 600 mg/day of imatinib as initial therapy. Blood, 2008, 112, 3965-3973.	0.6	160
52	Minimal residual disease after allogeneic bone marrow transplantation for chronic myeloid leukaemia in first chronic phase: correlations with acute graftâ€versusâ€host disease and relapse. British Journal of Haematology, 1993, 84, 67-74.	1.2	159
53	Long-term outcomes with frontline nilotinib versus imatinib in newly diagnosed chronic myeloid leukemia in chronic phase: ENESTnd 10-year analysis. Leukemia, 2021, 35, 440-453.	3.3	159
54	Early molecular response and female sex strongly predict stable undetectable BCR-ABL1, the criteria for imatinib discontinuation in patients with CML. Blood, 2013, 121, 3818-3824.	0.6	153

ТІМОТНУ Р HUGHES

#	Article	IF	CITATIONS
55	Integrative genomic analysis reveals cancer-associated mutations at diagnosis of CML in patients with high-risk disease. Blood, 2018, 132, 948-961.	0.6	152
56	Rac2-MRC-cIII–generated ROS cause genomic instability in chronic myeloid leukemia stem cells and primitive progenitors. Blood, 2012, 119, 4253-4263.	0.6	147
57	A phase 3, open-label, randomized study of asciminib, a STAMP inhibitor, vs bosutinib in CML after 2 or more prior TKIs. Blood, 2021, 138, 2031-2041.	0.6	147
58	Molecular monitoring of BCR–ABL as a guide to clinical management in chronic myeloid leukaemia. Blood Reviews, 2006, 20, 29-41.	2.8	145
59	Long-term imatinib therapy promotes bone formation in CML patients. Blood, 2008, 111, 2538-2547.	0.6	144
60	CML patients with deep molecular responses to TKI have restored immune effectors and decreased PD-1 and immune suppressors. Blood, 2017, 129, 1166-1176.	0.6	143
61	Establishment of the first World Health Organization International Genetic Reference Panel for quantitation of BCR-ABL mRNA. Blood, 2010, 116, e111-e117.	0.6	141
62	In vitro sensitivity to imatinib-induced inhibition of ABL kinase activity is predictive of molecular response in patients with de novo CML. Blood, 2005, 106, 2520-2526.	0.6	135
63	Persistent activation of nuclear factor-?B in cultured rat hepatic stellate cells involves the induction of potentially novel rel-like factors and prolonged changes in the expression of I?B family proteins. Hepatology, 1999, 30, 761-769.	3.6	131
64	BCR-ABL Messenger RNA Levels Continue to Decline in Patients with Chronic Phase Chronic Myeloid Leukemia Treated with Imatinib for More Than 5 Years and Approximately Half of All First-Line Treated Patients Have Stable Undetectable BCR-ABL Using Strict Sensitivity Criteria. Clinical Cancer Research, 2007, 13, 7080-7085.	3.2	131
65	Dysregulation of bone remodeling by imatinib mesylate. Blood, 2010, 115, 766-774.	0.6	126
66	Treatment-Free Remission After Second-Line Nilotinib Treatment in Patients With Chronic Myeloid Leukemia in Chronic Phase. Annals of Internal Medicine, 2018, 168, 461.	2.0	105
67	The Src/ABL kinase inhibitor dasatinib (BMS-354825) inhibits function of normal human T-lymphocytes in vitro. Clinical Immunology, 2008, 127, 330-339.	1.4	104
68	Plasma exposure of imatinib and its correlation with clinical response in the Tyrosine Kinase Inhibitor Optimization and Selectivity Trial. Haematologica, 2012, 97, 731-738.	1.7	103
69	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. Leukemia, 2019, 33, 1835-1850.	3.3	97
70	Association between imatinib transporters and metabolizing enzymes genotype and response in newly diagnosed chronic myeloid leukemia patients receiving imatinib therapy. Haematologica, 2013, 98, 193-200.	1.7	96
71	Chronic myeloid leukemia: reminiscences and dreams. Haematologica, 2016, 101, 541-558.	1.7	92
72	Nilotinib is associated with a reduced incidence of BCR-ABL mutations vs imatinib in patients with newly diagnosed chronic myeloid leukemia in chronic phase. Blood, 2013, 121, 3703-3708.	0.6	91

#	Article	IF	CITATIONS
73	Initial Molecular Response at 3 Months May Predict Both Response and Event-Free Survival at 24 Months in Imatinib-Resistant or -Intolerant Patients With Philadelphia Chromosome–Positive Chronic Myeloid Leukemia in Chronic Phase Treated With Nilotinib. Journal of Clinical Oncology, 2012, 30, 4323-4329.	0.8	90
74	Monoclonal antibody targeting of IL-3 receptor α with CSL362 effectively depletes CML progenitor and stem cells. Blood, 2014, 123, 1218-1228.	0.6	89
75	Compound mutations in BCR-ABL1 are not major drivers of primary or secondary resistance to ponatinib in CP-CML patients. Blood, 2016, 127, 703-712.	0.6	87
76	Sensitive Detection of <i>BCR-ABL1</i> Mutations in Patients With Chronic Myeloid Leukemia After Imatinib Resistance Is Predictive of Outcome During Subsequent Therapy. Journal of Clinical Oncology, 2011, 29, 4250-4259.	0.8	86
77	Population pharmacokinetic and exposure-response analysis of nilotinib in patients with newly diagnosed Ph+ chronic myeloid leukemia in chronic phase. European Journal of Clinical Pharmacology, 2012, 68, 723-733.	0.8	86
78	Front-Line and Salvage Therapies With Tyrosine Kinase Inhibitors and Other Treatments in Chronic Myeloid Leukemia. Journal of Clinical Oncology, 2011, 29, 524-531.	0.8	84
79	Deep molecular responses achieved in patients with CML-CP who are switched to nilotinib after long-term imatinib. Blood, 2014, 124, 729-736.	0.6	84
80	Clinical resistance to imatinib: mechanisms and implications. Hematology/Oncology Clinics of North America, 2004, 18, 641-656.	0.9	80
81	Signalling by the \hat{I}^2 c family of cytokines. Cytokine and Growth Factor Reviews, 2013, 24, 189-201.	3.2	80
82	International Randomized Study of Interferon Versus STI571 (IRIS) 7-Year Follow-up: Sustained Survival, Low Rate of Transformation and Increased Rate of Major Molecular Response (MMR) in Patients (pts) with Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase (CMLCP) Treated with Imatinib (IM). Blood, 2008, 112, 186-186.	0.6	80
83	Overall survival with ponatinib versus allogeneic stem cell transplantation in Philadelphia chromosomeâ€positive leukemias with the T315I mutation. Cancer, 2017, 123, 2875-2880.	2.0	79
84	TIDEL-II: first-line use of imatinib in CML with early switch to nilotinib for failure to achieve time-dependent molecular targets. Blood, 2015, 125, 915-923.	0.6	77
85	Interaction of the Efflux Transporters ABCB1 and ABCG2 With Imatinib, Nilotinib, and Dasatinib. Clinical Pharmacology and Therapeutics, 2014, 95, 294-306.	2.3	75
86	Imatinib as a potential antiresorptive therapy for bone disease. Blood, 2006, 107, 4334-4337.	0.6	74
87	Dasatinib suppresses in vitro natural killer cell cytotoxicity. Blood, 2008, 111, 4415-4416.	0.6	73
88	Poor response to second-line kinase inhibitors in chronic myeloid leukemia patients with multiple low-level mutations, irrespective of their resistance profile. Blood, 2012, 119, 2234-2238.	0.6	69
89	Long-term treatment-free remission of chronic myeloid leukemia with falling levels of residual leukemic cells. Leukemia, 2018, 32, 2572-2579.	3.3	66
90	Dynamics of chronic myeloid leukemia response to long-term targeted therapy reveal treatment effects on leukemic stem cells. Blood, 2011, 118, 1622-1631.	0.6	65

#	Article	IF	CITATIONS
91	Chronic Myeloid Leukemia CD34+ cells have reduced uptake of imatinib due to low OCT-1 Activity. Leukemia, 2010, 24, 765-770.	3.3	64
92	The GM-CSF receptor family: Mechanism of activation and implications for disease. Growth Factors, 2012, 30, 63-75.	0.5	64
93	Measurement of In Vivo BCR-ABL Kinase Inhibition to Monitor Imatinib-Induced Target Blockade and Predict Response in Chronic Myeloid Leukemia. Journal of Clinical Oncology, 2007, 25, 4445-4451.	0.8	62
94	Incidence, outcomes, and risk factors of pleural effusion in patients receiving dasatinib therapy for Philadelphia chromosome-positive leukemia. Haematologica, 2019, 104, 93-101.	1.7	62
95	Factors affecting the outcome of allogeneic bone marrow transplantation for adult patients with refractory or relapsed acute leukaemia. British Journal of Haematology, 1999, 107, 409-418.	1.2	59
96	Dasatinib inhibits recombinant viral antigen-specific murine CD4+ and CD8+ T-cell responses and NK-cell cytolytic activity in vitro and in vivo. Experimental Hematology, 2009, 37, 256-265.	0.2	58
97	BCR-ABL1 mutation development during first-line treatment with dasatinib or imatinib for chronic myeloid leukemia in chronic phase. Leukemia, 2015, 29, 1832-1838.	3.3	58
98	The impact of multiple low-level BCR-ABL1 mutations on response to ponatinib. Blood, 2016, 127, 1870-1880.	0.6	58
99	Tyrosine kinase inhibitor resistance in chronic myeloid leukemia cell lines: investigating resistance pathways. Leukemia and Lymphoma, 2011, 52, 2139-2147.	0.6	57
100	A phase 2 study of MK-0457 in patients with BCR-ABL T315I mutant chronic myelogenous leukemia and philadelphia chromosome-positive acute lymphoblastic leukemia. Blood Cancer Journal, 2014, 4, e238-e238.	2.8	57
101	Monitoring disease response to tyrosine kinase inhibitor therapy in CML. Hematology American Society of Hematology Education Program, 2009, 2009, 477-487.	0.9	55
102	The clinical significance of ABCB1 overexpression in predicting outcome of CML patients undergoing first-line imatinib treatment. Leukemia, 2017, 31, 75-82.	3.3	54
103	Chronic phase chronic myeloid leukemia patients with low OCT-1 activity randomized to high-dose imatinib achieve better responses and have lower failure rates than those randomized to standard-dose imatinib. Haematologica, 2012, 97, 907-914.	1.7	53
104	Therapeutic concentrations of dasatinib inhibit in vitro osteoclastogenesis. Leukemia, 2009, 23, 994-997.	3.3	52
105	High prevalence of relapse in children with Philadelphia-like acute lymphoblastic leukemia despite risk-adapted treatment. Haematologica, 2017, 102, e490-e493.	1.7	52
106	Successful treatmentâ€free remission in chronic myeloid leukaemia and its association with reduced immune suppressors and increased natural killer cells. British Journal of Haematology, 2020, 191, 433-441.	1.2	52
107	Plasma Adiponectin Levels Are Markedly Elevated in Imatinib-Treated Chronic Myeloid Leukemia (CML) Patients: A Mechanism for Improved Insulin Sensitivity in Type 2 Diabetic CML Patients?. Journal of Clinical Endocrinology and Metabolism, 2010, 95, 3763-3767.	1.8	51

108 Detection of <i>BCR-ABL</i>Mutations and Resistance to Imatinib Mesylate., 2006, 125, 93-106.

50

#	Article	IF	CITATIONS
109	Blocking cytokine signaling along with intense Bcr-Abl kinase inhibition induces apoptosis in primary CML progenitors. Leukemia, 2010, 24, 771-778.	3.3	50
110	BCR-ABL1 doubling times more reliably assess the dynamics of CML relapse compared with the BCR-ABL1 fold rise: implications for monitoring and management. Blood, 2012, 119, 4264-4271.	0.6	49
111	OCT1 and imatinib transport in CML: is it clinically relevant?. Leukemia, 2015, 29, 1960-1969.	3.3	49
112	How I determine if and when to recommend stopping tyrosine kinase inhibitor treatment for chronic myeloid leukaemia. British Journal of Haematology, 2014, 166, 3-11.	1.2	48
113	Early BCR-ABL1 kinetics are predictive of subsequent achievement of treatment-free remission in chronic myeloid leukemia. Blood, 2021, 137, 1196-1207.	0.6	48
114	Long-term response to imatinib is not affected by the initial dose in patients with Philadelphia chromosome-positive chronic myeloid leukemia in chronic phase: final update from the Tyrosine Kinase Inhibitor Optimization and Selectivity (TOPS) study. International Journal of Hematology, 2014, 99, 616-624.	0.7	47
115	Dasatinib treatment for Philadelphia chromosomeâ€positive leukemias. Cancer, 2009, 115, 1381-1394.	2.0	46
116	SHP-1 expression accounts for resistance to imatinib treatment in Philadelphia chromosome–positive cells derived from patients with chronic myeloid leukemia. Blood, 2011, 118, 3634-3644.	0.6	46
117	BCR–ABL Transcript Dynamics Support the Hypothesis That Leukemic Stem Cells Are Reduced during Imatinib Treatment. Clinical Cancer Research, 2011, 17, 6812-6821.	3.2	46
118	Establishment and Validation of Analytical Reference Panels for the Standardization of Quantitative BCR-ABL1 Measurements on the International Scale. Clinical Chemistry, 2013, 59, 938-948.	1.5	46
119	Which TKI? An embarrassment of riches for chronic myeloid leukemia patients. Hematology American Society of Hematology Education Program, 2013, 2013, 168-175.	0.9	45
120	HLAâ€identical sibling donor bone marrow transplantation for chronic myeloid leukaemia in first chronic phase: influence of GVHD prophylaxis on outcome. British Journal of Haematology, 1992, 81, 383-390.	1.2	44
121	Diagnosis and Monitoring of Chronic Myeloid Leukemia by Qualitative and Quantitative RT-PCR. , 2006, 125, 69-92.		43
122	Current Issues in Chronic Myeloid Leukemia: Monitoring, Resistance, and Functional Cure. Journal of the National Comprehensive Cancer Network: JNCCN, 2012, 10, S-1-S-13.	2.3	43
123	Long-Term Follow-up of Ponatinib Efficacy and Safety in the Phase 2 PACE Trial. Blood, 2014, 124, 3135-3135.	0.6	43
124	Dasatinib inhibits the secretion of TNF-α following TLR stimulation in vitro and in vivo. Experimental Hematology, 2009, 37, 1435-1444.	0.2	42
125	Imatinib mesylate causes growth plate closure in vivo. Leukemia, 2009, 23, 2155-2159.	3.3	42
126	Safety and efficacy of switching to nilotinib 400 mg twice daily for patients with chronic myeloid leukemia in chronic phase with suboptimal response or failure on front-line imatinib or nilotinib 300 mg twice daily. Haematologica, 2014, 99, 1204-1211.	1.7	42

#	Article	IF	CITATIONS
127	Efficacy and Safety of Nilotinib (NIL) vs Imatinib (IM) in Patients (pts) With Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase (CML-CP): Long-Term Follow-Up (f/u) of ENESTnd. Blood, 2014, 124, 4541-4541.	0.6	42
128	Apoptosis regulatory gene NEDD2 maps to human chromosome segment 7q34?35, a region frequently affected in haematological neoplasms. Human Genetics, 1995, 95, 641-4.	1.8	41
129	Sustained deep molecular responses in patients switched to nilotinib due to persistent BCR-ABL1 on imatinib: final ENESTcmr randomized trial results. Leukemia, 2017, 31, 2529-2531.	3.3	41
130	TARGET: a survey of realâ€world management of chronic myeloid leukaemia across 33 countries. British Journal of Haematology, 2020, 190, 869-876.	1.2	40
131	Potential mechanisms of disease progression and management of advanced-phase chronic myeloid leukemia. Leukemia and Lymphoma, 2014, 55, 1451-1462.	0.6	39
132	Accumulation of JAK activation loop phosphorylation is linked to type I JAK inhibitor withdrawal syndrome in myelofibrosis. Science Advances, 2018, 4, eaat3834.	4.7	39
133	Lineage of measurable residual disease in patients with chronic myeloid leukemia in treatment-free remission. Leukemia, 2020, 34, 1052-1061.	3.3	39
134	A Phase I/II study of nilotinib in Japanese patients with imatinib-resistant or -intolerant Ph+ CML or relapsed/refractory Ph+ ALL. International Journal of Hematology, 2009, 89, 679-688.	0.7	38
135	A pilot study of continuous imatinib vs pulsed imatinib with or without G-CSF in CML patients who have achieved a complete cytogenetic response. Leukemia, 2009, 23, 1199-1201.	3.3	38
136	Guidelines for whole genome bisulphite sequencing of intact and FFPET DNA on the Illumina HiSeq X Ten. Epigenetics and Chromatin, 2018, 11, 24.	1.8	38
137	Imatinib inhibits the functional capacity of cultured human monocytes. Immunology and Cell Biology, 2005, 83, 48-56.	1.0	37
138	OCT-1 activity measurement provides a superior imatinib response predictor than screening for single-nucleotide polymorphisms of OCT-1. Leukemia, 2010, 24, 1962-1965.	3.3	37
139	TGF-α and IL-6 plasma levels selectively identify CML patients who fail to achieve an early molecular response or progress in the first year of therapy. Leukemia, 2016, 30, 1263-1272.	3.3	37
140	The new allosteric inhibitor asciminib is susceptible to resistance mediated by ABCB1 and ABCG2 overexpression <i>in vitro</i> . Oncotarget, 2018, 9, 13423-13437.	0.8	37
141	Twentyâ€year followâ€up of newborn screening for patients with muscular dystrophy. Muscle and Nerve, 2016, 53, 570-578.	1.0	36
142	Reduced CD62L Expression on T Cells and Increased Soluble CD62L Levels Predict Molecular Response to Tyrosine Kinase Inhibitor Therapy in Early Chronic-Phase Chronic Myelogenous Leukemia. Journal of Clinical Oncology, 2017, 35, 175-184.	0.8	36
143	Dasatinib-associated major molecular responses in patients with chronic myeloid leukemia in chronic phase following imatinib failure: response dynamics and predictive value. Leukemia, 2009, 23, 1628-1633.	3.3	35
144	A Low Concentration of ABL001 Potentiates In Vitro TKI-Induced Bcr-Abl Kinase Inhibition in CML Cells. Blood. 2016, 128, 1121-1121.	0.6	35

#	Article	IF	CITATIONS
145	A Pivotal Phase 2 Trial of Ponatinib in Patients with Chronic Myeloid Leukemia (CML) and Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia (Ph+ALL) Resistant or Intolerant to Dasatinib or Nilotinib, or with the T315I BCR-ABL Mutation: 12-Month Follow-up of the PACE Trial. Blood, 2012, 120, 163-163.	0.6	34
146	ENESTnd Update: Nilotinib (NIL) Vs Imatinib (IM) In Patients (pts) With Newly Diagnosed Chronic Myeloid Leukemia In Chronic Phase (CML-CP) and The Impact Of Early Molecular Response (EMR) and Sokal Risk At Diagnosis On Long-Term Outcomes. Blood, 2013, 122, 92-92.	0.6	34
147	Do we have to kill the last CML cell?. Leukemia, 2011, 25, 193-200.	3.3	33
148	Treatment-free remission in patients with chronic myeloid leukaemia. Nature Reviews Clinical Oncology, 2020, 17, 493-503.	12.5	33
149	ABL Kinase Inhibitor Therapy for CML: Baseline Assessments and Response Monitoring. Hematology American Society of Hematology Education Program, 2006, 2006, 211-218.	0.9	32
150	Many BCR-ABL1 compound mutations reported in chronic myeloid leukemia patients may actually be artifacts due to PCR-mediated recombination. Blood, 2014, 124, 153-155.	0.6	31
151	<i>BCR-ABL1</i> genomic DNA PCR response kinetics during first-line imatinib treatment of chronic myeloid leukemia. Haematologica, 2018, 103, 2026-2032.	1.7	31
152	Dual transcription of b2a2 and b3a2 BCR-ABL transcripts in chronic myeloid leukaemia is confined to patients with a linked polymorphism within the BCR gene. British Journal of Haematology, 2002, 117, 875-877.	1.2	30
153	Role of Allogeneic Stem Cell Transplantation for Adult Chronic Myeloid Leukemia in the Imatinib Era. Biology of Blood and Marrow Transplantation, 2006, 12, 795-807.	2.0	30
154	International standardisation of quantitative real-time RT-PCR for BCR-ABL. Leukemia Research, 2008, 32, 505-506.	0.4	30
155	Sustained inhibition of STAT5, but not JAK2, is essential for TKI-induced cell death in chronic myeloid leukemia. Leukemia, 2015, 29, 76-85.	3.3	30
156	Nilotinib-mediated inhibition of ABCB1 increases intracellular concentration of dasatinib in CML cells: implications for combination TKI therapy. Leukemia, 2010, 24, 658-660.	3.3	28
157	ABCB1 Overexpression Is a Key Initiator of Resistance to Tyrosine Kinase Inhibitors in CML Cell Lines. PLoS ONE, 2016, 11, e0161470.	1.1	28
158	A dual role for the N-terminal domain of the IL-3 receptor in cell signalling. Nature Communications, 2018, 9, 386.	5.8	28
159	Pre-B acute lymphoblastic leukaemia recurrent fusion, EP300-ZNF384, is associated with a distinct gene expression. British Journal of Cancer, 2018, 118, 1000-1004.	2.9	28
160	Role of the β Common (βc) Family of Cytokines in Health and Disease. Cold Spring Harbor Perspectives in Biology, 2018, 10, a028514.	2.3	28
161	Update On Imatinib-Resistant Chronic Myeloid Leukemia Patients in Chronic Phase (CML-CP) On Nilotinib Therapy at 24 Months: Clinical Response, Safety, and Long-Term Outcomes Blood, 2009, 114, 1129-1129.	0.6	28
162	KIR2DL5B genotype predicts outcomes in CML patients treated with response-directed sequential imatinib/nilotinib strategy. Blood, 2015, 126, 2720-2723.	0.6	27

#	Article	IF	CITATIONS
163	Gene expression signature that predicts early molecular response failure in chronic-phase CML patients on frontline imatinib. Blood Advances, 2019, 3, 1610-1621.	2.5	27
164	Aberrant RAG-mediated recombination contributes to multiple structural rearrangements in lymphoid blast crisis of chronic myeloid leukemia. Leukemia, 2020, 34, 2051-2063.	3.3	27
165	Clarithromycin enhances dasatinib-induced cell death in chronic myeloid leukemia cells, by inhibition of late stage autophagy. Leukemia and Lymphoma, 2013, 54, 198-201.	0.6	26
166	Degree of kinase inhibition achievedin vitroby imatinib and nilotinib is decreased by high levels of ABCB1 but not ABCG2. Leukemia and Lymphoma, 2013, 54, 569-578.	0.6	26
167	Ponatinib is not transported by ABCB1, ABCG2 or OCT-1 in CML cells. Leukemia, 2015, 29, 1792-1794.	3.3	26
168	Therapeutic Targeting of BCR-ABL: Prognostic Markers of Response and Resistance Mechanism in Chronic Myeloid Leukaemia. Critical Reviews in Oncogenesis, 2012, 17, 17-30.	0.2	26
169	Nilotinib inhibits the Srcâ€family kinase LCK and Tâ€cell function <i>in vitro</i> . Journal of Cellular and Molecular Medicine, 2009, 13, 599-601.	1.6	25
170	Concurrent use of proton pump inhibitors or H2 blockers did not adversely affect nilotinib efficacy in patients with chronic myeloid leukemia. Cancer Chemotherapy and Pharmacology, 2012, 70, 345-350.	1.1	25
171	A Phase III, Randomized, Open-Label Study of 400 Mg Versus 800 Mg of Imatinib Mesylate (IM) in Patients (pts) with Newly Diagnosed, Previously Untreated Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Using Molecular Endpoints: 1-Year Results of TOPS (Tyrosine Kinase Inhibitor Optimization) Tj ETQq1	1 0.784314	Frgat /Overle
172	Mutational analysis in chronic myeloid leukemia: when and what to do?. Current Opinion in Hematology, 2011, 18, 111-116.	1.2	24
173	Prospective Histomorphometric and DXA Evaluation of Bone Remodeling in Imatinib-Treated CML Patients: Evidence for Site-Specific Skeletal Effects. Journal of Clinical Endocrinology and Metabolism, 2013, 98, 67-76.	1.8	24
174	ENESTnd Update: Continued Superiority of Nilotinib Versus Imatinib In Patients with Newly Diagnosed Chronic Myeloid Leukemia In Chronic Phase (CML-CP). Blood, 2010, 116, 207-207.	0.6	24
175	Optimizing Outcomes for Patients With Advanced Disease in Chronic Myelogenous Leukemia. Seminars in Oncology, 2008, 35, S1-S17.	0.8	23
176	Distribution of genomic breakpoints in chronic myeloid leukemia: analysis of 308 patients. Leukemia, 2013, 27, 2105-2107.	3.3	23
177	Prediction of outcomes in patients with Ph+ chronic myeloid leukemia in chronic phase treated with nilotinib after imatinib resistance/intolerance. Leukemia, 2013, 27, 907-913.	3.3	23
178	A DNA Real-Time Quantitative PCR Method Suitable for Routine Monitoring of Low Levels of MinimalÂResidual Disease in Chronic Myeloid Leukemia. Journal of Molecular Diagnostics, 2015, 17, 185-192.	1.2	23
179	Relapse of BCR-ABL1-like ALL mediated by the ABL1 kinase domain mutation T315I following initial response to dasatinib treatment. Leukemia, 2015, 29, 230-232.	3.3	23
180	Differential expression of MUC4, GPR110 and IL2RA defines two groups of CRLF2-rearranged acute lymphoblastic leukemia patients with distinct secondary lesions. Cancer Letters, 2017, 408, 92-101.	3.2	23

#	Article	IF	CITATIONS
181	Molecular monitoring in CML: how deep? How often? How should it influence therapy?. Hematology American Society of Hematology Education Program, 2018, 2018, 168-176.	0.9	22
182	ABL001, a Potent, Allosteric Inhibitor of BCR-ABL, Exhibits Safety and Promising Single- Agent Activity in a Phase I Study of Patients with CML with Failure of Prior TKI Therapy. Blood, 2015, 126, 138-138.	0.6	22
183	BCR-ABL1-positive CML and BCR-ABL1-negative chronic myeloproliferative disorders: some common and contrasting features. Leukemia, 2008, 22, 1975-1989.	3.3	21
184	ABCC6 plays a significant role in the transport of nilotinib and dasatinib, and contributes to TKI resistance in vitro, in both cell lines and primary patient mononuclear cells. PLoS ONE, 2018, 13, e0192180.	1.1	21
185	Enestnd 4-Year (y) Update: Continued Superiority of Nilotinib Vs Imatinib in Patients (pts) with Newly Diagnosed Philadelphia Chromosome–Positive (Ph+) Chronic Myeloid Leukemia in Chronic Phase (CML-CP). Blood, 2012, 120, 1676-1676.	0.6	21
186	Development of asciminib, a novel allosteric inhibitor of BCR-ABL1. Critical Reviews in Oncology/Hematology, 2022, 171, 103580.	2.0	21
187	The poor response to imatinib observed in CML patients with low OCT-1 activity is not attributable to lower uptake of imatinib into their CD34+ cells. Blood, 2010, 116, 2776-2778.	0.6	20
188	Suboptimal responses in chronic myeloid leukemia. Cancer, 2012, 118, 1181-1191.	2.0	20
189	Modeling the safe minimum frequency of molecular monitoring for CML patients attempting treatment-free remission. Blood, 2019, 134, 85-89.	0.6	20
190	Efficacy and Safety Results from ASCEMBL, a Multicenter, Open-Label, Phase 3 Study of Asciminib, a First-in-Class STAMP Inhibitor, vs Bosutinib (BOS) in Patients (Pts) with Chronic Myeloid Leukemia in		

#	Article	IF	CITATIONS
199	Dynamics of chronic myeloid leukemia response to dasatinib, nilotinib, and high-dose imatinib. Haematologica, 2014, 99, 1701-1709.	1.7	18
200	Long-term treatment-free remission in patients with chronic myeloid leukemia after second-line nilotinib: ENESTop 5-year update. Leukemia, 2021, 35, 1631-1642.	3.3	18
201	Continuing Reduction in Level of Residual Disease after 4 Years in Patients with CML in Chronic Phase Responding to First-Line Imatinib (IM) in the IRIS Study Blood, 2005, 106, 163-163.	0.6	18
202	ASXL1 and BIM germ line variants predict response and identify CML patients with the greatest risk of imatinib failure. Blood Advances, 2017, 1, 1369-1381.	2.5	17
203	24 Months Update of the TOPS Study: a Phase III, Randomized, Open-Label Study of 400mg/d (SD-IM) Versus 800mg/d (HD-IM) of Imatinib Mesylate (IM) in Patients (Pts) with Newly Diagnosed, Previously Untreated Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Blood, 2009, 114, 337-337.	0.6	17
204	Efficacy and Safety of Nilotinib In Chronic Phase (CP) Chronic Myeloid Leukemia (CML) Patients (Pts) with Type 2 Diabetes In the ENESTnd Trial Blood, 2010, 116, 3430-3430.	0.6	17
205	Outcome of Patients with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Based On Early Molecular Response and Factors Associated with Early Response: 4-Year Follow-up Data From Enestnd (Evaluating Nilotinib Efficacy and Safety in Clinical Trials Newly Diagnosed Patients). Blood, 2012, 120, 167-167.	0.6	17
206	Current and emerging tests for the laboratory monitoring of chronic myeloid leukaemia and related disorders. Pathology, 2008, 40, 231-246.	0.3	16
207	Rapid initial decline in BCR-ABL1 is associated with superior responses to second-line nilotinib in patients with chronic-phase chronic myeloid leukemia. BMC Cancer, 2013, 13, 173.	1.1	16
208	CYP2C8 Genotype Significantly Alters Imatinib Metabolism in Chronic Myeloid Leukaemia Patients. Clinical Pharmacokinetics, 2017, 56, 977-985.	1.6	16
209	Classification of Patients With Chronic Myeloid Leukemia on Basis of BCR-ABL Transcript Level at 3 Months Fails to Identify Patients With Low Organic Cation Transporter-1 Activity Destined to Have Poor Imatinib Response. Journal of Clinical Oncology, 2012, 30, 1144-1145.	0.8	15
210	Reduction of BCR-ABL Transcript Levels at 6, 12, and 18 Months (mo) Correlates with Long-Term Outcomes on Imatinib (IM) at 72 Mo: An Analysis from the International Randomized Study of Interferon versus STI571 (IRIS) in Patients (pts) with Chronic Phase Chronic Myeloid Leukemia (CML-CP). Blood, 2008, 112, 334-334.	0.6	15
211	Short-term intense Bcr–Abl kinase inhibition with nilotinib is adequate to trigger cell death in BCR-ABL+ cells. Leukemia, 2009, 23, 1205-1206.	3.3	14
212	Drug-interaction studies evaluating T-cell proliferation reveal distinct activity of dasatinib and imatinib in combination with cyclosporine A. Experimental Hematology, 2012, 40, 612-621.e6.	0.2	14
213	Nilotinib doseâ€optimization in newly diagnosed chronic myeloid leukaemia in chronic phase: final results from <scp>ENEST</scp> xtnd. British Journal of Haematology, 2017, 179, 219-228.	1.2	14
214	Validation of the International Scale for Measurement of BCR-ABL by RQ-PCR Based on Deriving Laboratory-Specific Conversion Factors Blood, 2007, 110, 1013-1013.	0.6	14
215	Reduced Activity of the OCT-1 Protein in Primitive CML Cells: A Likely Determinant of Stem Cell Resistance in Imatinib Treated CML Patients. Blood, 2008, 112, 196-196.	0.6	14
216	Upfront Imatinib Therapy in CML Patients with Rapid Switching to Nilotinib for Failure to Achieve Molecular Targets or Intolerance Achieves High Overall Rates of Molecular Response and a Low Risk of Progression - An Update of the TIDEL-II Trial. Blood, 2011, 118, 451-451.	0.6	14

#	Article	IF	CITATIONS
217	Asciminib: a new therapeutic option in chronic-phase CML with treatment failure. Blood, 2022, 139, 3474-3479.	0.6	14
218	Managing imatinib resistance in chronic myeloid leukaemia. Current Opinion in Hematology, 2010, 17, 97-103.	1.2	13
219	Dasatinib alters the metastatic phenotype of B16-OVA melanoma in vivo. Cancer Biology and Therapy, 2010, 10, 715-727.	1.5	13
220	OCT-1 function varies with cell lineage but is not influenced by BCR-ABL. Haematologica, 2011, 96, 213-220.	1.7	13
221	Low GFI1 expression in white blood cells of CP–CML patients at diagnosis is strongly associated with subsequent blastic transformation. Leukemia, 2013, 27, 1427-1430.	3.3	13
222	Long Term Follow up of Patients with CML in Chronic Phase Treated with First-Line Imatinib Suggests That Earlier Achievement of a Major Molecular Response Leads to Greater Stability of Response Blood, 2008, 112, 2113-2113.	0.6	13
223	CML Patients with Low OCT-1 Activity Achieve Better Molecular Responses on High Dose Imatinib Than on Standard Dose. Those with High OCT-1 Activity Have Excellent Responses on Either Dose: A TOPS Correlative Study. Blood, 2008, 112, 3187-3187.	0.6	13
224	Clinical impact of dose modification and dose intensity on response to ponatinib (PON) in patients (pts) with Philadelphia chromosome-positive (Ph+) leukemias Journal of Clinical Oncology, 2014, 32, 7084-7084.	0.8	13
225	Efficacy and Safety Results from Ascembl, a Multicenter, Open-Label, Phase 3 Study of Asciminib, a First-in-Class STAMP Inhibitor, Vs Bosutinib in Patients with Chronic Myeloid Leukemia in Chronic Phase after ≥2 Prior Tyrosine Kinase Inhibitors: Update after 48 Weeks. Blood, 2021, 138, 310-310.	0.6	13
226	Reverse Transcription with Random Pentadecamer Primers Improves the Detection Limit of a Quantitative PCR Assay for BCR-ABL Transcripts in Chronic Myeloid Leukemia: Implications for Defining Sensitivity in Minimal Residual Disease. Clinical Chemistry, 2008, 54, 1568-1571.	1.5	12
227	Nilotinib does not significantly reduce imatinib OCT-1 activity in either cell lines or primary CML cells. Leukemia, 2010, 24, 855-857.	3.3	12
228	Low incidence of peripheral arterial disease in patients receiving dasatinib in clinical trials. Leukemia, 2016, 30, 1593-1596.	3.3	12
229	Increased peroxisome proliferator-activated receptor Î ³ activity reduces imatinib uptake and efficacy in chronic myeloid leukemia mononuclear cells. Haematologica, 2017, 102, 843-853.	1.7	12
230	Cardiac Safety Profile of Imatinib and Nilotinib In Patients (pts) with Newly Diagnosed Chronic Myeloid Leukemia In Chronic Phase (CML-CP): Results From ENESTnd. Blood, 2010, 116, 2291-2291.	0.6	12
231	Incidence of Hyperglycemia by 3 Years in Patients (Pts) with Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Treated with Nilotinib (NIL) or Imatinib (IM) in ENESTnd. Blood, 2012, 120, 1686-1686.	0.6	12
232	Treatment-free remission (TFR) following nilotinib (NIL) in patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP): ENESTfreedom, ENESTop, ENESTgoal, and ENESTpath Journal of Clinical Oncology, 2014, 32, TPS7124-TPS7124.	0.8	12
233	Modelling ponatinib resistance in tyrosine kinase inhibitor-naÃ⁻ve and dasatinib resistant <i>BCR-ABL1</i> + cell lines. Oncotarget, 2018, 9, 34735-34747.	0.8	12
234	Dasatinib targets chronic myeloid leukemia-CD34+ progenitors as effectively as it targets mature cells. Haematologica, 2013, 98, 896-900.	1.7	11

#	Article	IF	CITATIONS
235	Modelling Predictors of Molecular Response to Frontline Imatinib for Patients with Chronic Myeloid Leukaemia. PLoS ONE, 2017, 12, e0168947.	1.1	11
236	Efficacy and safety of nilotinib 300 mg twice daily in patients with chronic myeloid leukemia in chronic phase who are intolerant to prior tyrosine kinase inhibitors: Results from the Phase IIIb ENESTswift study. Leukemia Research, 2018, 67, 109-115.	0.4	11
237	Patients with low OCT-1 activity and high ABCB1 fold rise have poor long-term outcomes in response to tyrosine kinase inhibitor therapy. Leukemia, 2018, 32, 2288-2291.	3.3	11
238	Molecular monitoring in CML: how deep? How often? How should it influence therapy?. Blood, 2018, 132, 2125-2133.	0.6	11
239	Dasatinib Efficacy in Patients with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) and Pre-Existing BCR-ABL Mutations. Blood, 2008, 112, 449-449.	0.6	11
240	Molecular Response at 3 Months On Nilotinib Therapy Predicts Response and Long-Term Outcomes in Patients with Imatinib-Resistant or -Intolerant Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Blood, 2009, 114, 3292-3292.	0.6	11
241	Nilotinib Versus Imatinib in Patients (pts) with Newly Diagnosed Philadelphia Chromosome-Positive (Ph+) Chronic Myeloid Leukemia in Chronic Phase (CML-CP): ENESTnd 36-Month (mo) Follow-up. Blood, 2011, 118, 452-452.	0.6	11
242	Mutation screening of the c-MYB negative regulatory domain in acute and chronic myeloid leukaemia. British Journal of Haematology, 2001, 114, 632-634.	1.2	10
243	Practical Considerations for Monitoring Patients With Chronic Myeloid Leukemia. Seminars in Hematology, 2010, 47, 327-334.	1.8	10
244	OCT-1 as a Determinant of Response to Antileukemic Treatment. Clinical Pharmacology and Therapeutics, 2011, 89, 608-611.	2.3	10
245	NPM1 mutations occur rarely or not at all in chronic myeloid leukaemia patients in chronic phase or blast crisis. Leukemia, 2013, 27, 489-490.	3.3	10
246	A novel somatic JAK2 kinase-domain mutation in pediatric acute lymphoblastic leukemia with rapid on-treatment development of LOH. Cancer Genetics, 2017, 216-217, 86-90.	0.2	10
247	First Approved Kinase Inhibitor for AML. Cell, 2017, 171, 981.	13.5	10
248	Widespread Aberrant Alternative Splicing despite Molecular Remission in Chronic Myeloid Leukaemia Patients. Cancers, 2020, 12, 3738.	1.7	10
249	Higher-Dose Imatinib (600 mg/Day) with Selective Intensification in Newly Diagnosed CML Patients in Chronic Phase; Cytogenetic Response Rates at 12 Months Are Superior to IRIS Blood, 2004, 104, 1001-1001.	0.6	10
250	The Majority of Chronic Myeloid Leukaemia Patients Who Cease Imatinib after Achieving a Sustained Complete Molecular Response (CMR) Remain in CMR, and Any Relapses Occur Early Blood, 2008, 112, 1102-1102.	0.6	10
251	Integrating genetic and epigenetic factors in chronic myeloid leukemia risk assessment: toward gene expression-based biomarkers. Haematologica, 2022, 107, 358-370.	1.7	10
252	Epigenetic modifier gene mutations in chronic myeloid leukemia (CML) at diagnosis are associated with risk of relapse upon treatment discontinuation. Blood Cancer Journal, 2022, 12, 69.	2.8	10

#	Article	IF	CITATIONS
253	Efficacy and safety of imatinib in patients with chronic myeloid leukemia and complete or near-complete cytogenetic response to interferon-α. Cancer, 2007, 110, 801-808.	2.0	9
254	Reply to â€~What do we mean by sensitivity when we talk about detecting minimal residual disease?' by Steinbach and Debatin. Leukemia, 2009, 23, 819-820.	3.3	9
255	Lenalidomide maintenance treatment after imatinib discontinuation: results of a phase 1 clinical trial in chronic myeloid leukaemia. British Journal of Haematology, 2019, 186, e56-e60.	1.2	9
256	Increasing Frequency and Marked Stability of Complete Molecular Response Is Observed in Imatinib-Treated CML Patients with Long-Term Follow Up Blood, 2006, 108, 430-430.	0.6	9
257	Dasatinib-Associated Major Molecular Responses Are Rapidly Achieved in Patients with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) Following Resistance, Suboptimal Response, or Intolerance on Imatinib Blood, 2008, 112, 1095-1095.	0.6	9
258	Nilotinib versus imatinib in patients (pts) with newly diagnosed chronic myeloid leukemia in chronic phase (CML-CP): ENESTnd 3-year (yr) follow-up (f/u) Journal of Clinical Oncology, 2012, 30, 6509-6509.	0.8	9
259	Ph+ ALL: resistance seeds sown early. Blood, 2007, 110, 472-472.	0.6	8
260	Predicting the response of CML patients to tyrosine kinase inhibitor therapy. Current Hematologic Malignancy Reports, 2009, 4, 59-65.	1.2	8
261	Measuring Minimal Residual Disease in Chronic Myeloid Leukemia: Fluorescence In Situ Hybridization and Polymerase Chain Reaction. Clinical Lymphoma and Myeloma, 2009, 9, S266-S271.	1.4	8
262	Predicting the Response of CML Patients to Tyrosine Kinase Inhibitor Therapy. Current Hematologic Malignancy Reports, 2011, 6, 88-95.	1.2	8
263	Safety and efficacy of pulsed imatinib with or without <scp>G</scp> â€ <scp>CSF </scp> <i>versus</i> continuous imatinib in chronic phase chronic myeloid leukaemia patients at 5Âyears followâ€up. British Journal of Haematology, 2013, 163, 674-676.	1.2	8
264	Imatinib-induced gastric antral vascular ectasia in three patients with chronic myeloid leukaemia. International Journal of Hematology, 2015, 102, 639-642.	0.7	8
265	The effect of co-occurring lesions on leukaemogenesis and drug response in T-ALL and ETP-ALL. British Journal of Cancer, 2020, 122, 455-464.	2.9	8
266	Asciminib, a Specific Allosteric BCR-ABL1 Inhibitor, in Patients with Chronic Myeloid Leukemia Carrying the T315I Mutation in a Phase 1 Trial. Blood, 2018, 132, 792-792.	0.6	8
267	Maintenance of Imatinib Dose Intensity in the First Six Months of Therapy for Newly Diagnosed Patients with CML Is Predictive of Molecular Response, Independent of the Ability To Increase Dose at a Later Point Blood, 2005, 106, 164-164.	0.6	8
268	Nilotinib Lowers the Incidence of BCR-ABL Mutations and Improves the Molecular Response Kinetics Compared with Imatinib in Patients (Pts) with Newly Diagnosed Chronic Myeloid Leukemia (CML) Blood, 2010, 116, 3431-3431.	0.6	8
269	Early Switch to Nilotinib Does Not Overcome the Adverse Outcome for CML Patients Failing to Achieve Early Molecular Response On Imatinib, Despite Excellent Overall Outcomes in the TIDEL II Trial. Blood, 2012, 120, 3771-3771.	0.6	8
	Efficacy and Safety of Ponatinih in Patients with Accelerated Phase or Blast Phase Chronic Myeloid		

Efficacy and Safety of Ponatinib in Patients with Accelerated Phase or Blast Phase Chronic Myeloid Leukemia (AP-CML or BP-CML) or Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia (Ph+) Tj ETQq0060 rgBT ØVerlock 1

#	Article	IF	CITATIONS
271	Peripheral Arterial Occlusive Disease (PAOD) In Patients (Pts) Receiving Dasatinib: Experience Across Multiple Clinical Trials. Blood, 2013, 122, 1489-1489.	0.6	8
272	Ponatinib Efficacy and Safety in Patients with the T315I Mutation: Long-Term Follow-up of Phase 1 and Phase 2 (PACE) Trials. Blood, 2014, 124, 4552-4552.	0.6	8
273	Five-year results of the ponatinib phase II PACE trial in heavily pretreated CP-CML patients (pts) Journal of Clinical Oncology, 2017, 35, 7012-7012.	0.8	8
274	HMGN1 plays a significant role in CRLF2 driven Down Syndrome leukemia and provides a potential therapeutic target in this high-risk cohort. Oncogene, 2022, 41, 797-808.	2.6	8
275	Genomic translocation breakpoint sequences are conserved in BCR-ABL1 cell lines despite the presence of amplification. Cancer Genetics and Cytogenetics, 2009, 189, 138-139.	1.0	7
276	Proton pump inhibitors significantly increase the intracellular concentration of nilotinib, but not imatinib in target CML cells. Leukemia, 2013, 27, 1201-1204.	3.3	7
277	<i>BCR-ABL1</i> expression, RT-qPCR and treatment decisions in chronic myeloid leukaemia. Journal of Clinical Pathology, 2016, 69, 817-821.	1.0	7
278	A Method for Next-Generation Sequencing of Paired Diagnostic and Remission Samples to Detect Mitochondrial DNA Mutations Associated with Leukemia. Journal of Molecular Diagnostics, 2017, 19, 711-721.	1.2	7
279	Bone marrow fibrosis associated with long-term imatinib therapy: resolution after switching to a second-generation TKI. Blood Advances, 2019, 3, 370-374.	2.5	7
280	Selective Escalation of Imatinib Therapy and Early Switching to Nilotinib In De Novo Chronic Phase CML Patients: Interim Results From the TIDEL-II Trial. Blood, 2010, 116, 209-209.	0.6	7
281	Nilotinib Shows Safety and Efficacy in Older Patients (≥ 65 years) with Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase Comparable with That in Younger Patients with Chronic Myeloid Leukemia in Chronic Phase: Results From ENESTnd,. Blood, 2011, 118, 3768-3768.	0.6	7
282	Steady-State Imatinib Trough Levels as Well as Dose Interruptions Are Associated with Clinical Response (CCyR and MMR) and Adverse Events (AEs) in Patients with Chronic Myeloid Leukemia (CML) Receiving IM as Frontline Therapy Blood, 2009, 114, 2213-2213.	0.6	7
283	Asciminib for chronic myeloid leukaemia: Next questions. British Journal of Haematology, 2022, 199, 322-331.	1.2	7
284	Clinical Strategies to Achieve an Early and Successful Response to Tyrosine Kinase Inhibitor Therapy. Seminars in Hematology, 2009, 46, S11-S15.	1.8	6
285	Living with CML: is death no longer the end (point)?. Blood, 2015, 126, 2-4.	0.6	6
286	Clinical utility of genomic DNA Q-PCR for the monitoring of a patient with atypical e19a2 <i>BCR-ABL1</i> transcripts in chronic myeloid leukemia. Leukemia and Lymphoma, 2020, 61, 2527-2529.	0.6	6
287	Combination of Nilotinib and Pegylated Interferon Alfa-2b Results in High Molecular Response Rates in Chronic Phase CML: Interim Results of the ALLG CML 11 Pinnacle Study. Blood, 2018, 132, 459-459.	0.6	6

Imatinib (IM) Pharmacokinetic (PK) Exposure and Its Correlation with Clinical Outcome in Patients with Chronic-Phase Chronic Myeloid Leukemia (CML-CP) for 400 Mg and 800 Mg Daily Doses (Tyrosine) Tj ETQq0 000 argBT /Overlock 10

#	Article	IF	CITATIONS
289	Multivariate Analyses of the Clinical and Molecular Parameters Associated with Efficacy and Safety in Patients with Chronic Myeloid Leukemia (CML) and Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia (Ph+ ALL) Treated with Ponatinib in the PACE Trial. Blood, 2012, 120, 3747-3747.	0.6	6
290	Impact Of Baseline (BL) Mutations, Including Low-Level and Compound Mutations, On Ponatinib Response and End Of Treatment (EOT) Mutation Analysis In Patients (Pts) With Chronic Phase Chronic Myeloid Leukemia (CP-CML). Blood, 2013, 122, 652-652.	0.6	6
291	Treatment-free remission (TFR) in patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP) treated with second-line nilotinib (NIL): First results from the ENESTop study Journal of Clinical Oncology, 2016, 34, 7054-7054.	0.8	6
292	ENESTop 192-week results: Treatment-free remission (TFR) in patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP) after stopping second-line (2L) nilotinib (NIL) Journal of Clinical Oncology, 2019, 37, 7005-7005.	0.8	6
293	Daclizumab has poor efficacy in steroid-refractory severe acute graft-versus-host disease: a single centre experience with 12 allograft patients. Bone Marrow Transplantation, 2008, 41, 409-410.	1.3	5
294	Elevated PTPN2 expression is associated with inferior molecular response in de-novo chronic myeloid leukaemia patients. Leukemia, 2014, 28, 702-705.	3.3	5
295	Production of GM-CSF by CML Cells Can Modulate the Anti-Proliferative and Pro-Apoptotic Effects of Imatinib on CML CD34+ Cells Blood, 2005, 106, 2865-2865.	0.6	5
296	Nilotinib in Imatinib-Resistant or -Intolerant Patients (pts) with Chronic Myeloid Leukemia in Chronic Phase (CML-CP): 48-Month Follow-up Results of a Phase 2 Study,. Blood, 2011, 118, 3770-3770.	0.6	5
297	Early Molecular Response and Female Sex Strongly Predict Achievement of Stable Undetectable BCR-ABL1, a Criterion for Imatinib Discontinuation in Patients with CML. Blood, 2012, 120, 165-165.	0.6	5
298	Molecular Responses with Ponatinib in Patients with Philadelphia Chromosome Positive (Ph+) Leukemia: Results From the PACE Trial. Blood, 2012, 120, 3763-3763.	0.6	5
299	The Impact of Ponatinib Versus Allogeneic Stem Cell Transplant (SCT) on Outcomes in Patients with Chronic Myeloid Leukemia (CML) or Philadelphia Chromosome-Positive Acute Lymphoblastic Leukemia (Ph+ ALL) with the T315I Mutation. Blood, 2015, 126, 480-480.	0.6	5
300	Nilotinib versus imatinib in patients (pts) with newly diagnosed chronic myeloid leukemia in chronic phase (CML-CP): ENESTnd 4-year (y) update Journal of Clinical Oncology, 2013, 31, 7052-7052.	0.8	5
301	COVID-19 in Patients with Chronic Myeloid Leukemia: Poor Outcomes for Patients with Comorbidities, Older Age, Advanced Phase Disease, and Those from Low-Income Countries: An Update of the Candid Study. Blood, 2021, 138, 634-634.	0.6	5
302	Strategies for the treatment of imatinib-resistant chronic myeloid leukemia. Clinical Advances in Hematology and Oncology, 2003, 1, 538-45, 559.	0.3	5
303	Remembrance of things past — discontinuation of second-generation TKI therapy for CML. Nature Reviews Clinical Oncology, 2017, 14, 201-202.	12.5	4
304	Monitoring Disease Response. , 2007, , 143-164.		4
305	Azacytidine Sensitizes AML Cells for Effective Elimination By CD123 CAR T-Cells. Blood, 2019, 134, 3904-3904.	0.6	4
306	Mutated Cancer-Related Genes Detected at Diagnosis of CML and a Novel Class of Variant Associated with the Philadelphia Translocation Are Both Independent Predictors of Inferior Outcomes. Blood, 2020, 136, 46-47.	0.6	4

#	Article	lF	CITATIONS
307	Major Cytogenetic Responses to BMS-354825 in Patients with Chronic Myeloid Leukemia Are Associated with a One to Two Log Reduction in BCR-ABL Transcript Blood, 2004, 104, 1008-1008.	0.6	4
308	The Initial Molecular Response of Chronic Phase CML Patients Treated with Second Generation ABL Inhibitor Therapy after Imatinib Failure Can Predict Inadequate Response and Provide Indications for Rational Mutation Screening. Blood, 2008, 112, 331-331.	0.6	4
309	A Review of Mutation Analysis In the TOPS Trial of Standard Dose Versus High Dose IM In CML Suggests That Refinements to the ELN Recommendations for Mutation Screening May Be Appropriate. Blood, 2010, 116, 889-889.	0.6	4
310	Commitment of CML Cells to Apoptotic Cell Death Depends On the Length of Exposure to Das and the Level of STAT5 Activity. Blood, 2012, 120, 3736-3736.	0.6	4
311	First-Line Treatment and Management of Chronic Myeloid Leukemia (CML) in Clinical Practice: Update of > 1800 Patients (Pts) in the WORLD CML Registry. Blood, 2012, 120, 3750-3750.	0.6	4
312	Ponatinib In Heavily Pretreated Patients With Chronic Phase Chronic Myeloid Leukemia (CP-CML): Management Of Adverse Events (AEs). Blood, 2013, 122, 1496-1496.	0.6	4
313	Impact of baseline mutations on response to ponatinib and end of treatment mutation analysis in patients with chronic myeloid leukemia Journal of Clinical Oncology, 2013, 31, 7001-7001.	0.8	4
314	Long-term treatment-free remission (TFR) in patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP) after stopping second-line (2L) nilotinib: ENESTop 144-wk results Journal of Clinical Oncology, 2018, 36, 7003-7003.	0.8	4
315	Randomized, Open-Label, Multicenter, Phase 2 Study of Asciminib (ABLOO1) As an Add-on to Imatinib Versus Continued Imatinib Versus Switch to Nilotinib in Patients with Chronic Myeloid Leukemia in Chronic Phase Who Have Not Achieved a Deep Molecular Response with Frontline Imatinib. Blood, 2019, 134, 5910-5910.	0.6	4
316	Exploring the oncogenic and therapeutic target potential of the MYB-TYK2 fusion gene in B-cell acute lymphoblastic leukemia. Cancer Gene Therapy, 2022, 29, 1140-1152.	2.2	4
317	An imatinib-only window followed by imatinib and chemotherapy for Philadelphia chromosome-positive acute leukemia: long-term results of the CMLALL1 trial. Leukemia and Lymphoma, 2015, 56, 630-638.	0.6	3
318	Comment on "KB004, a first in class monoclonal antibody targeting the receptor tyrosine kinase EphA3, in patients with advanced hematologic malignancies: Results from a phase 1 study― Leukemia Research, 2017, 55, 55-57.	0.4	3
319	Asciminib (ABL001) in Combination with Imatinib in Patients with Chronic Myeloid Leukemia in Chronic Phase Who Have Not Achieved a Deep Molecular Response with Long-Term Frontline Imatinib: A Randomized, Open-Label, Multicenter, Phase 2 Study. Clinical Lymphoma, Myeloma and Leukemia, 2018, 18, S222-S223.	0.2	3
320	Highâ€risk Bâ€cell acute lymphoblastic leukaemia presenting with hypereosinophilia and acquiring a novel <i>PAX5</i> fusion on relapse. British Journal of Haematology, 2020, 191, 301-304.	1.2	3
321	Analyses of Predictors of Durable Treatment-Free Remission in Patients with Chronic Myeloid Leukemia in Chronic Phase Following Frontline or Second-Line Nilotinib. Blood, 2019, 134, 2932-2932.	0.6	3
322	ENESTop 5-Year Update: Durability of Treatment-Free Remission Following Second-Line Nilotinib and Exploratory Analysis of Molecular Response Regain after Nilotinib Re-Initiation in Patients with Chronic Myeloid Leukemia. Blood, 2020, 136, 29-30.	0.6	3
323	Major Molecular Responses to Dasatinib (BMS-354825) Are Observed in Imatinib-Resistant Late Stage Chronic and Advanced CML Patients: Impact and Fate of Imatinib-Resistant Clones in Dasatinib-Treated Patients Blood, 2005, 106, 437-437.	0.6	3
324	The Most Common Dasatinib-Resistant BCR-ABL Kinase Domain Mutations in Patients with Chronic Myeloid Leukemia Are Sensitive to VX-680: Rationale for Early Combination Kinase Inhibitor Therapy Blood, 2006, 108, 2175-2175.	0.6	3

#	Article	IF	CITATIONS
325	Genome-Wide Analysis of Genetic Alterations in Chronic Myelogenous Leukemia Blood, 2008, 112, 1089-1089.	0.6	3
326	The Expression of shp-1 and SHP-2: A Novel Powerful Predictor of Major Molecular Response (MMR) Achievement in Chronic Myeloid Leukemia Gleevec-Treated Patients Enrolled into the TOPS Clinical Trial Blood, 2008, 112, 1106-1106.	0.6	3
327	Complete Molecular Response (CMR) Rate with Nilotinib in Patients (pts) with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) without CMR After ≥ 2 Years on Imatinib: Preliminary Results From the Randomized ENESTcmr Trial of Nilotinib 400 Mg Twice Daily (BID) Vs Imatinib. Blood, 2011, 118, 606-606.	0.6	3
328	Switching to Nilotinib Is Associated with Continued Deeper Molecular Responses in CML-CP Patients with Minimal Residual Disease After ≥ 2 Years On Imatinib: Enestcmr 2-Year Follow-up Results. Blood, 2012, 120, 694-694.	0.6	3
329	Detection of BCR-ABL1 Compound and Polyclonal Mutants in Chronic Myeloid Leukemia Patients Using a Novel Next Generation Sequencing Approach That Minimises PCR and Sequencing Errors. Blood, 2014, 124, 399-399.	0.6	3
330	Treatment-Free Remission (TFR) Eligibility in Patients (pts) with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) and Residual Disease on Long-Term Imatinib (IM) Who Switched to Second-Line Nilotinib (NIL). Blood, 2015, 126, 4029-4029.	0.6	3
331	Switch to nilotinib versus continued imatinib in patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP) with detectable BCR-ABL after 2 or more years on imatinib: ENESTcmr 12-month (mo) follow-up Journal of Clinical Oncology, 2012, 30, 6505-6505.	0.8	3
332	EPIC: A phase III trial of ponatinib (PON) versus imatinib (IM) in patients (pts) with newly diagnosed CP-CML Journal of Clinical Oncology, 2014, 32, 7023-7023.	0.8	3
333	ATP Dependent Efflux Transporters ABCB1 and ABCG2 Are Unlikely to Impact the Efficacy, or Mediate Resistance to the Tyrosine Kinase Inhibitor, Ponatinib. Blood, 2011, 118, 2745-2745.	0.6	3
334	Impact of early molecular response to nilotinib (NIL) or imatinib (IM) on the long-term outcomes of newly diagnosed patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP): Landmark analysis of 4-year (y) data from ENESTnd Journal of Clinical Oncology, 2013, 31, 7054-7054.	0.8	3
335	How complete is "complete―molecular response in imatinib-treated chronic myeloid leukemia?. Leukemia and Lymphoma, 2008, 49, 1230-1231.	0.6	2
336	Sudden blast crisis in chronic myeloid leukemia treated with tyrosine kinase inhibitors. Leukemia and Lymphoma, 2012, 53, 1251-1252.	0.6	2
337	Management of Pregnancy in Women With Chronic Myeloid Leukemia. Journal of Clinical Oncology, 2018, 36, 2657-2658.	0.8	2
338	Early Management of CML. Current Hematologic Malignancy Reports, 2019, 14, 480-491.	1.2	2
339	Counterpoint: There is a best duration of deep molecular response for treatmentâ€free remission, but it is patientâ€specific, and that is the challenge. British Journal of Haematology, 2021, 192, 24-27.	1.2	2
340	Lymphoid Foci in Bone Marrow of Patients with Chronic Myeloid Leukaemia Treated with Imatinib Blood, 2005, 106, 2008-2008.	0.6	2
341	ABCB1 Overexpression May Predispose Imatinib Treated CML Patients to the Development of Abl Kinase Domain Mutations, and May Be an Important Contributor to Acquired Resistance Blood, 2006, 108, 2144-2144.	0.6	2
342	A Phase 3 Pilot Study of Continuous Imatinib Versus Pulsed Imatinib with or without G-CSF in Patients with Chronic Phase CML Who Have Achieved a Complete Cytogenetic Response to Imatinib Blood, 2007, 110, 1033-1033.	0.6	2

#	Article	IF	CITATIONS
343	Comparison of Steady-State Imatinib (IM) Trough Levels, Clinical Response, and Safety Between Caucasian and Asian Patients with Chronic Lyeloid Leukemia in Chronic Phase (CML-CP) Treated with 400mg and 800mg Daily Doses of IM in the Tyrosine Kinase Inhibitor Optimization and Selectivity (TOPS) Study Blood, 2009, 114, 1127-1127.	0.6	2
344	Response and Outcomes to Nilotinib at 24 Months in Imatinib-Resistant Chronic Myeloid Leukemia Patients in Chronic Phase (CML-CP) and Accelerated Phase (CML-AP) with and without BCR-ABL Mutations Blood, 2009, 114, 1130-1130.	0.6	2
345	A Worldwide Observational Registry Collecting Longitudinal Data on Management of Chronic Myeloid Leukemia Patients (The WORLD CML Registry) – 2nd Annual Interim Analysis. Blood, 2010, 116, 2292-2292.	0.6	2
346	Imatinib Dose Interruption in Responding CML Patients Is Associated with Characteristic BCR-ABL Kinetics, Which Could Help to Differentiate Non-Adherence From Drug Resistance. Blood, 2011, 118, 113-113.	0.6	2
347	Results From the ENESTnd Extension Study: Efficacy and Safety of Patients (pts) with Chronic Myeloid Leukemia in Chronic Phase (CML-CP), Treated with Nilotinib 400 Mg Twice Daily (BID) After Suboptimal Response (SoR) or Treatment Failure (TF) to Imatinib 400 Mg Once Daily (QD) or Nilotinib 300 Mg BID. Blood. 2011, 118, 114-114.	0.6	2
348	Efficacy and Safety of Ponatinib According to Prior Approved Tyrosine Kinase Inhibitor (TKI) Therapy in Patients with Chronic Myeloid Leukemia in Chronic Phase (CP-CML): Results From the PACE Trial. Blood, 2012, 120, 3749-3749.	0.6	2
349	Dose-Optimized Nilotinib (NIL) in Patients (Pts) with Newly Diagnosed Chronic Myeloid Leukemia in Chronic Phase (CML-CP): Final Results from ENESTxtnd Study. Blood, 2015, 126, 344-344.	0.6	2
350	The Allosteric Inhibitor ABL001 Is Susceptible to Resistance in Vitro Mediated By Overexpression of the Drug Efflux Transporters ABCB1 and ABCG2. Blood, 2015, 126, 4841-4841.	0.6	2
351	Novel Fusion Genes at CML Diagnosis Reveal a Complex Pattern of Genomic Rearrangements and Sequence Inversions Associated with the Philadelphia Chromosome in Patients with Early Blast Crisis. Blood, 2016, 128, 1219-1219.	0.6	2
352	Efficacy and safety of ponatinib in heavily pretreated leukemia patients in the PACE trial: 3-year results Journal of Clinical Oncology, 2015, 33, e18052-e18052.	0.8	2
353	The IC50 Assay Is Predictive of Molecular Response, and Indicative of Optimal Dose in De-Novo CML Patients Blood, 2008, 112, 1109-1109.	0.6	2
354	Analysis of Molecular Data and the Emergence of Mutations for Chronic-Phase Chronic Myelogenous Leukemia (CML-CP) Patients Treated with Dasatinib After Imatinib Failure Blood, 2009, 114, 3282-3282.	0.6	2
355	RNA-Based Targeted Gene Sequencing Improves the Diagnostic Yield of Mutant Detection in Chronic Myeloid Leukemia. Journal of Molecular Diagnostics, 2022, 24, 803-822.	1.2	2
356	Highly sensitive droplet digital polymerase chain reaction for <i> <scp>BCR</scp> :: </i> <scp> <i>ABL1</i> messenger RNA </scp> identifies patients with chronic myeloid leukaemia with a low probability of achieving treatmentâ€free remission. British Journal of Haematology, 0, , .	1.2	2
357	Is drug treatment superior to allografting as first-line therapy in chronic myeloid leukemia?. Nature Clinical Practice Oncology, 2008, 5, 14-15.	4.3	1
358	Discontinuation of Therapy and Treatment-Free Remission in CML. , 2016, , 183-193.		1
359	Treatment-Free Remission After Second-Line Nilotinib Treatment. Annals of Internal Medicine, 2018, 169, 510.	2.0	1
360	What's NEXT for CML-NGS mutation screening. Blood, 2020, 135, 515-516.	0.6	1

#	Article	IF	CITATIONS
361	In-vitro modeling of TKI resistance in the high-risk B-cell acute lymphoblastic leukemia fusion gene RANBP2-ABL1 - implications for targeted therapy. Leukemia and Lymphoma, 2021, 62, 1157-1166.	0.6	1
362	Pre-Imatinib Factors Can Be Used To Define the Risk of BCR-ABL Mutations for Patients with CML in Chronic Phase and Identify a Minority Who Should Have Regular Mutation Screening Blood, 2005, 106, 1079-1079.	0.6	1
363	Enhancing the Functional Activity of the OCT-1 Influx Pump May Overcome the Negative Impact of Low OCT-1 Activity in Imatinib Treated CML Patients. Blood, 2008, 112, 723-723.	0.6	1
364	The Functional Activity of the OCT-1 Protein Is Predictive of Molecular Response and Survival in CP-CML Patients Treated with Imatinib: A 5 Year Update of the TIDEL Trial Blood, 2009, 114, 507-507.	0.6	1
365	Proton Pump Inhibitors Augment Nilotinib and Dasatinib Mediated Bcr-Abl Kinase Inhibition. Blood, 2010, 116, 3991-3991.	0.6	1
366	Survey of the Frontline Treatment and Management of Chronic Myeloid Leukemia (CML) in a Real-Word Setting: The 3rd Annual Update of the Worldwide Observational Registry Collecting Longitudinal Data on Management of Chronic Myeloid Leukemia Patients (The WORLD CML Registry). Blood, 2011, 118, 1695-1695.	0.6	1
367	The Clinical Significance of Early Imatinib Induced ABCB1 Overexpression in Chronic Phase CML Patients: A TIDEL II Sub-Study. Blood, 2015, 126, 348-348.	0.6	1
368	A 20 Gene Expression Signature That Predicts Early Molecular Response Failure in Chronic Phase CML Patients Treated with Frontline Imatinib. Blood, 2015, 126, 596-596.	0.6	1
369	IKZF1 (Ikaros) Deletions Are a Hallmark of BCR-ABL1 Positive Acute Lymphoblastic Leukemia Blood, 2007, 110, 721-721.	0.6	1
370	DNA-Based Monitoring of Minimal Residual Disease(MRD) in Chronic Myeloid Leukemia(CML) Blood, 2008, 112, 1111-1111.	0.6	1
371	Early Switching From Imatinib to Nilotinib In CML Patients Failing to Achieve Early Molecular Targets May Not Be An Effective Approach In Patients with Very Low OCT-1 Activity: A TIDEL II Sub-Study. Blood, 2010, 116, 356-356.	0.6	1
372	Achieving the Deep Molecular Response Levels Required for an Imatinib Discontinuation Trial Is Strongly Associated with the BCR-ABL Level at the First Qualifying Timepoint. Blood, 2014, 124, 4561-4561.	0.6	1
373	Impact of early landmark responses with ponatinib on 4-yr outcomes in CP-CML patients (pts) in PACE, a pivotal phase II trial Journal of Clinical Oncology, 2017, 35, 7050-7050.	0.8	1
374	Clinical development of asciminib (ABL001) in chronic myeloid leukemia (CML): A randomized phase 3 study vs. bosutinib Journal of Clinical Oncology, 2018, 36, TPS7081-TPS7081.	0.8	1
375	RNA Splicing Defects in Cancer-Linked Genes Indicate Mutation or Focal Gene Deletion and Are Associated with TKI Resistance in CML. Blood, 2019, 134, 662-662.	0.6	1
376	Distinct Senescent Bone Marrow Microenvironment in Therapy-Related Myeloid Neoplasms. Blood, 2021, 138, 2585-2585.	0.6	1
377	Trial in Progress: A Multicenter, Open Label, Randomized, Phase III Study of Asciminib (80 mg Once) Tj ETQq1 1 in Chronic Phase. Blood, 2021, 138, 1478-1478.	0.784314 0.6	rgBT /Overloc 1
378	Next Generation Genomic Analyses in T-ALL Patients Identify Recurrent and Novel Genomic Abnormalities. Blood, 2020, 136, 13-14.	0.6	1

#	Article	IF	CITATIONS
379	Response: Reliability of PCR for BCR-ABL transcripts. Blood, 2007, 109, 2263-2264.	0.6	Ο
380	Challenges of Treatment: Tyrosine Kinase Inhibitor-Resistant Chronic Myeloid Leukemia. , 2014, , 53-65.		0
381	Response to †Overexpression of ABCB1 as prediction marker for CML: How close we are to translation into clinics?'. Leukemia, 2017, 31, 769-770.	3.3	Ο
382	Treatment Free Remission for Chronic Myeloid Leukemia in 2017. Clinical Lymphoma, Myeloma and Leukemia, 2017, 17, S102-S104.	0.2	0
383	Treatment-Free Remission in CML: Selecting the Best Candidates. Clinical Lymphoma, Myeloma and Leukemia, 2018, 18, S3-S5.	0.2	Ο
384	Response-Related Predictors of Survival and of Treatment-Free Remission in CML. Hematologic Malignancies, 2021, , 245-264.	0.2	0
385	DNA-Based Measurement of BCR-ABL in Chronic Myeloid Leukemia (CML) Blood, 2007, 110, 2946-2946.	0.6	0
386	An MMR Control RNA for Reliable Monitoring of BCR-ABL Transcripts in Treated CML Patients Blood, 2007, 110, 2939-2939.	0.6	0
387	Mathematical Simulation of BCR-ABL Real Time Quantitative Polymerase Chain Reaction (RQ-PCR) for Chronic Myeloid Leukemia (CML) Response Monitoring Provides Insight on the Basis of International Standardization Blood, 2008, 112, 2124-2124.	0.6	Ο
388	A Worldwide Observational Registry Collecting Longitudinal Data On the Management of CML Patients (The WORLD CML Registry) - Summary of the First 1001 Patients Blood, 2009, 114, 4267-4267.	0.6	0
389	Association Between Imatinib (IM) Transporters and Metabolizing Enzymes Genotype and Response in Newly Diagnosed Chronic Myeloid Leukemia (CML) Patients (Pts) Is Influenced by Ethnicity Blood, 2009, 114, 3283-3283.	0.6	0
390	Imatinib PK: Observations From the TIDEL II Study. Blood, 2010, 116, 2288-2288.	0.6	0
391	Specific Drug Transporter Genotypes Are Significantly Associated with Increased Rates of Major and Complete Molecular Responses In Newly Diagnosed Chronic Myeloid Leukemia Patients Treated with Imatinib – A TOPS Correlative Substudy. Blood, 2010, 116, 670-670.	0.6	0
392	Detection of Low Level Nilotinib or Dasatinib Resistant BCR-ABL Mutations by Mass Spectrometry In CML Patients Who Fail Imatinib Is Highly Predictive of Their Subsequent Clonal Expansion When Treated with the Drug for Which Their Mutation Confers Resistance. Blood, 2010, 116, 891-891.	0.6	0
393	Towards DNA-Based Monitoring of Therapy In Chronic Myeloid Leukemia. Blood, 2010, 116, 2284-2284.	0.6	0
394	Modelling of TKI Resistance In CML Cell Lines: Kinase Domain Mutations Usually Arise In the Setting of BCR-ABL Overexpression Blood, 2010, 116, 3383-3383.	0.6	0
395	Mutation Analysis of BCR-ABL Tyrosine Kinase Domain In New Chronic Phase-Chronic Myeloid Leukemia Patients with Suboptimal Response or Treatment Failure From Imatinib Treatment Blood, 2010, 116, 3441-3441.	0.6	0
396	Targeting Rac2 - Mitochondrial Respiratory Chain Complex III Signaling to Prevent Genomic Instability in Leukemia Stem and Progenitor Cells. Blood, 2011, 118, 2736-2736.	0.6	0

#	ARTICLE	IF	CITATIONS
397	The Strategy of Early Nilotinib Switch Based on Failure to Achieve Optimal Molecular Targets on Imatinib May Not Overcome the Negative Impact of a Low OCT-1 Activity in De-Novo CP-CML Patients. Blood, 2011, 118, 1690-1690.	0.6	0
398	Non-Steroidal Anti-Inflammatory Drugs and Imatinib; Drug Interactions That May Impact Efficacy,. Blood, 2011, 118, 3501-3501.	0.6	0
399	Multiple Low Level Mutations Identifies Imatinib Resistant CML Patients At Risk of Poor Response to Second-Line Inhibitor Therapy, Irrespective of the Resistance Profile of the Mutations. Blood, 2011, 118, 111-111.	0.6	0
400	The patient's BCR-ABL1 Kinase Domain Mutation History Is Important for Decisions Regarding Tyrosine Kinase Inhibitor Therapy. Blood, 2012, 120, 1692-1692.	0.6	0
401	Switching patients (pts) with chronic myeloid leukemia in chronic phase (CML-CP) with residual disease on long-term imatinib (IM) to nilotinib (NIL): ENESTcmr 24-mo follow-up Journal of Clinical Oncology, 2013, 31, 7053-7053.	0.8	Ο
402	EPIC: A phase III randomized, open-label study of ponatinib versus imatinib in adult patients with newly diagnosed chronic myeloid leukemia in chronic phase Journal of Clinical Oncology, 2013, 31, TPS7129-TPS7129.	0.8	0
403	MicroRNA Dysregulation in Newly Diagnosed Chronic Myeloid Leukaemia Patients. Blood, 2013, 122, 4985-4985.	0.6	Ο
404	The Depth Of In Vivo Kinase Inhibition Achieved Over The First Month Of Nilotinib Therapy Predicts For Subsequent Molecular Response, and Is Closely Related To Nilotinib Plasma Levels. Blood, 2013, 122, 256-256.	0.6	0
405	Role Of Peroxisome Proliferator-Activated Receptor Gamma (PPARγ) and Its Ligands In The Regulation Of Functional OCT-1 Activity In CML Cells. Blood, 2013, 122, 1470-1470.	0.6	Ο
406	STAT5 Is a Critical Component Of The Time-Dependent Sensitivity Of CML Cells To TKI Treatment In a Bcr-Abl-Dependent, But JAK2-Independent Manner. Blood, 2013, 122, 2705-2705.	0.6	0
407	PCR-Mediated Recombination Can Lead To Artificial Chimera Formation, Which May Pose As BCR-ABL1 Compound Mutations. Blood, 2013, 122, 4014-4014.	0.6	Ο
408	Increasing Expression Of The Efflux Transporter ABCB1 May Predispose CML Cells To Overt TKI Resistance. Blood, 2013, 122, 5157-5157.	0.6	0
409	Additional BCR-ABL1 Mutations Identified By Sensitive Mass Spectrometry In Chronic Phase CML Patients With T315I Treated With Ponatinib Are Associated With Relatively Inferior Responses and Outcome. Blood, 2013, 122, 651-651.	0.6	Ο
410	High Recombination Activating Gene (RAG) Expression and RAG Mediated Recombination Is Associated with Oncogenic Rearrangement Observed with Tyrosine Kinase Inhibitor Resistant CML. Blood, 2018, 132, 3001-3001.	0.6	0
411	Accumulation of JAK Activation-Loop Phosphorylation Promotes Type I JAK Inhibitor Withdrawal Syndrome in Myelofibrosis. Blood, 2018, 132, 1787-1787.	0.6	0
412	Therapy-Related Myeloid Neoplasm Has a Distinct Pro-Inflammatory Bone Marrow Microenvironment and Delayed DNA Damage Repair. Blood, 2020, 136, 37-38.	0.6	0