

# Mohammad Azam

## List of Publications by Year in descending order

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Version: 2024-02-01

22  
papers

314  
citations

1163117

8  
h-index

1199594

12  
g-index

22  
all docs

22  
docs citations

22  
times ranked

678  
citing authors

#	ARTICLE	IF	CITATIONS
1	Momelotinib is a highly potent inhibitor of FLT3-mutant AML. <i>Blood Advances</i> , 2022, 6, 1186-1192.	5.2	10
2	PLaCatinG AML1-ETO. <i>Blood</i> , 2022, 139, 959-961.	1.4	2
3	Adaptive responses to <i>mTOR</i> gene targeting in hematopoietic stem cells reveal a proliferative mechanism evasive to mTOR inhibition. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2021, 118, .	7.1	19
4	Momelotinib Is a Highly Potent Inhibitor of FLT3-Mutant AML. <i>Blood</i> , 2021, 138, 206-206.	1.4	0
5	SWATH-Proteomics of Ibrutinib's Action in Myeloid Leukemia Initiating Mutated CSFR Signaling. <i>Proteomics - Clinical Applications</i> , 2020, 14, e1900144.	1.6	16
6	Methods for Evaluating the Role of c-Fos and Dusp1 in Oncogene Dependence. <i>Journal of Visualized Experiments</i> , 2019, , .	0.3	0
7	Phospho serine and threonine analysis of normal and mutated granulocyte colony stimulating factor receptors. <i>Scientific Data</i> , 2019, 6, 21.	5.3	29
8	Targeted Inhibition of the Dual Specificity Phosphatases DUSP1 and DUSP6 Suppress MPNST Growth via JNK. <i>Clinical Cancer Research</i> , 2019, 25, 4117-4127.	7.0	53
9	The high NRF2 expression confers chemotherapy resistance partly through up-regulated DUSP1 in myelodysplastic syndromes. <i>Haematologica</i> , 2019, 104, 485-496.	3.5	25
10	Expansion of EPOR-negative macrophages besides erythroblasts by elevated EPOR signaling in erythrocytosis mouse models. <i>Haematologica</i> , 2018, 103, 40-50.	3.5	30
11	Loss of DUSP1 Is Synthetic Lethal to JAK2V617F. <i>Blood</i> , 2018, 132, 51-51.	1.4	0
12	DUSP1 Confers Oncogene Dependence in CSF3R Induced Leukemia. <i>Blood</i> , 2018, 132, 1341-1341.	1.4	0
13	Targeting c-FOS and DUSP1 abrogates intrinsic resistance to tyrosine-kinase inhibitor therapy in BCR-ABL-induced leukemia. <i>Nature Medicine</i> , 2017, 23, 472-482.	30.7	75
14	MEK/ERK addiction in CNL/aCML. <i>Oncotarget</i> , 2017, 8, 99215-99216.	1.8	5
15	Compound Heterozygosity of Two Novel JAK2 Mutations in Hereditary Essential Thrombocythemia Implicates Important Monomer-Monomer Interactions in Thrombopoiesis Signaling. <i>Blood</i> , 2016, 128, 3137-3137.	1.4	2
16	A Common Signaling Node Constitute Non-Oncogene Addiction in Kinase Driven Leukemia: Mechanism of Oncogene Addiction in CML. <i>Blood</i> , 2016, 128, 3056-3056.	1.4	0
17	Enhanced MAPK Signaling Constitute Non-Oncogene Addiction in CSF3R Induced Leukemia. <i>Blood</i> , 2016, 128, 632-632.	1.4	0
18	Targeting substrate-site in Jak2 kinase prevents emergence of genetic resistance. <i>Scientific Reports</i> , 2015, 5, 14538.	3.3	45

#	ARTICLE	IF	CITATIONS
19	A Method for Screening and Validation of Resistant Mutations Against Kinase Inhibitors. Journal of Visualized Experiments, 2014, , .	0.3	3
20	Overcoming Drug Resistance Of FLT3-ITD By SAR302503. Blood, 2013, 122, 5023-5023.	1.4	0
21	Targeting the Genetic Resistance of JAK2 and BCR/ABL by TG101348. Blood, 2012, 120, 3765-3765.	1.4	0
22	Mechanisms of Drug Resistance Against JAK2 Inhibitors. Blood, 2011, 118, 1426-1426.	1.4	0