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List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	A common BIM deletion polymorphism mediates intrinsic resistance and inferior responses to tyrosine kinase inhibitors in cancer. Nature Medicine, 2012, 18, 521-528.	30.7	510
2	Effective and selective targeting of leukemia cells using a TORC1/2 kinase inhibitor. Nature Medicine, 2010, 16, 205-213.	30.7	329
3	Chemotherapy in malignant pleural mesothelioma. A review Journal of Clinical Oncology, 1996, 14, 1007-1017.	1.6	211
4	Targeting of the MNK–eIF4E axis in blast crisis chronic myeloid leukemia inhibits leukemia stem cell function. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, E2298-307.	7.1	132
5	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. Leukemia, 2019, 33, 1835-1850.	7.2	97
6	Physiologic hypoxia promotes maintenance of CML stem cells despite effective BCR-ABL1 inhibition. Blood, 2014, 123, 3316-3326.	1.4	87
7	Lymphadenopathy, splenomegaly, and altered immunoglobulin production in BCL3 transgenic mice. Oncogene, 1998, 16, 2333-2343.	5.9	70
8	Histone Deacetylase 3 Inhibition Overcomes <i>BIM</i> Deletion Polymorphism–Mediated Osimertinib Resistance in <i>EGFR-</i> Mutant Lung Cancer. Clinical Cancer Research, 2017, 23, 3139-3149.	7.0	69
9	The BCL2 inhibitor ABT-199 significantly enhances imatinib-induced cell death in chronic myeloid leukemia progenitors. Oncotarget, 2014, 5, 9033-9038.	1.8	56
10	Inhibition of Polysome Assembly Enhances Imatinib Activity against Chronic Myelogenous Leukemia and Overcomes Imatinib Resistance. Molecular and Cellular Biology, 2008, 28, 6496-6509.	2.3	55
11	Direct Cloning of DNA Sequences from the Common Fragile Site Region at Chromosome Band 3p14.2. Genomics, 1996, 35, 109-117.	2.9	52
12	An integrative model of pathway convergence in genetically heterogeneous blast crisis chronic myeloid leukemia. Blood, 2020, 135, 2337-2353.	1.4	49
13	A novel mechanism for Bcr-Abl action: Bcr-Abl-mediated induction of the eIF4F translation initiation complex and mRNA translation. Oncogene, 2007, 26, 1188-1200.	5.9	46
14	Phase I study of vorinostat with gefitinib in BIM deletion polymorphism/epidermal growth factor receptor mutation doubleâ€positive lung cancer. Cancer Science, 2020, 111, 561-570.	3.9	31
15	Precise localization of theFHIT gene to the common fragile site at 3p14.2 (FRA3B) and characterization of homozygous deletions within FRA3B that affectFHIT transcription in tumor cell lines. , 1997, 20, 16-23.		24
16	Identification of cis-Acting Elements and Splicing Factors Involved in the Regulation of BIM Pre-mRNA Splicing. PLoS ONE, 2014, 9, e95210.	2.5	21
17	A novel Bcr-Abl–mTOR–elF4A axis regulates IRES-mediated translation of LEF-1. Open Biology, 2014, 4, 140180.	3.6	21
18	Aberrant FHIT mRNA transcripts are present in malignant and normal haematopoiesis, but absence of FHIT protein is restricted to leukaemia. Oncogene, 1999, 18, 79-85.	5.9	20

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19	Overcoming imatinib resistance conferred by the <i>BIM</i> deletion polymorphism in chronic myeloid leukemia with splice-switching antisense oligonucleotides. Oncotarget, 2017, 8, 77567-77585.	1.8	18
20	The HDAC inhibitor SB939 overcomes resistance to BCR-ABL kinase Inhibitors conferred by the BIM deletion polymorphism in chronic myeloid leukemia. PLoS ONE, 2017, 12, e0174107.	2.5	17
21	Structure–Activity Relationship Studies of Mitogen Activated Protein Kinase Interacting Kinase (MNK) 1 and 2 and BCR-ABL1 Inhibitors Targeting Chronic Myeloid Leukemic Cells. Journal of Medicinal Chemistry, 2016, 59, 3063-3078.	6.4	16
22	The <i>BIM</i> deletion polymorphism: A paradigm of a permissive interaction between germline and acquired TKI resistance factors in chronic myeloid leukemia. Oncotarget, 2016, 7, 2721-2733.	1.8	16
23	A systematic review and meta-analysis of individual patient data on the impact of the BIM deletion polymorphism on treatment outcomes in epidermal growth factor receptor mutant lung cancer. Oncotarget, 2017, 8, 41474-41486.	1.8	13
24	Multiple joint effusions associated with high-dose imatinib therapy in a patient with chronic myelogenous leukaemia. European Journal of Haematology, 2006, 76, 444-446.	2.2	12
25	Viable Mice with Extensive Gene Humanization (25-kbp) Created Using Embryonic Stem Cell/Blastocyst and CRISPR/Zygote Injection Approaches. Scientific Reports, 2018, 8, 15028.	3.3	12
26	SRSF1 mediates cytokine-induced impaired imatinib sensitivity in chronic myeloid leukemia. Leukemia, 2020, 34, 1787-1798.	7.2	12
27	Integrating genetic and epigenetic factors in chronic myeloid leukemia risk assessment: toward gene expression-based biomarkers. Haematologica, 2022, 107, 358-370.	3.5	10
28	Expression profiling of a transformed thymocyte cell line undergoing maturation in vitro identifies multiple genes involved in positive selection. Cellular Immunology, 2003, 221, 64-79.	3.0	9
29	Multi-Agent Chemotherapy Overcomes Glucocorticoid Resistance Conferred by a BIM Deletion Polymorphism in Pediatric Acute Lymphoblastic Leukemia. PLoS ONE, 2014, 9, e103435.	2.5	9
30	The Role of Protein Phosphorylation in Therapy Resistance and Disease Progression in Chronic Myelogenous Leukemia. Progress in Molecular Biology and Translational Science, 2012, 106, 107-142.	1.7	8
31	Reply: The BIM deletion polymorphism cannot account for intrinsic TKI resistance of Chinese individuals with chronic myeloid leukemia. Nature Medicine, 2014, 20, 1090-1091.	30.7	8
32	The arginase inhibitor Nωâ~'hydroxyâ~'norâ~'arginine (norâ~'NOHA) induces apoptosis in leukemic cells specifically under hypoxic conditions but CRISPR/Cas9 excludes arginase 2 (ARG2) as the functional target. PLoS ONE, 2018, 13, e0205254.	2.5	8
33	RCA2: a scalable supervised clustering algorithm that reduces batch effects in scRNA-seq data. Nucleic Acids Research, 2021, 49, 8505-8519.	14.5	7
34	<i>BIM</i> deletion polymorphism profiling complements prognostic values of risk scores in imatinib-treated Asian chronic myeloid leukemia patients. Leukemia and Lymphoma, 2019, 60, 234-237.	1.3	5
35	THZ531 Induces a State of BRCAness in Multiple Myeloma Cells: Synthetic Lethality with Combination Treatment of THZ 531 with DNA Repair Inhibitors. International Journal of Molecular Sciences, 2022, 23, 1207.	4.1	4
36	The Genomic and Epigenomic Landscapes of Blast Crisis Transformation in Chronic Myeloid Leukemia. Blood, 2015, 126, 3737-3737.	1.4	3

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37	Resminostat, a histone deacetylase inhibitor, circumvents tolerance to EGFR inhibitors in EGFR-mutated lung cancer cells with <i>BIM</i> deletion polymorphism. Journal of Medical Investigation, 2020, 67, 343-350.	0.5	3
38	Validation and refinement of a RUNX1 mutation-associated gene expression signature in blast crisis chronic myeloid leukemia. Leukemia, 2022, 36, 892-896.	7.2	2
39	Targeting of a Novel MNK-elF4E-b-Catenin Axis in Blast Crisis Chronic Myelogenous Leukemia Inhibits Leukemia Stem Cell Function. Blood, 2011, 118, 963-963.	1.4	1
40	Dual Specific Inhibitors Of The BCR-ABL and MNK Kinases As Potential Therapeutics For Blast Crisis Chronic Myeloid Leukemia. Blood, 2013, 122, 2702-2702.	1.4	1
41	The BCL-2 Inhibitor ABT-199 Enhances Imatinib-Induced Cell Death In Chronic Phase CML Progenitors. Blood, 2013, 122, 3978-3978.	1.4	1
42	Molecular Mechanism of TKI Resistance and Potential Approaches to Overcome Resistance. , 2016, , 167-182.		1
43	Physiologic Hypoxia Promotes Maintenance of CML Stem Cells Despite Effective BCR-ABL Inhibition. Blood, 2011, 118, 450-450.	1.4	0
44	A Common Deletion Polymorphism in the BIM Gene Contributes to Intrinsic Imatinib Resistance in Chronic Myelogenous Leukemia. Blood, 2011, 118, 1666-1666.	1.4	0
45	The BIM Deletion Polymorphism: A Paradigm Of a Permissive Interaction Between Germline and Acquired TKI Resistance Factors In Chronic Myeloid Leukemia. Blood, 2013, 122, 3977-3977.	1.4	0
46	Multi-Agent Chemotherapy Overcomes Steroid Resistance Conferred by a BIM Deletion Polymorphism in Pediatric Acute Lymphoblastic Leukemia (ALL). Blood, 2013, 122, 2544-2544.	1.4	0
47	PML-RAR Binds to the +7kb Enhancer of CEBPE and Inhibits Its Expression. Blood, 2020, 136, 43-43.	1.4	0