Jamshid S Khorashad

List of Publications by Year
in descending order

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76 papers	3,645 citations	257450 24 h-index	133252 59 g-index
76 all docs	76 docs citations	76 times ranked	3560 citing authors

#	Article	IF	Citations
1	MS4A3 promotes differentiation in chronic myeloid leukemia by enhancing common Î²-chain cytokine receptor endocytosis. Blood, 2022, 139, 761-778.	1.4	7
2	Assessment of quantitative polymerase chain reaction for BCRâ€“ABL1 transcripts in chronic myeloid leukaemia: Are improved outcomes in patients with e14a2 transcripts an artefact ofÂtechnology?. British Journal of Haematology, 2022, 197, 52-62.	2.5	7
3	Identification of genetic targets in acute myeloid leukaemia for designing targeted therapy. British Journal of Haematology, 2021, 192, 137-145.	2.5	6
4	Qualification of tumour mutational burden by targeted nextâ€generation sequencing as a biomarker in hepatocellular carcinoma. Liver International, 2021, 41, 192-203.	3.9	32
5	Proteasome 26S subunit, non-ATPases 1 (PSMD1) and 3 (PSMD3), play an oncogenic role in chronic myeloid leukemia by stabilizing nuclear factor-kappa B. Oncogene, 2021, 40, 2697-2710.	5.9	20
6	SIRT5 Is a Druggable Metabolic Vulnerability in Acute Myeloid Leukemia. Blood Cancer Discovery, 2021, 2, 266-287.	5.0	37
7	Carfilzomib Enhances the Suppressive Effect of Ruxolitinib in Myelofibrosis. Cancers, 2021, 13, 4863.	3.7	1
8	A Role for the Bone Marrow Microenvironment in Drug Resistance of Acute Myeloid Leukemia. Cells, 2021, 10, 2833.	4.1	14
9	Genomic Abnormalities as Biomarkers and Therapeutic Targets in Acute Myeloid Leukemia. Cancers, 2021, 13, 5055.	3.7	4
10	TKI dose reduction can effectively maintain major molecular remission in patients with chronic myeloid leukaemia. British Journal of Haematology, 2021, 193, 346-355.	2.5	18
11	Applicability of Routine Targeted Next-generation Sequencing to Estimate Tumor Mutational Burden (TMB) in Patients Treated With Immune Checkpoint Inhibitor Therapy. Journal of Immunotherapy, 2020, 43, 53-56.	2.4	2
12	The KDR (VEGFR-2) Genetic Polymorphism Q472H and c-KIT Polymorphism M541L Are Associated With More Aggressive Behaviour in Astrocytic Gliomas. Cancer Genomics and Proteomics, 2020, 17, 715-727.	2.0	10
13	A British Society for Haematology Guideline on the diagnosis and management of chronic myeloid leukaemia. British Journal of Haematology, 2020, 191, 171-193.	2.5	38
14	An ex vivo investigation of interactions between primary acute myeloid leukaemia and mesenchymal stromal cells yields novel therapeutic targets. British Journal of Haematology, 2020, 190, e236-e239.	2.5	0
15	Prolonged treatment-free remission in chronic myeloid leukemia patients with previous BCR-ABL1 kinase domain mutations. Haematologica, 2020, 105, e225-e227.	3.5	7
16	Molecular Monitoring of Chronic Myeloid Leukemia. Methods in Molecular Biology, 2020, 2065, 153-173.	0.9	4
17	The influence of salivary amylase on total amylase elevation in CML patients treated with TKI therapy: a case series of 3 patients. Leukemia and Lymphoma, 2019, 60, 3333-3334.	1.3	2
18	Blast crisis of chronic myeloid leukemia with plasmacytoid dendritic cell phenotype associated with a rare fusion transcript, e13a3 BCRâ€“ABL1. Leukemia and Lymphoma, 2019, 60, 3090-3091.	1.3	1

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19	Somatic variants in epigenetic modifiers can predict failure of response to imatinib but not to second-generation tyrosine kinase inhibitors. Haematologica, 2019, 104, 2400-2409.	3.5	37
20	MR4 sustained for 12 months is associated with stable deep molecular responses in chronic myeloid leukemia. Haematologica, 2019, 104, 2206-2214.	3.5	10
21	The transcriptome of CMML monocytes is highly inflammatory and reflects leukemia-specific and age-related alterations. Blood Advances, 2019, 3, 2949-2961.	5.2	29
22	Nuclearâ€“Cytoplasmic Transport Is a Therapeutic Target in Myelofibrosis. Clinical Cancer Research, 2019, 25, 2323-2335.	7.0	24
23	Introducing a Predictive Score for Successful Treatment Free Remission in Chronic Myeloid Leukemia (CML). Blood, 2019, 134, 26-26.	1.4	8
24	NF-ÎºB-Dependent Activation of the Proteasome Components, PSMD1 and PSMD3, As a Mechanism of Resistance to Imatinib. Blood, 2019, 134, 2923-2923.	1.4	1
25	Alginate foam-based three-dimensional culture to investigate drug sensitivity in primary leukaemia cells. Journal of the Royal Society Interface, 2018, 15, 20170928.	3.4	11
26	Ongoing clonal evolution in chronic myelomonocytic leukemia on hypomethylating agents: a computational perspective. Leukemia, 2018, 32, 2049-2054.	7.2	4
27	Development of artificial bone marrow fibre scaffolds to study resistance to antiâ€leukaemia agents. British Journal of Haematology, 2018, 182, 924-927.	2.5	6
28	SIRT5 As a Therapeutic Target in Acute Myeloid Leukemia. Blood, 2018, 132, 907-907.	1.4	2
29	Dose Reduction of First and Second Generation TKIs Is Effective in the Maintenance of Major Molecular Response and May Predict Successful Tfr in CML Patients. Blood, 2018, 132, 3007-3007.	1.4	4
30	DNA-Based Digital PCR for the Quantification of Residual Disease in CML â€" Sensitivity or Specificity?. Blood, 2018, 132, 1738-1738.	1.4	0
31	"Function First" Screen of Primary AML Cells Identifies Common and Personalised Therapeutic Targets. Blood, 2018, 132, 1517-1517.	1.4	0
32	E14a2 BCR-ABL1 transcript is associated with a higher rate of treatment-free remission in individuals with chronic myeloid leukemia after stopping tyrosine kinase inhibitor therapy. Haematologica, 2017, 102, e297-e299.	3.5	42
33	Cognitive dysfunction after withdrawal of tyrosine kinase inhibitor therapy in chronic myeloid leukaemia. American Journal of Hematology, 2016, 91, E480-E481.	4.1	7
34	A phase II study of the efficacy, safety, and determinants of response to 5-azacitidine (VidazaÂ®) in patients with chronic myelomonocytic leukemia. Leukemia and Lymphoma, 2016, 57, 2441-2444.	1.3	20
35	shRNA library screening identifies nucleocytoplasmic transport as a mediator of BCR-ABL1 kinase-independent resistance. Blood, 2015, 125, 1772-1781.	1.4	41
36	Combined STAT3 and BCR-ABL1 inhibition induces synthetic lethality in therapy-resistant chronic myeloid leukemia. Leukemia, 2015, 29, 586-597.	7.2	111

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37	MS4A3 Improves Imatinib Response and Survival in BCR-ABL1 Primary TKI Resistance and in Blastic Transformation of Chronic Myeloid Leukemia. Blood, 2015, 126, 14-14.	1.4	2
38	BCR-ABL1 Compound Mutations Combining Key Kinase Domain Positions Confer Clinical Resistance to Ponatinib in Ph Chromosome-Positive Leukemia. Cancer Cell, 2014, 26, 428-442.	16.8	292
39	BCR-ABL1 compound mutations in tyrosine kinase inhibitorâ€“resistant CML: frequency and clonal relationships. Blood, 2013, 121, 489-498.	1.4	187
40	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. Cancer Research, 2013, 73, 5775-5786.	0.9	22
41	What challenges remain in chronic myeloid leukemia research?. Haematologica, 2013, 98, 1168-1172.	3.5	13
42	An Unbiased shRNA Library Screen Identifies Nucleocytoplasmic Transport As a Potential Target For Treatment Of Chronic Myeloid Leukemia. Blood, 2013, 122, 2707-2707.	1.4	1
43	New concepts for CML clonality. Oncotarget, 2013, 4, 7-8.	1.8	4
44	BP5-087, a Novel STAT3 Inhibitor, Combines With BCR-ABL1 Inhibition To Overcome Kinase-Independent Resistance In Chronic Myeloid Leukemia. Blood, 2013, 122, 854-854.	1.4	0
45	BCRâ€ABL1 kinase domain mutations: Methodology and clinical evaluation. American Journal of Hematology, 2012, 87, 298-304.	4.1	50
46	Selection of Therapy: Rational Decisions Based on Molecular Events. Hematology/Oncology Clinics of North America, 2011, 25, 1009-1023.	2.2	5
47	Poor adherence is the main reason for loss of CCyR and imatinib failure for chronic myeloid leukemia patients on long-term therapy. Blood, 2011, 117, 3733-3736.	1.4	292
48	Duplex quantitative PCR for molecular monitoring of BCRâ€ABL1â€associated hematological malignancies. American Journal of Hematology, 2011, 86, 313-315.	4.1	10
49	Partially or Fully BCR-ABL Independent Mechanisms of in Vitro Resistance to Ponatinib. Blood, 2011, 118, 2481-2481.	1.4	1
50	The Natural History of RTQ-PCR Levels After the Achievement of Complete Molecular Remission (CMR): Implications for â€˜Stopping' Studies. Blood, 2011, 118, 605-605.	1.4	6
51	Frequency and Clonality of BCR-ABL Compound Mutations in Chronic Myeloid Leukemia,. Blood, 2011, 118, 3744-3744.	1.4	0
52	EVI-1 oncogene expression predicts survival in chronic-phase CML patients resistant to imatinib treated with second-generation tyrosine kinase inhibitors. Blood, 2010, 116, 6014-6017.	1.4	29
53	Efficacy of tyrosine kinase inhibitors (TKIs) as third-line therapy in patients with chronic myeloid leukemia in chronic phase who have failed 2 prior lines of TKI therapy. Blood, 2010, 116, 5497-5500.	1.4	65
54	Early prediction of success or failure of treatment with second-generation tyrosine kinase inhibitors in patients with chronic myeloid leukemia. Haematologica, 2010, 95, 224-231.	3.5	112

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55	A gene expression signature of primary resistance to imatinib in chronic myeloid leukemia. Leukemia Research, 2010, 34, 254-257.	0.8	35
56	Adherence Is the Critical Factor for Achieving Molecular Responses in Patients With Chronic Myeloid Leukemia Who Achieve Complete Cytogenetic Responses on Imatinib. Journal of Clinical Oncology, 2010, 28, 2381-2388.	1.6	802
57	Variant Isoforms of BCR-ABL1 in Chronic Myelogenous Leukemia Reflect Alternative Splicing of ABL1 in Normal Tissue â€“ Letter. Molecular Cancer Therapeutics, 2010, 9, 2152-2152.	4.1	6
58	Response to Tyrosine Kinase Inhibitor Therapy In Patients Undergoing Allogeneic Hematopoietic Stem Cell Transplantation for Advanced Phase Chronic Myeloid Leukemia. Blood, 2010, 116, 3515-3515.	1.4	0
59	Analysis of BCR-ABL1 Tyrosine Kinase Domain Mutations In Primitive Chronic Myeloid Leukemia Cells Identifies a Unique Mutator Phenotype.. Blood, 2010, 116, 3397-3397.	1.4	0
60	The level of BCR-ABL1 kinase activity before treatment does not identify chronic myeloid leukemia patients who fail to achieve a complete cytogenetic response on imatinib. Haematologica, 2009, 94, 861-864.	3.5	12
61	Technical aspects and clinical applications of measuring BCRâ€ABL1 transcripts number in chronic myeloid leukemia. American Journal of Hematology, 2009, 84, 517-522.	4.1	40
62	Does a rise in the BCRâ€ABL1 transcript level identify chronic phase CML patients responding to imatinib who have a high risk of cytogenetic relapse?. British Journal of Haematology, 2009, 145, 373-375.	2.5	27
63	Long Term Adherence to Imatinib Therapy Is the Critical Factor for Achieving Molecular Responses in Chronic Myeloid Leukemia Patients.. Blood, 2009, 114, 3290-3290.	1.4	10
64	BCR-ABL1 Oncogene Down-regulates the Expression of OCT1 in CML.. Blood, 2009, 114, 3248-3248.	1.4	0
65	Pleural effusions in patients with chronic myeloid leukaemia treated with dasatinib may have an immuneâ€mediated pathogenesis. British Journal of Haematology, 2008, 141, 745-747.	2.5	132
66	Imatinib for Newly Diagnosed Patients With Chronic Myeloid Leukemia: Incidence of Sustained Responses in an Intention-to-Treat Analysis. Journal of Clinical Oncology, 2008, 26, 3358-3363.	1.6	524
67	Finding of Kinase Domain Mutations in Patients With Chronic Phase Chronic Myeloid Leukemia Responding to Imatinib May Identify Those at High Risk of Disease Progression. Journal of Clinical Oncology, 2008, 26, 4806-4813.	1.6	171
68	In vivo kinetics of kinase domain mutations in CML patients treated with dasatinib after failing imatinib. Blood, 2008, 111, 2378-2381.	1.4	85
69	Common Submicroscopic Genomic Imbalances Accompany the Ph Chromosome at Diagnosis in Chronic Myeloid Leukemia. Blood, 2008, 112, 3113-3113.	1.4	0
70	Long Term Durability of Major Molecular Responses for Patients Treated with Imatinib after Failure of Interferon-Alfa Is Equivalent to That of Patients Achieving Major Molecular Responses to Imatinib as Primary Therapy.. Blood, 2007, 110, 1037-1037.	1.4	2
71	Outcome, Prognostic Factors and Long-Term Follow-Up in 207 Chronic Phase CML Patients Receiving Front-Line Imatinib 400 mg at a Single Institution.. Blood, 2007, 110, 1045-1045.	1.4	1
72	Pleural Effusions Associated with Use of Dasatinib in Chronic Myeloid Leukemia May Have an Auto-Immune Pathogenesis.. Blood, 2007, 110, 2945-2945.	1.4	3

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73	For CML Patients in Chronic Phase Who Achieve a Cytogenetic Response to Imatinib the Finding of a BCR-ABL Mutation Predicts for Progression to Advanced Phase but It Has No Such Significance in Primary Resistance.. Blood, 2007, 110, 323-323.	1.4	6
74	Serial measurement of BCR-ABL transcripts in the peripheral blood after allogeneic stem cell transplantation for chronic myeloid leukemia: an attempt to define patients who may not require further therapy. Blood, 2006, 107, 4171-4176.	1.4	119
75	Abnormally Small BCR-ABL Transcripts in CML Patients before and during Imatinib Treatment.. Blood, 2006, 108, 2153-2153.	1.4	2
76	Imatinib preceding allogeneic stem cell transplantation in chronic myeloid leukemia. Haematologica, 2006, 91, 1145-6.	3.5	10