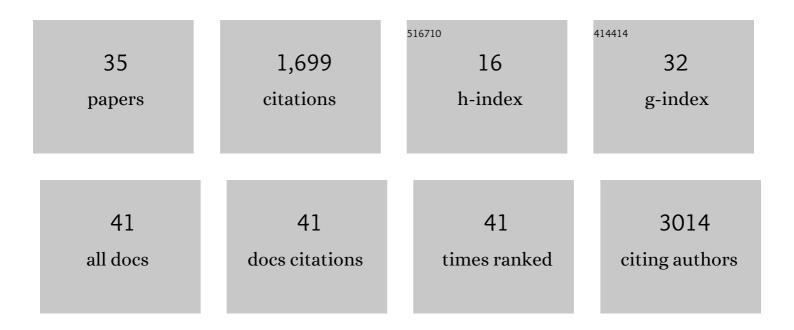
Aniruddha J Deshpande

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Differential roles of BAF and PBAF subunits, Arid1b and Arid2, in MLL-AF9 leukemogenesis. Leukemia, 2022, 36, 946-955.	7.2	8
2	Discovery of novel furanylbenzamide inhibitors that target oncogenic tyrosine phosphatase SHP2 in leukemia cells. Journal of Biological Chemistry, 2022, 298, 101477.	3.4	6
3	A JAK/STAT-mediated inflammatory signaling cascade drives oncogenesis in AF10-rearranged AML. Blood, 2021, 137, 3403-3415.	1.4	8
4	The role of the PZP domain of AF10 in acute leukemia driven by AF10 translocations. Nature Communications, 2021, 12, 4130.	12.8	8
5	The ubiquitin ligase RNF5 determines acute myeloid leukemia growth and susceptibility to histone deacetylase inhibitors. Nature Communications, 2021, 12, 5397.	12.8	20
6	A systematic genome-wide mapping of oncogenic mutation selection during CRISPR-Cas9 genome editing. Nature Communications, 2021, 12, 6512.	12.8	24
7	Loss of HIF1A From Pancreatic Cancer Cells Increases Expression of PPP1R1B and Degradation of p53 to Promote Invasion and Metastasis. Gastroenterology, 2020, 159, 1882-1897.e5.	1.3	79
8	Specific patterns of H3K79 methylation influence genetic interaction of oncogenes in AML. Blood Advances, 2020, 4, 3109-3122.	5.2	3
9	A Synthetic Lethal Approach to Eradicate AML Via Synergistic Activation of Pro-Apoptotic p53 By MDM2 and BET Inhibitors. Blood, 2020, 136, 14-14.	1.4	0
10	RNF5 Defines Acute Myeloid Leukemia Growth and Susceptibility to Histone Deacetylase Inhibitors. Blood, 2020, 136, 31-32.	1.4	0
11	High-Density Domain-Focused CRISPR Screens Reveal Epigenetic Regulators of Hox/Meis Gene Expression in Acute Myeloid Leukemia. Blood, 2020, 136, 2-3.	1.4	1
12	The role of TP53 in acute myeloid leukemia: Challenges and opportunities. Genes Chromosomes and Cancer, 2019, 58, 875-888.	2.8	79
13	Investigation of Genetic Dependencies Using CRISPR-Cas9-based Competition Assays. Journal of Visualized Experiments, 2019, , .	0.3	3
14	Acute myeloid leukemia driven by the CALM-AF10 fusion gene is dependent on BMI1. Experimental Hematology, 2019, 74, 42-51.e3.	0.4	15
15	A Multiscale Map of the Stem Cell State in Pancreatic Adenocarcinoma. Cell, 2019, 177, 572-586.e22.	28.9	107
16	Structural Variants Involving MLLT10/AF10 Are Associated with Adverse Outcome in AML Regardless of the Partner Gene - a COG/Tpaml Study. Blood, 2019, 134, 461-461.	1.4	12
17	The basic helix-loop-helix transcription factor SHARP1 is an oncogenic driver in MLL-AF6 acute myelogenous leukemia. Nature Communications, 2018, 9, 1622.	12.8	20
18	Epigenetic Regulators in the Development, Maintenance, and Therapeutic Targeting of Acute Myeloid Leukemia. Frontiers in Oncology, 2018, 8, 41.	2.8	56

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#	Article	IF	CITATIONS
19	Controlled stem cell amplification by HOXB4 depends on its unique proline-rich region near the N terminus. Blood, 2017, 129, 319-323.	1.4	11
20	Targeting Chromatin Regulators Inhibits Leukemogenic Gene Expression in <i>NPM1</i> Mutant Leukemia. Cancer Discovery, 2016, 6, 1166-1181.	9.4	171
21	MLL-AF9– and HOXA9-mediated acute myeloid leukemia stem cell self-renewal requires JMJD1C. Journal of Clinical Investigation, 2016, 126, 997-1011.	8.2	69
22	DOT1L inhibits SIRT1-mediated epigenetic silencing to maintain leukemic gene expression in MLL-rearranged leukemia. Nature Medicine, 2015, 21, 335-343.	30.7	200
23	The PZP Domain of AF10 Senses Unmodified H3K27 to Regulate DOT1L-Mediated Methylation of H3K79. Molecular Cell, 2015, 60, 319-327.	9.7	78
24	AF10 Regulates Progressive H3K79 Methylation and HOX Gene Expression in Diverse AML Subtypes. Cancer Cell, 2014, 26, 896-908.	16.8	153
25	DNA-damage-induced differentiation of leukaemic cells as an anti-cancer barrier. Nature, 2014, 514, 107-111.	27.8	174
26	Leukemic transformation by the MLL-AF6 fusion oncogene requires the H3K79 methyltransferase Dot1l. Blood, 2013, 121, 2533-2541.	1.4	149
27	MLL-AF6 Mediated Transformation Is Dependent On the H3K79 Methyl-transferase Dot1l. Blood, 2012, 120, 3502-3502.	1.4	0
28	The Interaction Between DOT1L and AF10 Is Required for H3K79 Dimethylation and MLL-AF9 Leukemia. Blood, 2012, 120, 401-401.	1.4	0
29	Abrogation of MLL-AF10 and CALM-AF10 Mediated Transformation Through Genetic Inactivation or Pharmacological Inhibition of the H3K79 Methyltransferase DOT1L Blood, 2012, 120, 2384-2384.	1.4	0
30	Global reduction of the epigenetic H3K79 methylation mark and increased chromosomal instability in CALM-AF10–positive leukemias. Blood, 2009, 114, 651-658.	1.4	59
31	Identification of Murine and Human Acute Myeloid Leukemia Stem Cells. Methods in Molecular Biology, 2009, 568, 21-35.	0.9	3
32	Lymphoid Progenitors as Candidate Cancer Stem Cells in AML: New Perspectives. Cell Cycle, 2007, 6, 543-545.	2.6	12
33	Knocking the Wnt out of the Sails of Leukemia Stem Cell Development. Cell Stem Cell, 2007, 1, 597-598.	11.1	12
34	Acute myeloid leukemia is propagated by a leukemic stem cell with lymphoid characteristics in a mouse model of CALM/AF10-positive leukemia. Cancer Cell, 2006, 10, 363-374.	16.8	119
35	A Lymphoid Progenitor Propagates AML in a Mouse Model of CALM/AF10 Positive Leukemia Blood, 2005, 106, 101-101.	1.4	0