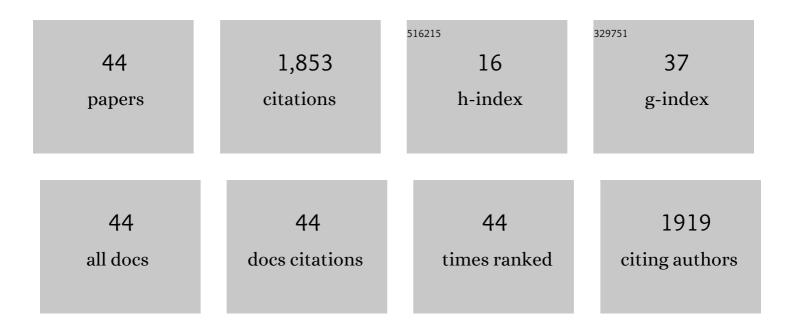
## Laura Villani

List of Publications by Year in descending order

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Ι λιισλ Μιιλινι

#	Article	IF	CITATIONS
1	Reduced CXCR4-expression on CD34-positive blood cells predicts outcomes of persons with primary myelofibrosis. Leukemia, 2021, 35, 468-475.	3.3	7
2	Gene expression profile correlates with molecular and clinical features in patients with myelofibrosis. Blood Advances, 2021, 5, 1452-1462.	2.5	8
3	Impact of the rs1024611 Polymorphism of CCL2 on the Pathophysiology and Outcome of Primary Myelofibrosis. Cancers, 2021, 13, 2552.	1.7	9
4	Clinical Relevance of VEGFA (rs3025039) +936 C>T Polymorphism in Primary Myelofibrosis: Susceptibility, Clinical Co-Variates, and Outcomes. Genes, 2021, 12, 1271.	1.0	4
5	Primary myelofibrosis: rs2010963 VEGFA polymorphism favors a prefibrotic phenotype and is associated with higher risk of thrombosis. Leukemia Research, 2021, 111, 106730.	0.4	3
6	VEGFA rs3025020 Polymorphism Contributes to CALR-Mutation Susceptibility and Is Associated with Low Risk of Deep Vein Thrombosis in Primary Myelofibrosis. TH Open, 2021, 05, e513-e520.	0.7	1
7	Plasma sIL-2Rα levels are associated with disease progression in myelofibrosis with JAK2V617F but not CALR mutation. Leukemia Research, 2020, 90, 106319.	0.4	7
8	Constitutive STAT5 phosphorylation in CD34+ cells of patients with primary myelofibrosis: Correlation with driver mutation status and disease severity. PLoS ONE, 2019, 14, e0220189.	1.1	3
9	Homozygosity for -2518 G Allele Variant of MCP-1 Predisposes to Adverse Presentation and Outcome in Primary Myelofibrosis. Blood, 2019, 134, 1689-1689.	0.6	0
10	Elevated Plasma sIL-2Rα Levels in Primary Myelofibrosis Play a Distinct Role on Disease Progression in JAK2V617F and Calr Mutants. Blood, 2019, 134, 1678-1678.	0.6	0
11	The spleen of patients with myelofibrosis harbors defective mesenchymal stromal cells. American Journal of Hematology, 2018, 93, 615-622.	2.0	8
12	Increased STAT5/STAT3 Intracellular Signaling in Circulating CD34+ Cells of Patients with PMF Correlates with Disease Severity. Blood, 2018, 132, 4337-4337.	0.6	0
13	Primary myelofibrosis: Older age and high JAK2V617F allele burden are associated with elevated plasma high-sensitivity C-reactive protein levels and a phenotype of progressive disease. Leukemia Research, 2017, 60, 18-23.	0.4	27
14	Reduced frequency of circulating CD4+CD25brightCD127lowFOXP3+ regulatory T cells in primary myelofibrosis. Blood, 2016, 128, 1660-1662.	0.6	13
15	Increased plasma nicotinamide phosphoribosyltransferase is associated with a hyperproliferative phenotype and restrains disease progression in MPNâ€associated myelofibrosis. American Journal of Hematology, 2016, 91, 709-713.	2.0	6
16	JAK2V617F allele burden ⩾50% is associated with response to ruxolitinib in persons with MPN-associated myelofibrosis and splenomegaly requiring therapy. Leukemia, 2016, 30, 1772-1775.	3.3	50
17	High Levels of High Sensitivity-C Reactive Protein (hs-CRP) Are Associated with Older Age, Chromosomal Abnormalities and JAK2V617F Mutation with High Allele Burden in Primary Myelofibrosis (PMF). Blood, 2016, 128, 1956-1956.	0.6	1
18	Tie2 Expressing Monocytes in the Spleen of Patients with Primary Myelofibrosis. PLoS ONE, 2016, 11, e0156990.	1.1	3

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19	Deregulated Genes in Hematopoietic Stem Cells Isolated from Spleen of Patients with Myelofibrosis. Blood, 2016, 128, 4279-4279.	0.6	0
20	Abnormal expression patterns of <i>WT1-as, MEG3</i> and <i>ANRIL</i> long non-coding RNAs in CD34+ cells from patients with primary myelofibrosis and their clinical correlations. Leukemia and Lymphoma, 2015, 56, 492-496.	0.6	14
21	JAK2 Exon 14 Skipping in Patients with Primary Myelofibrosis: A Minor Splice Variant Modulated by the JAK2-V617F Allele Burden. PLoS ONE, 2015, 10, e0116636.	1.1	8
22	V617FJAK2-Positive Endothelial Cells Are Present in Bone Marrow Neovessels of Patients with Myelofibrosis and Could Derive from the Transdifferentiation of Mutated Hematopoietic Cells. Blood, 2015, 126, 2833-2833.	0.6	0
23	Clinical effect of driver mutations of JAK2, CALR, or MPL in primary myelofibrosis. Blood, 2014, 124, 1062-1069.	0.6	340
24	Functional and genetic aberrations of in vitro-cultured marrow-derived mesenchymal stromal cells of patients with classical Philadelphia-negative myeloproliferative neoplasms. Leukemia, 2014, 28, 1742-1745.	3.3	30
25	What Is the True Response Rate to Ruxolitinib in Persons with Myeloproliferative Neoplasm (MPN)-Associated Myelofibrosis (MF) Needing Therapy for Splenomegaly ?. Blood, 2014, 124, 3191-3191.	0.6	15
26	Phenotypical, Functional and Genetic Characterization of Mesenchymal Stem Cells Derived from the Spleen of Patients with Myelofibrosis. Blood, 2014, 124, 3227-3227.	0.6	0
27	No association between the XPD Lys751Cln (rs13181) polymorphism and disease phenotype or leukemic transformation in primary myelofibrosis. Haematologica, 2013, 98, e83-e84.	1.7	4
28	Spleen endothelial cells from patients with myelofibrosis harbor the JAK2V617F mutation. Blood, 2013, 121, 360-368.	0.6	102
29	JAK2 V617F Genotype Is a Strong Determinant of Blast Transformation in Primary Myelofibrosis. PLoS ONE, 2013, 8, e59791.	1.1	15
30	A3669G polymorphism of glucocorticoid receptor is a susceptibility allele for primary myelofibrosis and contributes to phenotypic diversity and blast transformation. Blood, 2012, 120, 3112-3117.	0.6	33
31	Evidence that Prefibrotic Myelofibrosis Is Aligned along a Clinical and Biological Continuum Featuring Primary Myelofibrosis. PLoS ONE, 2012, 7, e35631.	1.1	85
32	JAK2 46/1 haplotype predisposes to splanchnic vein thrombosis-associated BCR-ABL negative classic myeloproliferative neoplasms. Leukemia Research, 2012, 36, e7-e9.	0.4	17
33	JAK2 V617F Genotype Is a Strong Determinant of Blast Transformation in Primary Myelofibrosis Blood, 2012, 120, 2829-2829.	0.6	Ο
34	Evaluation of the bioactive and total transforming growth factor β1 levels in primary myelofibrosis. Cytokine, 2011, 53, 100-106.	1.4	29
35	EZH2 mutational status predicts poor survival in myelofibrosis. Blood, 2011, 118, 5227-5234.	0.6	242
36	Prefibrotic Myelofibrosis (PreMF) Belongs to a Continuum of Epidemiological, Clinical and Histological Characteristics Featuring Primary Myelofibrosis (PMF). Blood, 2011, 118, 1743-1743.	0.6	1

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37	Prognostic Impact of EZH2 and ASXL1 Mutation in Myelofibrosis. Blood, 2011, 118, 2811-2811.	0.6	4
38	High Frequency of Endothelial Colony Forming Cells Marks a Non-Active Myeloproliferative Neoplasm with High Risk of Splanchnic Vein Thrombosis. PLoS ONE, 2010, 5, e15277.	1.1	30
39	Serum hepcidin: a novel diagnostic tool in disorders of iron metabolism. Haematologica, 2009, 94, 1631-1633.	1.7	23
40	Endothelial colony-forming cells from patients with chronic myeloproliferative disorders lack the disease-specific molecular clonality marker. Blood, 2009, 114, 3127-3130.	0.6	79
41	MPL and JAK2 exon 12 mutations in patients with the Budd-Chiari syndrome or extrahepatic portal vein obstruction. Blood, 2008, 111, 4418-4418.	0.6	30
42	Characteristics and clinical correlates of MPL 515W>L/K mutation in essential thrombocythemia. Blood, 2008, 112, 844-847.	0.6	216
43	JAK2 V617F mutational status predicts progression to large splenomegaly and leukemic transformation in primary myelofibrosis. Blood, 2007, 110, 4030-4036.	0.6	233
44	Anaemia characterises patients with myelofibrosis harbouring MplW515L/Kmutation. British Journal of Haematology, 2007, 137, 244-247.	1.2	153