Deborah L White

List of Publications by Year
in descending order

129 papers	6,520 citations	136885 32 h-index	66879 78 g-index
131 all docs	131 docs citations	131 times ranked	7247 citing authors

#	Article	IF	Citations
1	Targetable Kinase-Activating Lesions in Ph-like Acute Lymphoblastic Leukemia. New England Journal of Medicine, 2014, 371, 1005-1015.	13.9	1,161
2	BCRâ€“ABL1 lymphoblastic leukaemia is characterized by the deletion of Ikaros. Nature, 2008, 453, 110-114.	13.7	955
3	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
4	OCT-1â€“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
5	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
6	Dasatinib Cellular Uptake and Efflux in Chronic Myeloid Leukemia Cells: Therapeutic Implications. Clinical Cancer Research, 2008, 14, 3881-3888.	3.2	169
7	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
8	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
9	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
10	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
11	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
12	Monoclonal antibody targeting of IL-3 receptor Î± with CSL362 effectively depletes CML progenitor and stem cells. Blood, 2014, 123, 1218-1228.	0.6	89
13	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
14	Interaction of the Efflux Transporters ABCB1 and ABCG2 With Imatinib, Nilotinib, and Dasatinib. Clinical Pharmacology and Therapeutics, 2014, 95, 294-306.	2.3	75
15	Long-term treatment-free remission of chronic myeloid leukemia with falling levels of residual leukemic cells. Leukemia, 2018, 32, 2572-2579.	3.3	66
16	Chronic Myeloid Leukemia CD34+ cells have reduced uptake of imatinib due to low OCT-1 Activity. Leukemia, 2010, 24, 765-770.	3.3	64
17	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
18	Tyrosine kinase inhibitor resistance in chronic myeloid leukemia cell lines: investigating resistance pathways. Leukemia and Lymphoma, 2011, 52, 2139-2147.	0.6	57

#	Article	IF	Citations
19	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
20	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
21	High prevalence of relapse in children with Philadelphia-like acute lymphoblastic leukemia despite risk-adapted treatment. Haematologica, 2017, 102, e490-e493.	1.7	52
22	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
23	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
24	Blocking cytokine signaling along with intense Bcr-Abl kinase inhibition induces apoptosis in primary CML progenitors. Leukemia, 2010, 24, 771-778.	3.3	50
25	OCT1 and imatinib transport in CML: is it clinically relevant?. Leukemia, 2015, 29, 1960-1969.	3.3	49
26	Successful peripheral blood stem cell mobilisation with filgrastim in patients with chronic myeloid leukaemia achieving complete cytogenetic response with imatinib, without increasing disease burden as measured by quantitative real-time PCR. Leukemia, 2003, 17, 821-828.	3.3	44
27	Imatinib increases the intracellular concentration of nilotinib, which may explain the observed synergy between these drugs. Blood, 2007, 109, 3609-3610.	0.6	44
28	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
29	Lineage of measurable residual disease in patients with chronic myeloid leukemia in treatment-free remission. Leukemia, 2020, 34, 1052-1061.	3.3	39
30	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
31	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
32	TGF-Î± 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
33	The new allosteric inhibitor asciminib is susceptible to resistance mediated by ABCB1 and ABCG2 overexpression in vitro. Oncotarget, 2018, 9, 13423-13437.	0.8	37
34	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
35	BCR-ABL1 genomic DNA PCR response kinetics during first-line imatinib treatment of chronic myeloid leukemia. Haematologica, 2018, 103, 2026-2032.	1.7	31
36	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

#	Article	IF	Citations
37	KMT2A rearranged acute lymphoblastic leukaemia: Unravelling the genomic complexity and heterogeneity of this high-risk disease. Cancer Letters, 2020, 469, 410-418.	3.2	29
38	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
39	ABCB1 Overexpression Is a Key Initiator of Resistance to Tyrosine Kinase Inhibitors in CML Cell Lines. PLoS ONE, 2016, 11, e0161470.	1.1	28
40	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
41	KIR2DL5B genotype predicts outcomes in CML patients treated with response-directed sequential imatinib/nilotinib strategy. Blood, 2015, 126, 2720-2723.	0.6	27
42	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
43	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
44	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
45	Ponatinib is not transported by ABCB1, ABCG2 or OCT-1 in CML cells. Leukemia, 2015, 29, 1792-1794.	3.3	26
46	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
47	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
48	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
49	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
50	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
51	Enhancer retargeting of CDX2 and UBTF::ATXN7L3 define a subtype of high-risk B-progenitor acute lymphoblastic leukemia. Blood, 2022, 139, 3519-3531.	0.6	20
52	Contrasting effects of diclofenac and ibuprofen on active imatinib uptake into leukaemic cells. British Journal of Cancer, 2012, 106, 1772-1778.	2.9	19
53	Quantitative Phosphotyrosine Profiling of Patient-Derived Xenografts Identifies Therapeutic Targets in Pediatric Leukemia. Cancer Research, 2016, 76, 2766-2777.	0.4	16
54	CYP2C8 Genotype Significantly Alters Imatinib Metabolism in Chronic Myeloid Leukaemia Patients. Clinical Pharmacokinetics, 2017, 56, 977-985.	1.6	16

#	Article	IF	Citations
55	Outcomes for Australian children with relapsed/refractory acute lymphoblastic leukaemia treated with blinatumomab. Pediatric Blood and Cancer, 2021, 68, e28922.	0.8	16
56	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
57	The immunotoxicity, but not anti-tumor efficacy, of anti-CD40 and anti-CD137 immunotherapies is dependent on the gut microbiota. Cell Reports Medicine, 2021, 2, 100464.	3.3	15
58	Short-term intense Bcrâ€“Abl kinase inhibition with nilotinib is adequate to trigger cell death in BCR-ABL+ cells. Leukemia, 2009, 23, 1205-1206.	3.3	14
59	DUX Huntingâ€”Clinical Features and Diagnostic Challenges Associated with DUX4-Rearranged Leukaemia. Cancers, 2020, 12, 2815.	1.7	14
60	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
61	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
62	OCT-1 function varies with cell lineage but is not influenced by BCR-ABL. Haematologica, 2011, 96, 213-220.	1.7	13
63	Low GFI1 expression in white blood cells of CPâ€“CML patients at diagnosis is strongly associated with subsequent blastic transformation. Leukemia, 2013, 27, 1427-1430.	3.3	13
64	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
65	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
66	Increased peroxisome proliferator-activated receptor Î³ activity reduces imatinib uptake and efficacy in chronic myeloid leukemia mononuclear cells. Haematologica, 2017, 102, 843-853.	1.7	12
67	Modelling ponatinib resistance in tyrosine kinase inhibitor-naÃ¯ve and dasatinib resistant BCR-ABL1+ cell lines. Oncotarget, 2018, 9, 34735-34747.	0.8	12
68	Bâ€cell acute lymphoblastic leukaemia: recent discoveries in molecular pathology, their prognostic significance, and a review of the current classification. British Journal of Haematology, 2022, 197, 13-27.	1.2	12
69	Dasatinib targets chronic myeloid leukemia-CD34+ progenitors as effectively as it targets mature cells. Haematologica, 2013, 98, 896-900.	1.7	11
70	Modelling Predictors of Molecular Response to Frontline Imatinib for Patients with Chronic Myeloid Leukaemia. PLoS ONE, 2017, 12, e0168947.	1.1	11
71	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
72	JAK2 Alterations in Acute Lymphoblastic Leukemia: Molecular Insights for Superior Precision Medicine Strategies. Frontiers in Cell and Developmental Biology, 0, 10, .	1.8	11

#	Article	IF	Citations
73	OCT-1 as a Determinant of Response to Antileukemic Treatment. Clinical Pharmacology and Therapeutics, 2011, 89, 608-611.	2.3	10
74	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
75	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
76	<scp>MLLT10</scp> rearranged acute leukemia: Incidence, prognosis, and possible therapeutic strategies. Genes Chromosomes and Cancer, 2020, 59, 709-721.	1.5	10
77	Widespread Aberrant Alternative Splicing despite Molecular Remission in Chronic Myeloid Leukaemia Patients. Cancers, 2020, 12, 3738.	1.7	10
78	Acquired JAK2 mutations confer resistance to JAK inhibitors in cell models of acute lymphoblastic leukemia. Npj Precision Oncology, 2021, 5, 75.	2.3	10
79	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
80	COBL is a novel hotspot for IKZF1 deletions in childhood acute lymphoblastic leukemia. Oncotarget, 2016, 7, 53064-53073.	0.8	9
81	Multi-Cohort Transcriptomic Subtyping of B-Cell Acute Lymphoblastic Leukemia. International Journal of Molecular Sciences, 2022, 23, 4574.	1.8	9
82	Detection of minimal residual disease in an AML patient with trisomy 8 using interphase FISH. Pathology, 1997, 29, 289-293.	0.3	8
83	Predicting the response of CML patients to tyrosine kinase inhibitor therapy. Current Hematologic Malignancy Reports, 2009, 4, 59-65.	1.2	8
84	Predicting the Response of CML Patients to Tyrosine Kinase Inhibitor Therapy. Current Hematologic Malignancy Reports, 2011, 6, 88-95.	1.2	8
85	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
86	Constitutive JAK/STAT signaling is the primary mechanism of resistance to JAKi in TYK2-rearranged acute lymphoblastic leukemia. Cancer Letters, 2021, 512, 28-37.	3.2	8
87	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
88	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
89	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
90	Optimizing the selection of kinase inhibitors for chronic myeloid leukemia patients. Expert Review of Hematology, 2011, 4, 285-299.	1.0	6

#	Article	IF	Citations
91	Living with CML: is death no longer the end (point)?. Blood, 2015, 126, 2-4.	0.6	6
92	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
93	The expression of mature myeloid cell differentiation markers in acute leukemia. Pathology, 1987, 19, 137-142.	0.3	6
94	Elevated PTPN2 expression is associated with inferior molecular response in de-novo chronic myeloid leukaemia patients. Leukemia, 2014, 28, 702-705.	3.3	5
95	Precision medicine approaches may be the future for CRLF2 rearranged Down Syndrome Acute Lymphoblastic Leukaemia patients. Cancer Letters, 2018, 432, 69-74.	3.2	5
96	Azacytidine Sensitizes AML Cells for Effective Elimination By CD123 CAR T-Cells. Blood, 2019, 134, 3904-3904.	0.6	4
97	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
98	Two novel cases of NUTM1â€rearranged Bâ€cell acute lymphoblastic leukaemia presenting with highâ€risk features. British Journal of Haematology, 2022, 196, 1407-1411.	1.2	4
99	Selected CD34 blood cell allografts for older patients: low transplant-related mortality, graft failure and acute GvHD. Cytotherapy, 2003, 5, 534-541.	0.3	3
100	Highâ€risk Bâ€cell acute lymphoblastic leukaemia presenting with hypereosinophilia and acquiring a novel PAX5 fusion on relapse. British Journal of Haematology, 2020, 191, 301-304.	1.2	3
101	Genome-Wide Analysis of Genetic Alterations in Chronic Myelogenous Leukemia.. Blood, 2008, 112, 1089-1089.	0.6	3
102	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
103	Simvastatin enhances the efficacy of nilotinib in chronic myeloid leukaemia by post-translational modification and drug transporter modulation. Anti-Cancer Drugs, 2021, 32, 526-536.	0.7	2
104	CKLF and IL1B transcript levels at diagnosis are predictive of relapse in children with preâ€Bâ€cell acute lymphoblastic leukaemia. British Journal of Haematology, 2021, 193, 171-175.	1.2	2
105	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
106	Identification of a novel GOLGA4â€“JAK2 fusion gene in Bâ€cell acute lymphoblastic leukaemia. British Journal of Haematology, 2021, , .	1.2	2
107	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
108	Acquired Mutations within the JAK2 Kinase Domain Confer Resistance to JAK Inhibitors in an in Vitro model of a High-Risk Acute Lymphoblastic Leukemia. Blood, 2020, 136, 5-6.	0.6	2

#	Article	IF	Citations
109	Monocytoid switch in an adult with B-cell precursor acute lymphoblastic leukaemia characterised by the PAX5 P80R mutation. Pathology, 2022, 54, 631-634.	0.3	2
110	Case Report: Precision Medicine Target Revealed by In Vitro Modeling of Relapsed, Refractory Acute Lymphoblastic Leukemia From a Child With Neurofibromatosis. Frontiers in Oncology, 2022, 12, 851572.	1.3	2
111	Measurable residual disease analysis in paediatric acute lymphoblastic leukaemia patients with ABL-class fusions. British Journal of Cancer, 2022, 127, 908-915.	2.9	2
112	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
113	TYK2 Activating Alterations in Acute Lymphoblastic Leukemia: Novel Driver Oncogenes with Potential Avenues for Precision Medicine?. Journal of Cancer Science and Clinical Therapeutics, 2021, 05, .	0.2	1
114	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
115	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
116	Next Generation Genomic Analyses in T-ALL Patients Identify Recurrent and Novel Genomic Abnormalities. Blood, 2020, 136, 13-14.	0.6	1
117	TP53 lossâ€ofâ€function mutations reduce sensitivity of acute leukaemia to the curaxin CBL0137. Oncology Reports, 2022, 47, .	1.2	1
118	Is telomerase a player in chronic phase chronic myeloid leukemia, disease progression and imatinib resistance?. Leukemia and Lymphoma, 2008, 49, 1022-1023.	0.6	0
119	Imatinib trough levels in chronic myelogenous leukemia: does one dose fit all?. Leukemia and Lymphoma, 2011, 52, 165-167.	0.6	0
120	Response to â€˜Overexpression of ABCB1 as prediction marker for CML: How close we are to translation into clinics?â€™. Leukemia, 2017, 31, 769-770.	3.3	0
121	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 â€“ A TOPS Correlative Substudy. Blood, 2010, 116, 670-670.	0.6	0
122	Non-Steroidal Anti-Inflammatory Drugs and Imatinib; Drug Interactions That May Impact Efficacy,. Blood, 2011, 118, 3501-3501.	0.6	0
123	MicroRNA Dysregulation in Newly Diagnosed Chronic Myeloid Leukaemia Patients. Blood, 2013, 122, 4985-4985.	0.6	0
124	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
125	Divergent Evolutionary Trajectories of Erk- and Stat5-Activating Lesions in Acute Lymphoblastic Leukemia. Blood, 2018, 132, 568-568.	0.6	0
126	High Risk Genomic Alterations Identified at the Time of Diagnosis Are Strongly Associated with MRD and Subsequent Poor Outcomes in AYA ALL Patients Treated on a Pediatric Inspired Chemotherapy Regimen. Blood, 2019, 134, 3949-3949.	0.6	0

#	Article	IF	Citations
127	A Novel Role for HMGN1 in Down Syndrome Acute Lymphoblastic Leukemia. Blood, 2019, 134, 1462-1462.	0.6	0
128	Persistent Activation of JAK/STAT Signaling Plays an Important Role inin VitroJaki Resistance inTYK2-rearranged B-Cell Acute Lymphoblastic Leukaemia. Blood, 2020, 136, 3-3.	0.6	0
129	Inducible Knockout of HMGN1 in an In Vivo xenograft Model Reduces Down Syndrome Leukemic Burden and Increases Survival Outcomes. Blood, 2020, 136, 25-25.	0.6	0