# Jamshid S Khorashad

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/5727472/publications.pdf

Version: 2024-02-01

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

| # | Article | IF | Citations |
|---|---|---|---|
| 1 | 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 |
| 2 | 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 |
| 3 | 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 |
| 4 | 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 |
| 5 | BCR-ABL1 compound mutations in tyrosine kinase inhibitorâ€“resistant CML: frequency and clonal relationships. Blood, 2013, 121, 489-498. | 1.4 | 187 |
| 6 | 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 |
| 7 | 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 |
| 8 | 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 |
| 9 | 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 |
| 10 | Combined STAT3 and BCR-ABL1 inhibition induces synthetic lethality in therapy-resistant chronic myeloid leukemia. Leukemia, 2015, 29, 586-597. | 7.2 | 111 |
| 11 | In vivo kinetics of kinase domain mutations in CML patients treated with dasatinib after failing imatinib. Blood, 2008, 111, 2378-2381. | 1.4 | 85 |
| 12 | 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 |
| 13 | BCRâ€ABL1 kinase domain mutations: Methodology and clinical evaluation. American Journal of Hematology, 2012, 87, 298-304. | 4.1 | 50 |
| 14 | 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 |
| 15 | shRNA library screening identifies nucleocytoplasmic transport as a mediator of BCR-ABL1 kinase-independent resistance. Blood, 2015, 125, 1772-1781. | 1.4 | 41 |
| 16 | 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 |
| 17 | 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 |
| 18 | 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 |

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|---|---|---|---|
| 19 | SIRT5 Is a Druggable Metabolic Vulnerability in Acute Myeloid Leukemia. Blood Cancer Discovery, 2021, 2, 266-287. | 5.0 | 37 |
| 20 | A gene expression signature of primary resistance to imatinib in chronic myeloid leukemia. Leukemia Research, 2010, 34, 254-257. | 0.8 | 35 |
| 21 | 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 |
| 22 | 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 |
| 23 | 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 |
| 24 | 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 |
| 25 | Nuclearâ€“Cytoplasmic Transport Is a Therapeutic Target in Myelofibrosis. Clinical Cancer Research, 2019, 25, 2323-2335. | 7.0 | 24 |
| 26 | KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. Cancer Research, 2013, 73, 5775-5786. | 0.9 | 22 |
| 27 | 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 |
| 28 | 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 |
| 29 | 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 |
| 30 | A Role for the Bone Marrow Microenvironment in Drug Resistance of Acute Myeloid Leukemia. Cells, 2021, 10, 2833. | 4.1 | 14 |
| 31 | What challenges remain in chronic myeloid leukemia research?. Haematologica, 2013, 98, 1168-1172. | 3.5 | 13 |
| 32 | 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 |
| 33 | 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 |
| 34 | Duplex quantitative PCR for molecular monitoring of BCRâ€ABL1â€associated hematological malignancies. American Journal of Hematology, 2011, 86, 313-315. | 4.1 | 10 |
| 35 | MR4 sustained for 12 months is associated with stable deep molecular responses in chronic myeloid leukemia. Haematologica, 2019, 104, 2206-2214. | 3.5 | 10 |
| 36 | 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 |

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|---|---|---|---|
| 37 | 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 |
| 38 | Imatinib preceding allogeneic stem cell transplantation in chronic myeloid leukemia. Haematologica, 2006, 91, 1145-6. | 3.5 | 10 |
| 39 | Introducing a Predictive Score for Successful Treatment Free Remission in Chronic Myeloid Leukemia (CML). Blood, 2019, 134, 26-26. | 1.4 | 8 |
| 40 | Cognitive dysfunction after withdrawal of tyrosine kinase inhibitor therapy in chronic myeloid leukaemia. American Journal of Hematology, 2016, 91, E480-E481. | 4.1 | 7 |
| 41 | Prolonged treatment-free remission in chronic myeloid leukemia patients with previous BCR-ABL1 kinase domain mutations. Haematologica, 2020, 105, e225-e227. | 3.5 | 7 |
| 42 | MS4A3 promotes differentiation in chronic myeloid leukemia by enhancing common Î²-chain cytokine receptor endocytosis. Blood, 2022, 139, 761-778. | 1.4 | 7 |
| 43 | 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 |
| 44 | 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 |
| 45 | 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 |
| 46 | Identification of genetic targets in acute myeloid leukaemia for designing targeted therapy. British Journal of Haematology, 2021, 192, 137-145. | 2.5 | 6 |
| 47 | 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 |
| 48 | 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 |
| 49 | Selection of Therapy: Rational Decisions Based on Molecular Events. Hematology/Oncology Clinics of North America, 2011, 25, 1009-1023. | 2.2 | 5 |
| 50 | Ongoing clonal evolution in chronic myelomonocytic leukemia on hypomethylating agents: a computational perspective. Leukemia, 2018, 32, 2049-2054. | 7.2 | 4 |
| 51 | Molecular Monitoring of Chronic Myeloid Leukemia. Methods in Molecular Biology, 2020, 2065, 153-173. | 0.9 | 4 |
| 52 | 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 |
| 53 | New concepts for CML clonality. Oncotarget, 2013, 4, 7-8. | 1.8 | 4 |
| 54 | Genomic Abnormalities as Biomarkers and Therapeutic Targets in Acute Myeloid Leukemia. Cancers, 2021, 13, 5055. | 3.7 | 4 |

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| 55 | 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 |
| 56 | 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 |
| 57 | 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 |
| 58 | SIRT5 As a Therapeutic Target in Acute Myeloid Leukemia. Blood, 2018, 132, 907-907. | 1.4 | 2 |
| 59 | Abnormally Small BCR-ABL Transcripts in CML Patients before and during Imatinib Treatment.. Blood, 2006, 108, 2153-2153. | 1.4 | 2 |
| 60 | 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 |
| 61 | 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 |
| 62 | 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 |
| 63 | Carfilzomib Enhances the Suppressive Effect of Ruxolitinib in Myelofibrosis. Cancers, 2021, 13, 4863. | 3.7 | 1 |
| 64 | 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 |
| 65 | Partially or Fully BCR-ABL Independent Mechanisms of in Vitro Resistance to Ponatinib. Blood, 2011, 118, 2481-2481. | 1.4 | 1 |
| 66 | 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 |
| 67 | NF-Î°B-Dependent Activation of the Proteasome Components, PSMD1 and PSMD3, As a Mechanism of Resistance to Imatinib. Blood, 2019, 134, 2923-2923. | 1.4 | 1 |
| 68 | 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 |
| 69 | Common Submicroscopic Genomic Imbalances Accompany the Ph Chromosome at Diagnosis in Chronic Myeloid Leukemia. Blood, 2008, 112, 3113-3113. | 1.4 | 0 |
| 70 | BCR-ABL1 Oncogene Down-regulates the Expression of OCT1 in CML.. Blood, 2009, 114, 3248-3248. | 1.4 | 0 |
| 71 | 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 |
| 72 | 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 |

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|---|---|---|---|
| 73 | Frequency and Clonality of BCR-ABL Compound Mutations in Chronic Myeloid Leukemia,. Blood, 2011, 118, 3744-3744. | 1.4 | 0 |
| 74 | 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 |
| 75 | DNA-Based Digital PCR for the Quantification of Residual Disease in CML â€" Sensitivity or Specificity?. Blood, 2018, 132, 1738-1738. | 1.4 | 0 |
| 76 | "Function First" Screen of Primary AML Cells Identifies Common and Personalised Therapeutic Targets. Blood, 2018, 132, 1517-1517. | 1.4 | 0 |