Susan Branford

List of Publications by Year in Descending Order

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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.

 179
 11,056
 42
 104

 papers
 citations
 h-index
 g-index

 187
 12,605
 4.6
 5.62

 ext. papers
 ext. citations
 avg, IF
 L-index

#	Paper	IF	Citations
179	Epigenetic modifier gene mutations in chronic myeloid leukemia (CML) at diagnosis are associated with risk of relapse upon treatment discontinuation <i>Blood Cancer Journal</i> , 2022 , 12, 69	7	O
178	Integrating genetic and epigenetic factors in chronic myeloid leukemia risk assessment: toward gene expression-based biomarkers. <i>Haematologica</i> , 2021 ,	6.6	1
177	Clonal evolution and clinical implications of genetic abnormalities in blastic transformation of chronic myeloid leukaemia. <i>Nature Communications</i> , 2021 , 12, 2833	17.4	7
176	RUNX1 mutations in blast-phase chronic myeloid leukemia associate with distinct phenotypes, transcriptional profiles, and drug responses. <i>Leukemia</i> , 2021 , 35, 1087-1099	10.7	13
175	Early BCR-ABL1 kinetics are predictive of subsequent achievement of treatment-free remission in chronic myeloid leukemia. <i>Blood</i> , 2021 , 137, 1196-1207	2.2	21
174	Response-Related Predictors of Survival and of Treatment-Free Remission in CML. <i>Hematologic Malignancies</i> , 2021 , 245-264	Ο	
173	Clinical utility of genomic DNA Q-PCR for the monitoring of a patient with atypical e19a2 transcripts in chronic myeloid leukemia. <i>Leukemia and Lymphoma</i> , 2020 , 61, 2527-2529	1.9	2
172	Aberrant RAG-mediated recombination contributes to multiple structural rearrangements in lymphoid blast crisis of chronic myeloid leukemia. <i>Leukemia</i> , 2020 , 34, 2051-2063	10.7	11
171	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. <i>Blood</i> , 2020 , 136, 46-47	2.2	2
170	Why is it critical to achieve a deep molecular response in chronic myeloid leukemia?. <i>Haematologica</i> , 2020 , 105, 2730-2737	6.6	1
169	Lineage of measurable residual disease in patients with chronic myeloid leukemia in treatment-free remission. <i>Leukemia</i> , 2020 , 34, 1052-1061	10.7	23
168	Widespread Aberrant Alternative Splicing despite Molecular Remission in Chronic Myeloid Leukaemia Patients. <i>Cancers</i> , 2020 , 12,	6.6	3
167	Chronic myeloid leukaemia: The dangers of not knowing your BCR-ABL1 transcript. <i>Leukemia Research</i> , 2019 , 87, 106231	2.7	5
166	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. <i>Leukemia</i> , 2019 , 33, 1835-1850	10.7	50
165	The mutational burden of therapy-related myeloid neoplasms is similar to primary myelodysplastic syndrome but has a distinctive distribution. <i>Leukemia</i> , 2019 , 33, 2842-2853	10.7	19
164	Modeling the safe minimum frequency of molecular monitoring for CML patients attempting treatment-free remission. <i>Blood</i> , 2019 , 134, 85-89	2.2	14
163	mutations are recurrently acquired during chronic myeloid leukemia progression and interfere with myeloid differentiation pathways. <i>Haematologica</i> , 2019 , 104, 1789-1797	6.6	12

(2018-2019)

162	Lenalidomide maintenance treatment after imatinib discontinuation: results of a phase 1 clinical trial in chronic myeloid leukaemia. <i>British Journal of Haematology</i> , 2019 , 186, e56-e60	4.5	7
161	Bone marrow fibrosis associated with long-term imatinib therapy: resolution after switching to a second-generation TKI. <i>Blood Advances</i> , 2019 , 3, 370-374	7.8	2
160	The Hidden Pathogenesis of CML: Is BCR-ABL1 the First Event?. <i>Current Hematologic Malignancy Reports</i> , 2019 , 14, 501-506	4.4	5
159	Gene Expression Signature Predicts Deep Molecular Response (DMR) in Chronic Myeloid Leukemia (CML): An Exploratory Biomarker Analysis from ENESTnd. <i>Blood</i> , 2019 , 134, 665-665	2.2	2
158	Combination of Nilotinib and Pegylated Interferon Alfa-2B Results in High Rates of MR4.5 at 24 Months - Primary Analysis of the ALLG CML 11 Pinnacle Study. <i>Blood</i> , 2019 , 134, 2926-2926	2.2	3
157	An RNA-Based Next Generation Sequencing (NGS) Strategy Detects More Cancer Gene Mutations Than a DNA-Based Approach for the Prediction and Assessment of Resistance in CML. <i>Blood</i> , 2019 , 134, 2918-2918	2.2	
156	Pro-Active Dasatinib Dose Reduction Based on Trough Levels May Minimise Toxicity and Preserve Efficacy - Interim Analysis of the ALLG CML 12 Direct Study. <i>Blood</i> , 2019 , 134, 4150-4150	2.2	
155	RNA Splicing Defects in Cancer-Linked Genes Indicate Mutation or Focal Gene Deletion and Are Associated with TKI Resistance in CML. <i>Blood</i> , 2019 , 134, 662-662	2.2	O
154	Familial Clustering of Hematological Malignancies: Harbingers of Wider Germline Cancer Susceptibility. <i>Blood</i> , 2019 , 134, 3794-3794	2.2	
153	NGS in CML - New standard diagnostic procedure?. HemaSphere, 2019 , 3,	0.3	1
152	The effect of tyrosine kinase inhibitor interruption and interferon use on pregnancy outcomes and long-term disease control in chronic myeloid leukemia. <i>Leukemia and Lymphoma</i> , 2019 , 60, 1796-1802	1.9	13
151	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. <i>Leukemia Research</i> , 2018 , 67, 109-115	2.7	8
150	genomic DNA PCR response kinetics during first-line imatinib treatment of chronic myeloid leukemia. <i>Haematologica</i> , 2018 , 103, 2026-2032	6.6	22
149	High Recombination Activating Gene (RAG) Expression and RAG Mediated Recombination Is Associated with Oncogenic Rearrangement Observed with Tyrosine Kinase Inhibitor Resistant CML. <i>Blood</i> , 2018 , 132, 3001-3001	2.2	
148	Development of a Data Portal for Aggregation and Analysis of Genomics Data in Familial Platelet Disorder with Predisposition to Myeloid Malignancy - the RUNX1.DB. <i>Blood</i> , 2018 , 132, 5241-5241	2.2	
147	The new allosteric inhibitor asciminib is susceptible to resistance mediated by ABCB1 and ABCG2 overexpression. <i>Oncotarget</i> , 2018 , 9, 13423-13437	3.3	24
146	Long-term treatment-free remission of chronic myeloid leukemia with falling levels of residual leukemic cells. <i>Leukemia</i> , 2018 , 32, 2572-2579	10.7	37

144	Long-Term Outcomes of Imatinib Treatment for Chronic Myeloid Leukemia. <i>New England Journal of Medicine</i> , 2017 , 376, 917-927	59.2	618
143	The allosteric inhibitor ABL001 enables dual targeting of BCR-ABL1. <i>Nature</i> , 2017 , 543, 733-737	50.4	256
142	CRISPR-Cas9-mediated saturated mutagenesis screen predicts clinical drug resistance with improved accuracy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017 , 114, 11751-11756	11.5	27
141	and germ line variants predict response and identify CML patients with the greatest risk of imatinib failure. <i>Blood Advances</i> , 2017 , 1, 1369-1381	7.8	9
140	The impact of multiple low-level BCR-ABL1 mutations on response to ponatinib. <i>Blood</i> , 2016 , 127, 1870	-80≥	45
139	Current developments in molecular monitoring in chronic myeloid leukemia. <i>Therapeutic Advances in Hematology</i> , 2016 , 7, 237-251	5.7	21
138	Monitoring and defining early response: Where to draw the line?. <i>Best Practice and Research in Clinical Haematology</i> , 2016 , 29, 284-294	4.2	4
137	Validation of a rapid one-step high sensitivity real-time quantitative PCR system for detecting major BCR-ABL1 mRNA on an International Scale. <i>SpringerPlus</i> , 2016 , 5, 569		3
136	Optimal Monitoring of CML Treatment: Molecular and Mutation Analysis 2016 , 101-129		
135	BCR-ABL1 expression, RT-qPCR and treatment decisions in chronic myeloid leukaemia. <i>Journal of Clinical Pathology</i> , 2016 , 69, 817-21	3.9	7
134	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. <i>Blood</i> , 2016 , 128, 1219-1219	2.2	2
133	Upfront Imatinib with Selective Early Switching to Nilotinib Leads to Excellent Achievement of Deep Molecular Response in Chronic Phase CML: 5 Year (Final) Analysis of the TIDEL-II Study. <i>Blood</i> , 2016 , 128, 939-939	2.2	
132	Molecular monitoring in chronic myeloid leukemia-how low can you go?. <i>Hematology American Society of Hematology Education Program</i> , 2016 , 2016, 156-163	3.1	17
131	BAM-matcher: a tool for rapid NGS sample matching. <i>Bioinformatics</i> , 2016 , 32, 2699-701	7.2	22
130	Paper or plastic? BCR-ABL1 quantitation and mutation detection from dried blood spots. <i>Blood</i> , 2016 , 127, 2773-4	2.2	12
129	Compound mutations in BCR-ABL1 are not major drivers of primary or secondary resistance to ponatinib in CP-CML patients. <i>Blood</i> , 2016 , 127, 703-12	2.2	65
128	TIDEL-II: first-line use of imatinib in CML with early switch to nilotinib for failure to achieve time-dependent molecular targets. <i>Blood</i> , 2015 , 125, 915-23	2.2	65
127	KIR2DL5B genotype predicts outcomes in CML patients treated with response-directed sequential imatinib/nilotinib strategy. <i>Blood</i> , 2015 , 126, 2720-3	2.2	18

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126	A longitudinal evaluation of performance of automated BCR-ABL1 quantitation using cartridge-based detection system. <i>Pathology</i> , 2015 , 47, 570-4	1.6	9
125	A DNA real-time quantitative PCR method suitable for routine monitoring of low levels of minimal residual disease in chronic myeloid leukemia. <i>Journal of Molecular Diagnostics</i> , 2015 , 17, 185-92	5.1	19
124	Rare and Common Germline Variants Contribute to Occurrence of Myelodysplastic Syndrome. <i>Blood</i> , 2015 , 126, 1644-1644	2.2	1
123	BCR-ABL Assay Sensitivity of MR4.5 Achieved in >90%, and MR5 in >75% of Samples, through mRNA Selection before qRT-PCR. <i>Blood</i> , 2015 , 126, 2777-2777	2.2	2
122	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). <i>Blood</i> , 2015 , 126, 4029-4029	2.2	3
121	The Allosteric Inhibitor ABL001 Is Susceptible to Resistance in Vitro Mediated By Overexpression of the Drug Efflux Transporters ABCB1 and ABCG2. <i>Blood</i> , 2015 , 126, 4841-4841	2.2	2
120	High Incidence of Mutated Cancer-Associated Genes at Diagnosis in CML Patients with Early Transformation to Blast Crisis. <i>Blood</i> , 2015 , 126, 600-600	2.2	3
119	A Multi-Institutional Retrospective Analysis of Tyrosine Kinase Inhibitor (TKI) Clinical and Preclinical Efficacy According to BCR-ABL Mutation Status in CP-CML Patients. <i>Blood</i> , 2015 , 126, 2790-2790	2.2	O
118	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. <i>International Journal of Hematology</i> ,	2.3	35
117	2014 , 99, 616-24 Dynamics of chronic myeloid leukemia response to dasatinib, nilotinib, and high-dose imatinib. <i>Haematologica</i> , 2014 , 99, 1701-9	6.6	12
116	Prognosis for patients with CML and >10% BCR-ABL1 after 3 months of imatinib depends on the rate of BCR-ABL1 decline. <i>Blood</i> , 2014 , 124, 511-8	2.2	145
115	Many BCR-ABL1 compound mutations reported in chronic myeloid leukemia patients may actually be artifacts due to PCR-mediated recombination. <i>Blood</i> , 2014 , 124, 153-5	2.2	22
114	Deep molecular responses achieved in patients with CML-CP who are switched to nilotinib after long-term imatinib. <i>Blood</i> , 2014 , 124, 729-36	2.2	75
113	Implications of BCR-ABL1 kinase domain-mediated resistance in chronic myeloid leukemia. Leukemia Research, 2014 , 38, 10-20	2.7	97
112	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. <i>Blood</i> , 2014 , 124, 399-399	2.2	3
111	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. <i>Blood</i> , 2014 , 124, 4561-4	567	1
110	Aberrant Activation of Epidermal Growth Factor Receptor in MPN May Respond to the Kinase Inhibitor Gefitinib. <i>Blood</i> , 2014 , 124, 1882-1882	2.2	
109	Odk-1201, One-Step RT-qPCR Major BCR-ABL/ABL mRNA Kit for the International Scale, with High Sensitivity to Detect Deeper MR. <i>Blood</i> , 2014 , 124, 1805-1805	2.2	

108	Rapid initial decline in BCR-ABL1 is associated with superior responses to second-line nilotinib in patients with chronic-phase chronic myeloid leukemia. <i>BMC Cancer</i> , 2013 , 13, 173	4.8	13
107	Establishment and validation of analytical reference panels for the standardization of quantitative BCR-ABL1 measurements on the international scale. <i>Clinical Chemistry</i> , 2013 , 59, 938-48	5.5	38
106	A comparative analysis of algorithms for somatic SNV detection in cancer. <i>Bioinformatics</i> , 2013 , 29, 222	3 7 3 <u>2</u> 0	71
105	Early molecular response and female sex strongly predict stable undetectable BCR-ABL1, the criteria for imatinib discontinuation in patients with CML. <i>Blood</i> , 2013 , 121, 3818-24	2.2	123
104	Safety and efficacy of imatinib cessation for CML patients with stable undetectable minimal residual disease: results from the TWISTER study. <i>Blood</i> , 2013 , 122, 515-22	2.2	519
103	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). <i>Blood</i> , 2013 , 122, 652-652	2.2	6
102	PCR-Mediated Recombination Can Lead To Artificial Chimera Formation, Which May Pose As BCR-ABL1 Compound Mutations. <i>Blood</i> , 2013 , 122, 4014-4014	2.2	
101	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. <i>Blood</i> , 2013 , 122, 651-651	2.2	
100	Poor response to second-line kinase inhibitors in chronic myeloid leukemia patients with multiple low-level mutations, irrespective of their resistance profile. <i>Blood</i> , 2012 , 119, 2234-8	2.2	55
99	BCR-ABL1 doubling times more reliably assess the dynamics of CML relapse compared with the BCR-ABL1 fold rise: implications for monitoring and management. <i>Blood</i> , 2012 , 119, 4264-71	2.2	36
98	Harmonization of molecular monitoring of chronic myeloid leukemia therapy in Japan. <i>International Journal of Clinical Oncology</i> , 2012 , 17, 584-9	4.2	28
97	International reporting scale of BCR-ABL1 fusion transcript in chronic myeloid leukemia: first report from India. <i>Acta Haematologica</i> , 2012 , 127, 135-42	2.7	8
96	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. <i>Journal of Clinical Oncology</i> , 2012 , 30, 4323-9	2.2	78
95	Monitoring after successful therapy for chronic myeloid leukemia. <i>Hematology American Society of Hematology Education Program</i> , 2012 , 2012, 105-110	3.1	6
94	Monitoring disease response in chronic-phase chronic myeloid leukemia: the age of molecular assays?. <i>Hematology American Society of Hematology Education Program</i> , 2012 , 2012, 111-114	3.1	2
93	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. <i>Blood</i> , 2012 , 120, 3771-3771	2.2	4
92	Single Molecule Real Time (SMRT) Sequencing Sensitively Detects the Evolution of Polyclonal and Compound BCR-ABL Mutations in Patients Who Relapse On Kinase Inhibitor Therapy. <i>Blood</i> , 2012 , 120, 917-917	2.2	3
91	The patient@BCR-ABL1 Kinase Domain Mutation History Is Important for Decisions Regarding Tyrosine Kinase Inhibitor Therapy. <i>Blood</i> , 2012 , 120, 1692-1692	2.2	

(2010-2012)

90	Monitoring after successful therapy for chronic myeloid leukemia. <i>Hematology American Society of Hematology Education Program</i> , 2012 , 2012, 105-10	3.1	2
89	Monitoring disease response in chronic-phase chronic myeloid leukemia: the age of molecular assays?. <i>Hematology American Society of Hematology Education Program</i> , 2012 , 2012, 111-4	3.1	2
88	Mutational analysis in chronic myeloid leukemia: when and what to do?. <i>Current Opinion in Hematology</i> , 2011 , 18, 111-6	3.3	20
87	Molecular methods in diagnosis and monitoring of haematological malignancies. <i>Pathology</i> , 2011 , 43, 566-79	1.6	7
86	Dynamics of chronic myeloid leukemia response to long-term targeted therapy reveal treatment effects on leukemic stem cells. <i>Blood</i> , 2011 , 118, 1622-31	2.2	52
85	SHP-1 expression accounts for resistance to imatinib treatment in Philadelphia chromosome-positive cells derived from patients with chronic myeloid leukemia. <i>Blood</i> , 2011 , 118, 3634	1 -2 124	41
84	Chronic myelogenous leukemia: monitoring response to therapy. <i>Current Hematologic Malignancy Reports</i> , 2011 , 6, 75-81	4.4	4
83	Practical advice for determining the role of BCR-ABL mutations in guiding tyrosine kinase inhibitor therapy in patients with chronic myeloid leukemia. <i>Cancer</i> , 2011 , 117, 1800-11	6.4	61
82	Minimal residual disease: the advantages of digital over analog polymerase chain reaction. <i>Leukemia and Lymphoma</i> , 2011 , 52, 1161-3	1.9	8
81	Sensitive detection of BCR-ABL1 mutations in patients with chronic myeloid leukemia after imatinib resistance is predictive of outcome during subsequent therapy. <i>Journal of Clinical Oncology</i> , 2011 , 29, 4250-9	2.2	67
80	BCR-ABL transcript dynamics support the hypothesis that leukemic stem cells are reduced during imatinib treatment. <i>Clinical Cancer Research</i> , 2011 , 17, 6812-21	12.9	37
79	Imatinib Dose Interruption in Responding CML Patients Is Associated with Characteristic BCR-ABL Kinetics, Which Could Help to Differentiate Non-Adherence From Drug Resistance. <i>Blood</i> , 2011 , 118, 113-113	2.2	2
78	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,. <i>Blood</i> , 2011 , 118, 3770-3770	2.2	4
77	Complete Molecular Response (CMR) Rate with Nilotinib in Patients (pts) with Chronic Myeloid Leukemia in Chronic Phase (CML-CP) without CMR After IZ Years on Imatinib: Preliminary Results From the Randomized ENESTcmr Trial of Nilotinib 400 Mg Twice Daily (BID) Vs Imatinib. <i>Blood</i> ,	2.2	1
76	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. <i>Blood</i> , 2011 , 118, 111-111	2.2	
75	Establishment of the first World Health Organization International Genetic Reference Panel for quantitation of BCR-ABL mRNA. <i>Blood</i> , 2010 , 116, e111-7	2.2	120
74	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. <i>Journal of Clinical</i>	2.2	235
73	Oncology, 2010 , 28, 424-30 Practical considerations for monitoring patients with chronic myeloid leukemia. <i>Seminars in Hematology</i> , 2010 , 47, 327-34	4	7

72	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). <i>Blood</i> , 2010 , 116, 3758-65	2.2	382
71	Selective Escalation of Imatinib Therapy and Early Switching to Nilotinib In De Novo Chronic Phase CML Patients: Interim Results From the TIDEL-II Trial. <i>Blood</i> , 2010 , 116, 209-209	2.2	4
70	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. <i>Blood</i> , 2010 , 116, 889-889	2.2	4
69	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. <i>Blood</i> , 2010 , 116, 891-891	2.2	
68	Towards DNA-Based Monitoring of Therapy In Chronic Myeloid Leukemia. <i>Blood</i> , 2010 , 116, 2284-2284	2.2	
67	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 <i>Blood</i> , 2010 , 116, 3441-3441	2.2	
66	Selecting optimal second-line tyrosine kinase inhibitor therapy for chronic myeloid leukemia patients after imatinib failure: does the BCR-ABL mutation status really matter?. <i>Blood</i> , 2009 , 114, 5426	- 3.2	141
65	Impact of baseline BCR-ABL mutations on response to nilotinib in patients with chronic myeloid leukemia in chronic phase. <i>Journal of Clinical Oncology</i> , 2009 , 27, 4204-10	2.2	248
64	Genomic translocation breakpoint sequences are conserved in BCR-ABL1 cell lines despite the presence of amplification. <i>Cancer Genetics and Cytogenetics</i> , 2009 , 189, 138-9		5
63	A Phase I/II study of nilotinib in Japanese patients with imatinib-resistant or -intolerant Ph+ CML or relapsed/refractory Ph+ ALL. <i>International Journal of Hematology</i> , 2009 , 89, 679-88	2.3	32
62	Measuring minimal residual disease in chronic myeloid leukemia: fluorescence in situ hybridization and polymerase chain reaction. <i>Clinical Lymphoma and Myeloma</i> , 2009 , 9 Suppl 3, S266-71		8
61	Dasatinib treatment of chronic-phase chronic myeloid leukemia: analysis of responses according to preexisting BCR-ABL mutations. <i>Blood</i> , 2009 , 114, 4944-53	2.2	230
60	Monitoring disease response to tyrosine kinase inhibitor therapy in CML. <i>Hematology American Society of Hematology Education Program</i> , 2009 , 477-87	3.1	48
59	Maintaining Imatinib B00 Mg Daily in the First 12 Months of Chronic Phase CML Treatment Is Associated with Superior Event-Free Survival at 5 Years <i>Blood</i> , 2009 , 114, 1125-1125	2.2	1
58	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 <i>Blood</i> , 2009 , 114, 1130-1130	2.2	2
57	Early Dose-Escalation in Chronic Myeloid Leukaemia Patients with Low Plasma Imatinib Levels Leads to Equivalent BCR-ABL Values and Drug Levels at 6 Months to Those with Optimal Drug Levels: First Analysis From the TIDEL II Trial of De-Novo Patients Treated with 600mg Imatinib	2.2	3
56	Harmonization of Molecular Monitoring of CML Therapy in Europe IPerspective of Widespread Competence in BCR-ABL Quantification <i>Blood</i> , 2009 , 114, 2616-2616	2.2	1
55	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) <i>Blood</i> , 2009 , 114, 3292-3292	2.2	3

54	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) <i>Blood</i> , 2009 , 114, 337-337	2.2	11
53	Reduced Expression Level of SHP1 Gives An Additive Survival Advantage to the Ph+ Cells of Chronic Myeloid Leukemia (CML) Patients and Provides a Novel Pretreatment Predictor of Major Molecular Response Achievement in CML Patients <i>Blood</i> , 2009 , 114, 2212-2212	2.2	
52	Analysis of Molecular Data and the Emergence of Mutations for Chronic-Phase Chronic Myelogenous Leukemia (CML-CP) Patients Treated with Dasatinib After Imatinib Failure <i>Blood</i> , 2009 , 114, 3282-3282	2.2	
51	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. <i>Clinical Chemistry</i> , 2008 , 54, 1568-71	5.5	11
50	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. <i>Blood</i> , 2008 , 112, 3330-8	2.2	306
49	Impact of early dose intensity on cytogenetic and molecular responses in chronic-phase CML patients receiving 600 mg/day of imatinib as initial therapy. <i>Blood</i> , 2008 , 112, 3965-73	2.2	151
48	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 <i>Blood</i> , 2008 , 112, 1095-1095	2.2	5
47	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 <i>Blood</i> , 2008 , 112, 1102-1102	2.2	7
46	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 <i>Blood</i> , 2008 , 112, 1106-1106	2.2	1
45	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 <i>Blood</i> , 2008 , 112, 2113-2113	2.2	11
44	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. <i>Blood</i> , 2008 , 112, 331-331	2.2	2
43	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	2.2	12
42	DNA-Based Monitoring of Minimal Residual Disease(MRD) in Chronic Myeloid Leukemia(CML) Blood, 2008, 112, 1111-1111	2.2	1
41	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 <i>Blood</i> , 2008 , 112, 2124-2124	2.2	
40	Dasatinib induces significant hematologic and cytogenetic responses in patients with imatinib-resistant or -intolerant chronic myeloid leukemia in accelerated phase. <i>Blood</i> , 2007 , 109, 4143-	5 ² 0 ²	321
39	Efficacy and safety of imatinib in patients with chronic myeloid leukemia and complete or near-complete cytogenetic response to interferon-alpha. <i>Cancer</i> , 2007 , 110, 801-8	6.4	5
38	Chronic myeloid leukemia: molecular monitoring in clinical practice. <i>Hematology American Society of Hematology Education Program</i> , 2007 , 2007, 376-83	3.1	44
37	Sequential ABL kinase inhibitor therapy selects for compound drug-resistant BCR-ABL mutations with altered oncogenic potency. <i>Journal of Clinical Investigation</i> , 2007 , 117, 2562-9	15.9	315

36	Response: Reliability of PCR for BCR-ABL transcripts. <i>Blood</i> , 2007 , 109, 2263-2264	2.2	
35	Ph+ ALL: resistance seeds sown early. <i>Blood</i> , 2007 , 110, 472-472	2.2	8
34	Dasatinib induces complete hematologic and cytogenetic responses in patients with imatinib-resistant or -intolerant chronic myeloid leukemia in blast crisis. <i>Blood</i> , 2007 , 109, 3207-13	2.2	354
33	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. <i>Clinical Cancer Research</i> ,	12.9	116
32	Validation of the International Scale for Measurement of BCR-ABL by RQ-PCR Based on Deriving Laboratory-Specific Conversion Factors <i>Blood</i> , 2007 , 110, 1013-1013	2.2	2
31	An MMR Control RNA for Reliable Monitoring of BCR-ABL Transcripts in Treated CML Patients <i>Blood</i> , 2007 , 110, 2939-2939	2.2	
30	Monitoring Disease Response 2007 , 143-164		3
29	Detection of BCR-ABL mutations and resistance to imatinib mesylate. <i>Methods in Molecular Medicine</i> , 2006 , 125, 93-106		40
28	Diagnosis and monitoring of chronic myeloid leukemia by qualitative and quantitative RT-PCR. <i>Methods in Molecular Medicine</i> , 2006 , 125, 69-92		37
27	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. <i>Blood</i> , 2006 , 108, 28-37	2.2	977
26	Molecular monitoring of BCR-ABL as a guide to clinical management in chronic myeloid leukaemia. <i>Blood Reviews</i> , 2006 , 20, 29-41	11.1	116
25	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 <i>Blood</i> , 2006 , 108, 2175-2175	2.2	2
24	Increasing Frequency and Marked Stability of Complete Molecular Response Is Observed in Imatinib-Treated CML Patients with Long-Term Follow Up <i>Blood</i> , 2006 , 108, 430-430	2.2	8
23	Reverse Transcription with a Random Pentadecamer Primer Increases the Sensitivity of Quantitative PCR for BCR-ABL <i>Blood</i> , 2006 , 108, 2339-2339	2.2	1
22	Monitoring Chronic Myeloid Leukemia in 2006 2006 , 45-58		
21	In vitro sensitivity to imatinib-induced inhibition of ABL kinase activity is predictive of molecular response in patients with de novo CML. <i>Blood</i> , 2005 , 106, 2520-6	2.2	121
20	Dynamics of chronic myeloid leukaemia. <i>Nature</i> , 2005 , 435, 1267-70	50.4	667
19	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 <i>Blood</i> , 2005 , 106, 1079-1079	2.2	1

18	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 <i>Blood</i> , 2005 , 106, 163-163	2.2	13
17	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 <i>Blood</i> , 2005 , 106, 164-164	2.2	7
16	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 <i>Blood</i> , 2005 , 106, 437-437	2.2	2
15	Real-time quantitative PCR analysis can be used as a primary screen to identify patients with CML treated with imatinib who have BCR-ABL kinase domain mutations. <i>Blood</i> , 2004 , 104, 2926-32	2.2	206
14	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 <i>Blood</i> , 2004 , 104, 1008-1008	2.2	4
13	Comparison of "Log Reduction from Median Pretherapeutic Value" vs Ratio BCR-ABL/ABL to Express the Therapeutic Response in CML Patients <i>Blood</i> , 2004 , 104, 1013-1013	2.2	5
12	BCR-ABL Levels Continue To Decrease up to 42 Months after Commencement of Standard Dose Imatinib in Patients with Newly Diagnosed Chronic Phase CML Who Achieve a Major Molecular Response <i>Blood</i> , 2004 , 104, 274-274	2.2	7
11	The Frequency of Detection of BCR-ABL Mutations in Imatinib Treated Patients with Chronic Phase CML Who Attain a Complete Cytogenetic Response (CCR) Does Not Diminish with Increasing Duration of CCR but the Associated Loss of Response Is Usually Gradual <i>Blood</i> , 2004 , 104, 271-271	2.2	2
10	CML with E8A2 BCR-ABL Fusion: The Fourth Breakpoint Cluster Region? <i>Blood</i> , 2004 , 104, 1018-1018	2.2	
9	Detection of BCR-ABL mutations in patients with CML treated with imatinib is virtually always accompanied by clinical resistance, and mutations in the ATP phosphate-binding loop (P-loop) are associated with a poor prognosis. <i>Blood</i> , 2003 , 102, 276-83	2.2	635
8	Frequency of major molecular responses to imatinib or interferon alfa plus cytarabine in newly diagnosed chronic myeloid leukemia. <i>New England Journal of Medicine</i> , 2003 , 349, 1423-32	59.2	996
7	Molecular monitoring of chronic myeloid leukemia. Seminars in Hematology, 2003, 40, 62-8	4	73
6	Molecular monitoring of chronic myeloid leukemia. Seminars in Hematology, 2003, 40, 62-68	4	2
5	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. <i>British Journal of Haematology</i> , 2002 , 117, 875-7	4.5	21
4	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. <i>Blood</i> , 2002 , 99, 3472-5	2.2	582
3	Indirect sandwich enzyme-linked immunosorbent assay (ELISA) for plasma apolipoprotein E. <i>Annals of Clinical Biochemistry</i> , 1996 , 33 (Pt 2), 119-26	2.2	4
2	Reflotron Method for High-Density Lipoprotein Evaluated for Venous and Capillary Blood. <i>Clinical Chemistry</i> , 1992 , 38, 164-166	5.5	