## Tomas Stopka

List of Publications by Year in descending order

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TOMAS STORKA

#	Article	IF	CITATIONS
1	Analysis of 5-Azacytidine Resistance Models Reveals a Set of Targetable Pathways. Cells, 2022, 11, 223.	4.1	5
2	Chromatin Remodeler Smarca5 Is Required for Cancer-Related Processes of Primary Cell Fitness and Immortalization. Cells, 2022, 11, 808.	4.1	3
3	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
4	Combined Approach to Leukemic Differentiation Using Transcription Factor PU.1-Enhancing Agents. International Journal of Molecular Sciences, 2022, 23, 6729.	4.1	3
5	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
6	The chromatin remodeler Snf2h is essential for oocyte meiotic cell cycle progression. Genes and Development, 2020, 34, 166-178.	5.9	21
7	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
8	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
9	ISWI ATPase Smarca5 Regulates Differentiation of Thymocytes Undergoing Î <sup>2</sup> -Selection. Journal of Immunology, 2019, 202, 3434-3446.	0.8	10
10	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
11	Lenalidomide treatment in lower risk myelodysplastic syndromes—The experience of a Czech hematology center. (Positive effect of erythropoietin ± prednisone addition to lenalidomide in) Tj ETQq1 1 0.78	343 <b>d.%</b> rgB1	[ /@verlock ]
12	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
13	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 ETQq1 1 0.:	784 <b>134</b> 14 rgl	3T <b>‡</b> Overlock
14	Mechanisms of Azacitidine Chemotherapy Resistance in AML and MDS and New Therapy Options. Blood, 2018, 132, 5506-5506.	1.4	1
15	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
16	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
17	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
18	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

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19	Somatic mutation dynamics in MDS patients treated with azacitidine indicate clonal selection in patients-responders. Oncotarget, 2017, 8, 111966-111978.	1.8	8
20	Oncogenic microRNAs to predict relapse in early breast cancer patients Journal of Clinical Oncology, 2017, 35, e23021-e23021.	1.6	0
21	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
22	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
23	Chromatin remodeling enzyme Snf2h regulates embryonic lens differentiation and denucleation. Development (Cambridge), 2016, 143, 1937-1947.	2.5	41
24	Frequency and Clinical Impact of Cytogenetic Clonal Evolution in Myelodysplastic Syndromes (MDS) with Isolated Del(5q). Blood, 2016, 128, 1982-1982.	1.4	0
25	Graded PU.1 Levels Regulate Granulocyte Vs. Macrophage Genes Via Multiple Enhancer Elements. Blood, 2016, 128, 403-403.	1.4	0
26	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
27	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
28	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
29	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
30	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
31	Tracking the Somatic Mutations in Azacitidine-Treated MDS Patients Documents Clonal Development and AZA Responsiveness. Blood, 2015, 126, 4103-4103.	1.4	0
32	Azacitidine Blocks GATA-1-Mediated Repression of the PU.1 Gene in Human Leukemic Cells. Blood, 2015, 126, 5220-5220.	1.4	0
33	Snf2h-mediated chromatin organization and histone H1 dynamics govern cerebellar morphogenesis and neural maturation. Nature Communications, 2014, 5, 4181.	12.8	71
34	Transcriptional and Epigenetic Regulation in the Development of Myeloid Cells: Normal and Diseased Myelopoiesis. Epigenetics and Human Health, 2014, , 223-245.	0.2	0
35	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
36	Epigenetic Control of SPI1 Gene by CTCF and ISWI ATPase SMARCA5. PLoS ONE, 2014, 9, e87448.	2.5	25

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37	Role of Epigenetics in Chronic Myeloid Leukemia. Current Hematologic Malignancy Reports, 2013, 8, 28-36.	2.3	52
38	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
39	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
40	Erythroid Transcription Factor GATA-1 Binds and Represses PU.1 Gene – Candidate Mechanism Of Epigenetic Repression Of PU.1 and Inefficient Erythropoiesis In MDS. Blood, 2013, 122, 1558-1558.	1.4	0
41	Divalent Metal Transporter 1 (DMT1) Regulates EPO Receptor Gene Expression Via GATA-1. Blood, 2012, 120, 991-991.	1.4	0
42	MYB transcriptionally regulates the miR-155 host gene in chronic lymphocytic leukemia. Blood, 2011, 117, 3816-3825.	1.4	128
43	Expression patterns of microRNAs associated with CML phases and their disease related targets. Molecular Cancer, 2011, 10, 41.	19.2	124
44	Epigenetic silencing of the oncogenic miR-17-92 cluster during PU.1-directed macrophage differentiation. EMBO Journal, 2011, 30, 4450-4464.	7.8	85
45	The role of PU.1 and GATA-1 transcription factors during normal and leukemogenic hematopoiesis. Leukemia, 2010, 24, 1249-1257.	7.2	151
46	GATA-1 directly regulates p21 gene expression during erythroid differentiation. Cell Cycle, 2010, 9, 1972-1980.	2.6	36
47	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
48	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
49	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
50	ISWI Chromatin Remodeling ATPase Smarca5 (Snf2h) Is Required for Murine Erythroid Development and Globin Gene Regulation. Blood, 2010, 116, 2062-2062.	1.4	0
51	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
52	PU.1 Activation Relieves GATA-1–Mediated Repression of <i>Cebpa</i> and <i>Cbfb</i> during Leukemia Differentiation. Molecular Cancer Research, 2009, 7, 1693-1703.	3.4	22
53	MicroRNA Mir-155 and Myb Proto-Oncogene Family Members Cooperate in Pathogenesis of Chronic Lymphocytic Leukemia Blood, 2009, 114, 58-58.	1.4	3
54	Nuclear localization of ISWI ATPase Smarca5 Snf2h in mouse. Frontiers in Bioscience - Elite, 2009, E1, 553-559.	1.8	3

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#	Article	IF	CITATIONS
55	Chromatin remodeling and SWI/SNF2 factors in human disease. Frontiers in Bioscience - Landmark, 2008, Volume, 6126.	3.0	14
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	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
59	Modifiers of epigenetic reprogramming show paternal effects in the mouse. Nature Genetics, 2007, 39, 614-622.	21.4	154
60	Mutual Regulatory Loop between miR-155 and PU.1 Is a Candidate Pathogenesis Factor in CLL Blood, 2007, 110, 1130-1130.	1.4	1
61	ISWI ATPase Snf2h Is Required for Both Heterochromatin and Euchromatin Structure in ES Cells Blood, 2007, 110, 4062-4062.	1.4	0
62	Fog1 and Cebpa Are DNA Targets of GATA-1/PU.1 Antagonism during Leukemia Differentiation Blood, 2007, 110, 4121-4121.	1.4	0
63	Regulation of αA-crystallin via Pax6, c-Maf, CREB and a broad domain of lens-specific chromatin. EMBO Journal, 2006, 25, 2107-2118.	7.8	93
64	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
65	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
66	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
67	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
68	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
69	TRAIL (Apo2L) suppresses growth of primary human leukemia and myelodysplasia progenitors. Leukemia, 2002, 16, 67-73.	7.2	108
70	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
71	Ribosomal proteins S3a, S13, S16, and S24 are not mutated in patients with Diamond-Blackfan anemia. Blood, 2001, 97, 579-580.	1.4	8
72	Diagnosis of Polycythemia Vera in an Anemic Patient. Southern Medical Journal, 2000, 93, 710-712.	0.7	1

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73	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
74	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
75	Angiotensin II stimulates proliferation of normal early erythroid progenitors Journal of Clinical Investigation, 1997, 100, 2310-2314.	8.2	231