

# Ruibao Ren

## List of Publications by Year in descending order

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55  
papers

3,277  
citations

304743

22  
h-index

182427

51  
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56  
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56  
docs citations

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times ranked

4235  
citing authors

#	ARTICLE	IF	CITATIONS
1	IRF4 and IRF8 expression are associated with clinical phenotype and clinico-hematological response to hydroxyurea in essential thrombocythemia. <i>Frontiers of Medicine</i> , 2022, 16, 403-415.	3.4	3
2	GNA13 regulates BCL2 expression and the sensitivity of GCB-DLBCL cells to BCL2 inhibitors in a palmitoylation-dependent manner. <i>Cell Death and Disease</i> , 2021, 12, 54.	6.3	17
3	Blinatumomab-induced T cell activation at single cell transcriptome resolution. <i>BMC Genomics</i> , 2021, 22, 145.	2.8	11
4	DDB1- and CUL4-associated factor 8 plays a critical role in spermatogenesis. <i>Frontiers of Medicine</i> , 2021, 15, 302-312.	3.4	4
5	The potential of cord blood to replenish young immune cells against cancer. <i>Aging and Cancer</i> , 2021, 2, 36-44.	1.6	0
6	Focal Adhesion Kinase (FAK) Inhibition Synergizes with KRAS G12C Inhibitors in Treating Cancer through the Regulation of the FAK-YAP Signaling. <i>Advanced Science</i> , 2021, 8, e2100250.	11.2	28
7	A dual inhibitor overcomes drug-resistant FLT3-ITD acute myeloid leukemia. <i>Journal of Hematology and Oncology</i> , 2021, 14, 105.	17.0	18
8	IRF8 Impacts Self-Renewal of Hematopoietic Stem Cells by Regulating TLR9 Signaling Pathway of Innate Immune Cells. <i>Advanced Science</i> , 2021, 8, e2101031.	11.2	4
9	ARHGEF12 regulates erythropoiesis and is involved in erythroid regeneration after chemotherapy in acute lymphoblastic leukemia patients. <i>Haematologica</i> , 2020, 105, 925-936.	3.5	19
10	PTPN2 regulates the activation of KRAS and plays a critical role in proliferation and survival of KRAS-driven cancer cells. <i>Journal of Biological Chemistry</i> , 2020, 295, 18343-18354.	3.4	11
11	Combination therapy of BCR-ABL-positive B cell acute lymphoblastic leukemia by tyrosine kinase inhibitor dasatinib and c-JUN N-terminal kinase inhibition. <i>Journal of Hematology and Oncology</i> , 2020, 13, 80.	17.0	12
12	Low-dose decitabine priming with intermediate-dose cytarabine followed by umbilical cord blood infusion as consolidation therapy for elderly patients with acute myeloid leukemia: a phase II single-arm study. <i>BMC Cancer</i> , 2019, 19, 819.	2.6	8
13	Cooperation of Dnmt3a R878H with Nras G12D promotes leukemogenesis in knock-in mice: a pilot study. <i>BMC Cancer</i> , 2019, 19, 1072.	2.6	4
14	Application of next-generation sequencing technology to precision medicine in cancer: joint consensus of the Tumor Biomarker Committee of the Chinese Society of Clinical Oncology. <i>Cancer Biology and Medicine</i> , 2019, 16, 189.	3.0	16
15	Zfyve16 regulates the proliferation of B-lymphoid cells. <i>Frontiers of Medicine</i> , 2018, 12, 559-565.	3.4	4
16	A Novel Microtubule Inhibitor Overcomes Multidrug Resistance in Tumors. <i>Cancer Research</i> , 2018, 78, 5949-5957.	0.9	18
17	Patient's T Cell Functionality Determines Blinatumomab-Mediated Killing of B-ALL Leukemia Cells. <i>Blood</i> , 2018, 132, 4079-4079.	1.4	0
18	N-Arachidonoyl Dopamine Inhibits NRAS Neoplastic Transformation by Suppressing Its Plasma Membrane Translocation. <i>Molecular Cancer Therapeutics</i> , 2017, 16, 57-67.	4.1	13

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19	Targeting BRK-Positive Breast Cancers with Small-Molecule Kinase Inhibitors. <i>Cancer Research</i> , 2017, 77, 175-186.	0.9	22
20	BCR/ABL can promote CD19+ cell growth but not render them long-term stemness. <i>Stem Cell Investigation</i> , 2016, 3, 85-85.	3.0	5
21	Roles of palmitoylation and the KKK membrane-targeting motif in leukemogenesis by oncogenic KRAS4A. <i>Journal of Hematology and Oncology</i> , 2015, 8, 132.	17.0	20
22	WT1 Recruits TET2 to Regulate Its Target Gene Expression and Suppress Leukemia Cell Proliferation. <i>Molecular Cell</i> , 2015, 57, 662-673.	9.7	242
23	IRF8 Regulates Cell Cycle of Hematopoietic Stem Cells. <i>Blood</i> , 2015, 126, 2353-2353.	1.4	3
24	Palmitoyl Acyltransferase DHHC9 Is Required for Efficient Induction of Leukemia By Oncogenic NRAS. <i>Blood</i> , 2014, 124, 893-893.	1.4	1
25	The role of RAS effectors in BCR/ABL induced chronic myelogenous leukemia. <i>Frontiers of Medicine</i> , 2013, 7, 452-461.	3.4	9
26	IRF-4 Suppresses BCR/ABL Transformation of Myeloid Cells in a DNA Binding-independent Manner. <i>Journal of Biological Chemistry</i> , 2012, 287, 1770-1778.	3.4	9
27	Cooperation between deficiencies of IRF-4 and IRF-8 promotes both myeloid and lymphoid tumorigenesis. <i>Blood</i> , 2010, 116, 2759-2767.	1.4	30
28	Palmitoylation of oncogenic NRAS is essential for leukemogenesis. <i>Blood</i> , 2010, 115, 3598-3605.	1.4	72
29	Dominant Negative Effect of Palmitoylation-Deficient NRAS In Suppression of BCR/ABL Leukemogenesis. <i>Blood</i> , 2010, 116, 3157-3157.	1.4	2
30	Proteasome Inhibition Causes Regression of Leukemia and Abrogates BCR-ABL-Induced Evasion of Apoptosis in Part through Regulation of Forkhead Tumor Suppressors. <i>Cancer Research</i> , 2009, 69, 6546-6555.	0.9	50
31	Effect of Ras Inhibition in Hematopoiesis and BCR/ABL Leukemogenesis. <i>Journal of Hematology and Oncology</i> , 2008, 1, 5.	17.0	22
32	IRF-4 functions as a tumor suppressor in early B-cell development. <i>Blood</i> , 2008, 112, 3798-3806.	1.4	47
33	Ubp43 regulates BCR-ABL leukemogenesis via the type 1 interferon receptor signaling. <i>Blood</i> , 2007, 110, 305-312.	1.4	45
34	Oncogenic <i>NRAS</i> , <i>KRAS</i> , and <i>HRAS</i> Exhibit Different Leukemogenic Potentials in Mice. <i>Cancer Research</i> , 2007, 67, 7139-7146.	0.9	76
35	IRF-4 Functions as a Tumor Suppressor in Early Stages of B Cell Development.. <i>Blood</i> , 2007, 110, 154-154.	1.4	0
36	Models of hematopoietic malignancies: chronic myeloid leukemia. <i>Drug Discovery Today: Disease Models</i> , 2006, 3, 183-189.	1.2	2

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37	Oncogenic NRAS rapidly and efficiently induces CMML- and AML-like diseases in mice. <i>Blood</i> , 2006, 108, 2349-2357.	1.4	79
38	Mechanisms of BCR-ABL in the pathogenesis of chronic myelogenous leukaemia. <i>Nature Reviews Cancer</i> , 2005, 5, 172-183.	28.4	896
39	Modeling the dosage effect of oncogenes in leukemogenesis. <i>Current Opinion in Hematology</i> , 2004, 11, 25-34.	2.5	20
40	Loss of IRF-4 Exacerbates the CML-Like Phenotype of IRF-8 Knockout Mice.. <i>Blood</i> , 2004, 104, 2952-2952.	1.4	0
41	c-CBL is not required for leukemia induction by Bcr-Abl in mice. <i>Oncogene</i> , 2003, 22, 8852-8860.	5.9	13
42	Localization of BCR-ABL to F-actin regulates cell adhesion but does not attenuate CML development. <i>Blood</i> , 2003, 102, 2220-2228.	1.4	51
43	Overriding BCR/ABL mitotic signal by ICSBP-induced differentiation. <i>Blood</i> , 2003, 102, 4251-4252.	1.4	1
44	Dissecting the Molecular Mechanism of Chronic Myelogenous Leukemia Using Murine Models. <i>Leukemia and Lymphoma</i> , 2002, 43, 1549-1561.	1.3	6
45	The coiled-coil domain and Tyr177 of bcr are required to induce a murine chronic myelogenous leukemia-like disease by bcr/abl. <i>Blood</i> , 2002, 99, 2957-2968.	1.4	105
46	The molecular mechanism of chronic myelogenous leukemia and its therapeutic implications: studies in a murine model. <i>Oncogene</i> , 2002, 21, 8629-8642.	5.9	38
47	The SH2 domain of Bcr-Abl is not required to induce a murine myeloproliferative disease; however, SH2 signaling influences disease latency and phenotype. <i>Blood</i> , 2001, 97, 277-287.	1.4	32
48	Cooperation of BCR-ABL and AML1/MDS1/EVI1 in blocking myeloid differentiation and rapid induction of an acute myelogenous leukemia. <i>Oncogene</i> , 2001, 20, 8236-8248.	5.9	72
49	The NH 2 -Terminal Coiled-Coil Domain and Tyrosine 177 Play Important Roles in Induction of a Myeloproliferative Disease in Mice by Bcr-Abl. <i>Molecular and Cellular Biology</i> , 2001, 21, 840-853.	2.3	111
50	Bcr-Abl has a greater intrinsic capacity than v-Abl to induce the neoplastic expansion of myeloid cells. <i>Oncogene</i> , 2000, 19, 6286-6296.	5.9	18
51	Expression of Interferon Consensus Sequence Binding Protein (ICSBP) Is Downregulated in Bcr-Abl-Induced Murine Chronic Myelogenous Leukemia-Like Disease, and Forced Coexpression of ICSBP Inhibits Bcr-Abl-Induced Myeloproliferative Disorder. <i>Molecular and Cellular Biology</i> , 2000, 20, 1149-1161.	2.3	137
52	Polarized distribution of Bcr-Abl in migrating myeloid cells and co-localization of Bcr-Abl and its target proteins. <i>Oncogene</i> , 1999, 18, 1165-1176.	5.9	29
53	Bcr-Abl with an SH3 Deletion Retains the Ability To Induce a Myeloproliferative Disease in Mice, yet c-Abl Activated by an SH3 Deletion Induces Only Lymphoid Malignancy. <i>Molecular and Cellular Biology</i> , 1999, 19, 6918-6928.	2.3	63
54	Bcr-Abl Efficiently Induces a Myeloproliferative Disease and Production of Excess Interleukin-3 and Granulocyte-Macrophage Colony-Stimulating Factor in Mice: A Novel Model for Chronic Myelogenous Leukemia. <i>Blood</i> , 1998, 92, 3829-3840.	1.4	263

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55	Activation of the c-Abl tyrosine kinase in the stress response to DMA-damaging agents. <i>Nature</i> , 1995, 376, 785-788.	27.8	496