Niccol Bartalucci

List of Publications by Year in Descending Order

36 papers	743 citations	13 h-index	27 g-index
44 ext. papers	970 ext. citations	5.6 avg, IF	3.42 L-index

#	Paper	IF	Citations
36	Nanopore sequencing for the screening of myeloid and lymphoid neoplasms with eosinophilia and rearrangement of PDGFR, PDGFR, FGFR1 or PCM1-JAK2. *Biomarker Research*, **2021**, 9, 83	8	
35	Peripheral Nerve Resident Macrophages and Schwann Cells Mediate Cancer-Induced Pain. *Cancer Research*, **2021**, 81, 3387-3401	10.1	7
34	Activated IL-6 signaling contributes to the pathogenesis of, and is a novel therapeutic target for, CALR-mutated MPNs. *Blood Advances*, **2021**, 5, 2184-2195	7.8	2
33	Polycythemia vera: the current status of preclinical models and therapeutic targets. *Expert Opinion on Therapeutic Targets*, **2020**, 24, 615-628	6.4	1
32	Characteristics and clinical correlates of NFE2 mutations in chronic Myeloproliferative neoplasms. *American Journal of Hematology*, **2020**, 95, E23-E26	7.1	3
31	RAS/CBL mutations predict resistance to JAK inhibitors in myelofibrosis and are associated with poor prognostic features. *Blood Advances*, **2020**, 4, 3677-3687	7.8	17
30	NanoR: A user-friendly R package to analyze and compare nanopore sequencing data. *PLoS ONE*, **2019**, 14, e0216471	3.7	7
29	Nano-GLADIATOR: real-time detection of copy number alterations from nanopore sequencing data. *Bioinformatics*, **2019**, 35, 4213-4221	7.2	6
28	Calreticulin Ins5 and Del52 mutations impair unfolded protein and oxidative stress responses in K562 cells expressing CALR mutants. *Scientific Reports*, **2019**, 9, 10558	4.9	12
27	Long Reads, Short Time: Feasibility of Prenatal Sample Karyotyping by Nanopore Genome Sequencing. *Clinical Chemistry*, **2019**, 65, 1605-1608	5.5	1
26	GIPSS: genetically inspired prognostic scoring system for primary myelofibrosis. *Leukemia*, **2018**, 32, 1631-1642	10.7	117
25	Calreticulin Affects Hematopoietic Stem/Progenitor Cell Fate by Impacting Erythroid and Megakaryocytic Differentiation. *Stem Cells and Development*, **2018**, 27, 225-236	4.4	9
24	Involvement of MAF/SPP1 axis in the development of bone marrow fibrosis in PMF patients. *Leukemia*, **2018**, 32, 438-449	10.7	15
23	Familial dominant epilepsy and mild pachygyria associated with a constitutional LIS1 mutation. *American Journal of Medical Genetics, Part A*, **2018**, 176, 2808-2812	2.5	2
22	Calreticulin Ins5 and Del52 Mutations Impair Unfolded Protein and Oxidative Stress Responses in Hematopoietic Cells. *Blood*, **2018**, 132, 4332-4332	2.2	0
21	Large Genomic Alterations Occurring in the Transition from Chronic to Blast Phase of Chronic Myeloproliferative Neoplasms. *Blood*, **2018**, 132, 3028-3028	2.2	
20	Mutation landscape in patients with myelofibrosis receiving ruxolitinib or hydroxyurea. *Blood Cancer Journal*, **2018**, 8, 122	7	14

19	Role of TGF-β1/miR-382-5p/SOD2 axis in the induction of oxidative stress in CD34+ cells from primary myelofibrosis. *Molecular Oncology*, **2018**, 12, 2102-2123	7.9	13
18	Endothelial-to-Mesenchymal Transition in Bone Marrow and Spleen of Primary Myelofibrosis. *American Journal of Pathology*, **2017**, 187, 1879-1892	5.8	12
17	Inhibitors of the PI3K/mTOR pathway prevent STAT5 phosphorylation in mutated cells through PP2A/CIP2A axis. *Oncotarget*, **2017**, 8, 96710-96724	3.3	22
16	Tie2 Expressing Monocytes in the Spleen of Patients with Primary Myelofibrosis. *PLoS ONE*, **2016**, 11, e0156990	3.7	2
15	A data-driven network model of primary myelofibrosis: transcriptional and post-transcriptional alterations in CD34+ cells. *Blood Cancer Journal*, **2016**, 6, e439	7	12
14	Evaluation of plitidepsin in patients with primary myelofibrosis and post polycythemia vera/essential thrombocythemia myelofibrosis: results of preclinical studies and a phase II clinical trial. *Blood Cancer Journal*, **2015**, 5, e286	7	4
13	Rationale for combination therapies in myelofibrosis **2015**, 136-150		
12	Complete Inhibition of STAT5 Phosphorylation Is Achieved By Combination of JAK1/2 and PI3K/mTOR Inhibitors in in Vitro and In Vivo MPN Models. *Blood*, **2015**, 126, 2824-2824	2.2	
11	Calreticulin mutation-specific immunostaining in myeloproliferative neoplasms: pathogenetic insight and diagnostic value. *Leukemia*, **2014**, 28, 1811-8	10.7	65
10	Preclinical models for drug selection in myeloproliferative neoplasms. *Current Hematologic Malignancy Reports*, **2013**, 8, 317-24	4.4	2
9	Rationale for targeting the PI3K/Akt/mTOR pathway in myeloproliferative neoplasms. *Clinical Lymphoma, Myeloma and Leukemia*, **2013**, 13 Suppl 2, S307-9	2	32
8	Co-targeting the PI3K/mTOR and JAK2 signalling pathways produces synergistic activity against myeloproliferative neoplasms. *Journal of Cellular and Molecular Medicine*, **2013**, 17, 1385-96	5.6	68
7	mTOR inhibitors alone and in combination with JAK2 inhibitors effectively inhibit cells of myeloproliferative neoplasms. *PLoS ONE*, **2013**, 8, e54826	3.7	71
6	The PI3K Specific Inhibitor BKM120 Results Effective and Synergizes With Ruxolitinib In Preclinical Models Of Myeloproliferative Neoplasms. *Blood*, **2013**, 122, 1599-1599	2.2	
5	Safety and efficacy of everolimus, a mTOR inhibitor, as single agent in a phase 1/2 study in patients with myelofibrosis. *Blood*, **2011**, 118, 2069-76	2.2	126
4	Hydroxyurea does not appreciably reduce JAK2 V617F allele burden in patients with polycythemia vera or essential thrombocythemia. *Haematologica*, **2010**, 95, 1435-8	6.6	37
3	Characterization of Targets of Plitidepsin In JAK2V617F-Mutated Cells From Myeloproliferative Neoplasms. *Blood*, **2010**, 116, 4093-4093	2.2	
2	The JAK2V617 mutation induces constitutive activation and agonist hypersensitivity in basophils from patients with polycythemia vera. *Haematologica*, **2009**, 94, 1537-45	6.6	48

1	No role for CXCL12-G801A polymorphism in the development of extramedullary disease in acute myeloid leukemia. *Leukemia*, **2008**, 22, 669-71	10.7	7