

Gary W Reuther

List of Publications by Year in descending order

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Version: 2024-02-01

20
papers

502
citations

759233

12
h-index

888059

17
g-index

21
all docs

21
docs citations

21
times ranked

902
citing authors

#	ARTICLE	IF	CITATIONS
1	Transformation of hematopoietic cells and activation of JAK2-V617F by IL-27R, a component of a heterodimeric type I cytokine receptor. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2007, 104, 18502-18507.	7.1	55
2	CRLF2 and JAK2 in B-Progenitor Acute Lymphoblastic Leukemia: A Novel Association in Oncogenesis. <i>Cancer Research</i> , 2010, 70, 7347-7352.	0.9	52
3	Structural Insights into JAK2 Inhibition by Ruxolitinib, Fedratinib, and Derivatives Thereof. <i>Journal of Medicinal Chemistry</i> , 2021, 64, 2228-2241.	6.4	49
4	JAK2-V617F-mediated signalling is dependent on lipid rafts and statins inhibit JAK2-V617F-dependent cell growth. <i>British Journal of Haematology</i> , 2013, 160, 177-187.	2.5	40
5	Potent Dual BET Bromodomain-Kinase Inhibitors as Value-Added Multitargeted Chemical Probes and Cancer Therapeutics. <i>Molecular Cancer Therapeutics</i> , 2017, 16, 1054-1067.	4.1	40
6	HDAC11 deficiency disrupts oncogene-induced hematopoiesis in myeloproliferative neoplasms. <i>Blood</i> , 2020, 135, 191-207.	1.4	40
7	Preclinical characterization of INCB053914, a novel pan-PIM kinase inhibitor, alone and in combination with anticancer agents, in models of hematologic malignancies. <i>PLoS ONE</i> , 2018, 13, e0199108.	2.5	39
8	The PIM inhibitor AZD1208 synergizes with ruxolitinib to induce apoptosis of ruxolitinib sensitive and resistant JAK2-V617F-driven cells and inhibit colony formation of primary MPN cells. <i>Oncotarget</i> , 2015, 6, 40141-40157.	1.8	35
9	ALK-Activating Homologous Mutations in LTK Induce Cellular Transformation. <i>PLoS ONE</i> , 2012, 7, e31733.	2.5	32
10	Activation of JAK2-V617F by Components of Heterodimeric Cytokine Receptors. <i>Journal of Biological Chemistry</i> , 2010, 285, 16651-16663.	3.4	28
11	Aggressive myeloid leukemia formation is directed by the Musashi 2/Numb pathway. <i>Cancer Biology and Therapy</i> , 2010, 10, 979-982.	3.4	26
12	The pan-PIM inhibitor INCB053914 displays potent synergy in combination with ruxolitinib in models of MPN. <i>Blood Advances</i> , 2019, 3, 3503-3514.	5.2	22
13	Finding a Jill for JAK: Assessing Past, Present, and Future JAK Inhibitor Combination Approaches in Myelofibrosis. <i>Cancers</i> , 2020, 12, 2278.	3.7	15
14	JAK2 activation in myeloproliferative neoplasms: A potential role for heterodimeric receptors. <i>Cell Cycle</i> , 2008, 7, 714-719.	2.6	11
15	JAK2 inhibitor persistence in MPN: uncovering a central role of ERK activation. <i>Blood Cancer Journal</i> , 2022, 12, 13.	6.2	9
16	Metabolic Vulnerabilities and Epigenetic Dysregulation in Myeloproliferative Neoplasms. <i>Frontiers in Immunology</i> , 2020, 11, 604142.	4.8	5
17	RMC-4550, an Allosteric Inhibitor of SHP2, Displays Therapeutic Efficacy in Pre-Clinical Models of Myeloproliferative Neoplasms. <i>Blood</i> , 2019, 134, 4198-4198.	1.4	3
18	A Component of a Heterodimeric Receptor Can Functionally Replace a Homodimeric Receptor in Supporting the Activation and Transforming Activity of JAK2-V617F. <i>Blood</i> , 2008, 112, 3732-3732.	1.4	0

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19	Activation of JAK2-V617F by Heterodimeric Receptor Components.. Blood, 2009, 114, 2955-2955.	1.4	0
20	Single Molecule Dual JAK2-BET Inhibition As an MPN Therapeutic. Blood, 2015, 126, 2826-2826.	1.4	0