Tomas Stopka

List of Publications by Year in descending order

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75 papers	2,453 citations	257450 24 h-index	197818 49 g-index
75 all docs	75 docs citations	75 times ranked	4050 citing authors

#	Article	IF	Citations
1	Angiotensin II stimulates proliferation of normal early erythroid progenitors.. Journal of Clinical Investigation, 1997, 100, 2310-2314.	8.2	231
2	The ISWI ATPase Snf2h is required for early mouse development. Proceedings of the National Academy of Sciences of the United States of America, 2003, 100, 14097-14102.	7.1	178
3	Modifiers of epigenetic reprogramming show paternal effects in the mouse. Nature Genetics, 2007, 39, 614-622.	21.4	154
4	The role of PU.1 and GATA-1 transcription factors during normal and leukemogenic hematopoiesis. Leukemia, 2010, 24, 1249-1257.	7.2	151
5	Oncogenic MicroRNAs: miR-155, miR-19a, miR-181b, and miR-24 enable monitoring of early breast cancer in serum. BMC Cancer, 2014, 14, 448.	2.6	149
6	PU.1 inhibits the erythroid program by binding to GATA-1 on DNA and creating a repressive chromatin structure. EMBO Journal, 2005, 24, 3712-3723.	7.8	138
7	MYB transcriptionally regulates the miR-155 host gene in chronic lymphocytic leukemia. Blood, 2011, 117, 3816-3825.	1.4	128
8	Expression patterns of microRNAs associated with CML phases and their disease related targets. Molecular Cancer, 2011, 10, 41.	19.2	124
9	Two New EPO Receptor Mutations: Truncated EPO Receptors Are Most Frequently Associated With Primary Familial and Congenital Polycythemias. Blood, 1997, 90, 2057-2061.	1.4	116
10	TRAIL (Apo2L) suppresses growth of primary human leukemia and myelodysplasia progenitors. Leukemia, 2002, 16, 67-73.	7.2	108
11	Regulation of Î±A-crystallin via Pax6, c-Maf, CREB and a broad domain of lens-specific chromatin. EMBO Journal, 2006, 25, 2107-2118.	7.8	93
12	PU.1 and pRB Interact and Cooperate To Repress GATA-1 and Block Erythroid Differentiation. Molecular and Cellular Biology, 2003, 23, 7460-7474.	2.3	87
13	Epigenetic silencing of the oncogenic miR-17-92 cluster during PU.1-directed macrophage differentiation. EMBO Journal, 2011, 30, 4450-4464.	7.8	85
14	Snf2h-mediated chromatin organization and histone H1 dynamics govern cerebellar morphogenesis and neural maturation. Nature Communications, 2014, 5, 4181.	12.8	71
15	Next-generation deep sequencing improves detection of BCR-ABL1 kinase domain mutations emerging under tyrosine kinase inhibitor treatment of chronic myeloid leukemia patients in chronic phase. Journal of Cancer Research and Clinical Oncology, 2015, 141, 887-899.	2.5	67
16	Role of Epigenetics in Chronic Myeloid Leukemia. Current Hematologic Malignancy Reports, 2013, 8, 28-36.	2.3	52
17	Ribosomal Protein S19 Gene Mutations in Patients with Diamond-Blackfan Anemia and Identification of Ribosomal Protein S19 Pseudogenes. Blood Cells, Molecules, and Diseases, 2000, 26, 124-132.	1.4	44
18	Chromatin remodeling enzyme Snf2h regulates embryonic lens differentiation and denucleation. Development (Cambridge), 2016, 143, 1937-1947.	2.5	41

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19	Inhibition of Smad5 in Human Hematopoietic Progenitors Blocks Erythroid Differentiation Induced by BMP4. Blood Cells, Molecules, and Diseases, 2002, 28, 221-233.	1.4	39
20	The ISWI ATPase Smarca5 (Snf2h) Is Required for Proliferation and Differentiation of Hematopoietic Stem and Progenitor Cells. Stem Cells, 2017, 35, 1614-1623.	3.2	37
21	GATA-1 directly regulates p21 gene expression during erythroid differentiation. Cell Cycle, 2010, 9, 1972-1980.	2.6	36
22	Plasma miR-155, miR-203, and miR-205 are Biomarkers for Monitoring of Primary Cutaneous T-Cell Lymphomas. International Journal of Molecular Sciences, 2017, 18, 2136.	4.1	33
23	BCR-ABL1 mediated miR-150 downregulation through MYC contributed to myeloid differentiation block and drug resistance in chronic myeloid leukemia. Haematologica, 2018, 103, 2016-2025.	3.5	30
24	Prediction Potential of Serum miR-155 and miR-24 for Relapsing Early Breast Cancer. International Journal of Molecular Sciences, 2017, 18, 2116.	4.1	27
25	Epigenetic Control of SPI1 Gene by CTCF and ISWI ATPase SMARCA5. PLoS ONE, 2014, 9, e87448.	2.5	25
26	PU.1 Activation Relieves GATA-1â€“Mediated Repression of Cebpa and Cbfb during Leukemia Differentiation. Molecular Cancer Research, 2009, 7, 1693-1703.	3.4	22
27	The chromatin remodeler Snf2h is essential for oocyte meiotic cell cycle progression. Genes and Development, 2020, 34, 166-178.	5.9	21
28	Loss of ISWI ATPase SMARCA5 (SNF2H) in Acute Myeloid Leukemia Cells Inhibits Proliferation and Chromatid Cohesion. International Journal of Molecular Sciences, 2020, 21, 2073.	4.1	19
29	Oncogenic microRNA-155 and its target PU.1: an integrative gene expression study in six of the most prevalent lymphomas. International Journal of Hematology, 2015, 102, 441-450.	1.6	17
30	GATA-1 Inhibits PU.1 Gene via DNA and Histone H3K9 Methylation of Its Distal Enhancer in Erythroleukemia. PLoS ONE, 2016, 11, e0152234.	2.5	17
31	Chromatin remodeling and SWI/SNF2 factors in human disease. Frontiers in Bioscience - Landmark, 2008, Volume, 6126.	3.0	14
32	Paraproteinemic keratopathy associated with monoclonal gammopathy of undetermined significance (<scp>MGUS</scp>): clinical findings in twelve patients including recurrence after keratoplasty. Acta Ophthalmologica, 2019, 97, e987-e992.	1.1	13
33	Diamond blackfan anemia stem cells fail to repopulate erythropoiesis in NOD/SCID mice. Blood Cells, Molecules, and Diseases, 2003, 31, 93-97.	1.4	10
34	ISWI ATPase Smarca5 Regulates Differentiation of Thymocytes Undergoing Î²-Selection. Journal of Immunology, 2019, 202, 3434-3446.	0.8	10
35	Ribosomal proteins S3a, S13, S16, and S24 are not mutated in patients with Diamond-Blackfan anemia. Blood, 2001, 97, 579-580.	1.4	8
36	Distinct and overlapping DNMT1 interactions with multiple transcription factors in erythroid cells: Evidence for co-repressor functions. Biochimica Et Biophysica Acta - Gene Regulatory Mechanisms, 2016, 1859, 1515-1526.	1.9	8

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37	Somatic mutation dynamics in MDS patients treated with azacitidine indicate clonal selection in patients-responders. Oncotarget, 2017, 8, 111966-111978.	1.8	8
38	Aberrantly elevated suprabasin in the bone marrow as a candidate biomarker of advanced disease state in myelodysplastic syndromes. Molecular Oncology, 2020, 14, 2403-2419.	4.6	7
39	Disruption of a Functional Relationship Between PU.1 and Mir-155 during the Pathogenesis of Chronic Lymphocytic Leukemia (CLL). Blood, 2008, 112, 3148-3148.	1.4	5
40	Analysis of 5-Azacytidine Resistance Models Reveals a Set of Targetable Pathways. Cells, 2022, 11, 223.	4.1	5
41	Lenalidomide treatment in lower risk myelodysplastic syndromesâ€"The experience of a Czech hematology center. (Positive effect of erythropoietin Â± prednisone addition to lenalidomide in) Tj ETQq1 1 0.784314 rgBT /Overlock	1.4	4
42	MicroRNA Mir-155 and Myb Proto-Oncogene Family Members Cooperate in Pathogenesis of Chronic Lymphocytic Leukemia.. Blood, 2009, 114, 58-58.	1.4	3
43	Nuclear localization of ISWI ATPase Smarca5 Snf2h in mouse. Frontiers in Bioscience - Elite, 2009, E1, 553-559.	1.8	3
44	Chromatin Remodeler Smarca5 Is Required for Cancer-Related Processes of Primary Cell Fitness and Immortalization. Cells, 2022, 11, 808.	4.1	3
45	Combined Approach to Leukemic Differentiation Using Transcription Factor PU.1-Enhancing Agents. International Journal of Molecular Sciences, 2022, 23, 6729.	4.1	3
46	Chromothripsis in High-Risk Myelodysplastic Syndromes: Incidence, Genetic Features, Clinical Implications, and Impact on Survival of Patients Treated with Azacytidine (Data from Czech MDS) Tj ETQq0 0 0 rgBT /Overlock 10 Tf 50	1.4	2
47	Circulating microRNAs in Cerebrospinal Fluid and Plasma: Sensitive Tool for Detection of Secondary CNS Involvement, Monitoring of Therapy and Prediction of CNS Relapse in Aggressive B-NHL Lymphomas. Cancers, 2022, 14, 2305.	3.7	2
48	G-CSF plus azacitidine versus azacitidine alone for patients with high-risk myelodysplastic syndrome: academic, open label, randomized trial. Blood Cancer Journal, 2022, 12, .	6.2	2
49	Diagnosis of Polycythemia Vera in an Anemic Patient. Southern Medical Journal, 2000, 93, 710-712.	0.7	1
50	Mechanisms of Azacitidine Chemotherapy Resistance in AML and MDS and New Therapy Options. Blood, 2018, 132, 5506-5506.	1.4	1
51	Mutual Regulatory Loop between miR-155 and PU.1 Is a Candidate Pathogenesis Factor in CLL.. Blood, 2007, 110, 1130-1130.	1.4	1
52	Transcriptional and Epigenetic Regulation in the Development of Myeloid Cells: Normal and Diseased Myelopoiesis. Epigenetics and Human Health, 2014, , 223-245.	0.2	0
53	PU.1 and pRb Bind GATA-1 on DNA and Recruit a Histone H3K9 Methyl Transferase-Containing Complex to Repress the Erythroid Transcription Program.. Blood, 2004, 104, 1614-1614.	1.4	0
54	ISWI ATPase Snf2h Is Required for Both Heterochromatin and Euchromatin Structure in ES Cells.. Blood, 2007, 110, 4062-4062.	1.4	0

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55	Fog1 and Cebpa Are DNA Targets of GATA-1/PU.1 Antagonism during Leukemia Differentiation.. Blood, 2007, 110, 4121-4121.	1.4	0
56	Gata1 Regulates Erythroid Transcription by Cooperating with Chromatin Remodeling Protein Snf2h. Blood, 2008, 112, 4759-4759.	1.4	0
57	Transcription Factors PU.1 and EGR2 Inhibits the Oncogenic Microrna Cluster Mir-17-92 during Macrophage Differentiation. Blood, 2008, 112, 473-473.	1.4	0
58	The Oncogenic Mir-17-92 MicroRNA Cluster Is Inhibited by EGR2 During Macrophage Differentiation Via JARID1b-Mediated Histone 3 Lysine 4 Demethylation. Blood, 2010, 116, 390-390.	1.4	0
59	Smarca5 Regulates Ctcf Recruitment to Chromatin, Including to Regulatory Loci Involved In Control of Globin Gene Expression In Erythroleukemia. Blood, 2010, 116, 5159-5159.	1.4	0
60	5-Azacytidine and G-CSF Derepressed Chromatin Structure of PU.1 and Its Targets Cebpa and Cbfb In Myelodysplastic Syndrome (MDS). Blood, 2010, 116, 124-124.	1.4	0
61	ISWI Chromatin Remodeling ATPase Smarca5 (Snf2h) Is Required for Murine Erythroid Development and Globin Gene Regulation. Blood, 2010, 116, 2062-2062.	1.4	0
62	Active Chromatin Structure near MYB Occupancy at the Mir-155 Host Gene Promoter Coincides with Increased Mir-155 and MYB Levels In Chronic Lymphocytic Leukemia. Blood, 2010, 116, 3589-3589.	1.4	0
63	Divalent Metal Transporter 1 (DMT1) Regulates EPO Receptor Gene Expression Via GATA-1. Blood, 2012, 120, 991-991.	1.4	0
64	Mutation Of The Divalent Metal Transporter (Dmt1) Gene Results In Inefficient Induction Of The Erythroid Transcriptional Program Due To Latter Onset Of GATA-1 and Epor Expression. Blood, 2013, 122, 2197-2197.	1.4	0
65	Oncogenic Micrornas In Cerebrospinal Fluid and Sera Reflect Therapy Efficacy and Their Reappearance Precedes Clinical Relapse In Primary and Secondary CNS Lymphoma. Blood, 2013, 122, 1777-1777.	1.4	0
66	Erythroid Transcription Factor GATA-1 Binds and Represses PU.1 Gene â€“ Candidate Mechanism Of Epigenetic Repression Of PU.1 and Inefficient Erythropoiesis In MDS. Blood, 2013, 122, 1558-1558.	1.4	0
67	Patients with Chronic Myeloid Leukemia Show Different Modulation of MYB-Dependent Oncogenic Pathway in the Course of Hematopoietic Differentiation upon Sensitivity to the TKI Treatment. Blood, 2015, 126, 1243-1243.	1.4	0
68	Tracking the Somatic Mutations in Azacitidine-Treated MDS Patients Documents Clonal Development and AZA Responsiveness. Blood, 2015, 126, 4103-4103.	1.4	0
69	Azacitidine Blocks GATA-1-Mediated Repression of the PU.1 Gene in Human Leukemic Cells. Blood, 2015, 126, 5220-5220.	1.4	0
70	Frequency and Clinical Impact of Cytogenetic Clonal Evolution in Myelodysplastic Syndromes (MDS) with Isolated Del(5q). Blood, 2016, 128, 1982-1982.	1.4	0
71	Graded PU.1 Levels Regulate Granulocyte Vs. Macrophage Genes Via Multiple Enhancer Elements. Blood, 2016, 128, 403-403.	1.4	0
72	Clonal Architecture of MDS Somatic Mutations Dynamically Changes during Azacitidine Therapy and Has Very Limited Potential to Predict Patient Outcome. Blood, 2016, 128, 4294-4294.	1.4	0

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73	Myristoylated Alanine-Rich C-Kinase Substrate (MARCKS) Is a New Biomarker for Mantle Cell Lymphoma: Expression, Localization, and Phosphorylation Study. Blood, 2016, 128, 1767-1767.	1.4	0
74	Oncogenic microRNAs to predict relapse in early breast cancer patients.. Journal of Clinical Oncology, 2017, 35, e23021-e23021.	1.6	0
75	Azacitidine Response Prediction in MDS Patients with NGS Data Using a Computational Biology Modeling (CBM) Based Clinical Decision Support System. Blood, 2018, 132, 3087-3087.	1.4	0