Matt Teater

List of Publications by Year
in descending order

Source: https://exaly.com/author-pdf/2114793/publications.pdf

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35 papers	3,169 citations	304743 22 h-index	454955 30 g-index
35 all docs	35 docs citations	35 times ranked	5390 citing authors

#	Article	IF	Citations
1	EZH2 Is Required for Germinal Center Formation and Somatic EZH2 Mutations Promote Lymphoid Transformation. Cancer Cell, 2013, 23, 677-692.	16.8	706
2	Loss of BAP1 function leads to EZH2-dependent transformation. Nature Medicine, 2015, 21, 1344-1349.	30.7	297
3	CREBBP Inactivation Promotes the Development of HDAC3-Dependent Lymphomas. Cancer Discovery, 2017, 7, 38-53.	9.4	218
4	Molecular and Genetic Characterization of MHC Deficiency Identifies EZH2 as Therapeutic Target for Enhancing Immune Recognition. Cancer Discovery, 2019, 9, 546-563.	9.4	213
5	EZH2 and BCL6 Cooperate to Assemble CBX8-BCOR Complex to Repress Bivalent Promoters, Mediate Germinal Center Formation and Lymphomagenesis. Cancer Cell, 2016, 30, 197-213.	16.8	200
6	Histone H1 loss drives lymphoma by disrupting 3D chromatin architecture. Nature, 2021, 589, 299-305.	27.8	155
7	CTCF Haploinsufficiency Destabilizes DNA Methylation and Predisposes to Cancer. Cell Reports, 2014, 7, 1020-1029.	6.4	154
8	Chemotherapy Induces Senescence-Like Resilient Cells Capable of Initiating AML Recurrence. Cancer Discovery, 2021, 11, 1542-1561.	9.4	133
9	EZH2 enables germinal centre formation through epigenetic silencing of CDKN1A and an Rb-E2F1 feedback loop. Nature Communications, 2017, 8, 877.	12.8	132
10	TET2 Deficiency Causes Germinal Center Hyperplasia, Impairs Plasma Cell Differentiation, and Promotes B-cell Lymphomagenesis. Cancer Discovery, 2018, 8, 1632-1653.	9.4	120
11	Multi-tiered Reorganization of the Genome during B Cell Affinity Maturation Anchored by a Germinal Center-Specific Locus Control Region. Immunity, 2016, 45, 497-512.	14.3	112
12	Selective Inhibition of HDAC3 Targets Synthetic Vulnerabilities and Activates Immune Surveillance in Lymphoma. Cancer Discovery, 2020, 10, 440-459.	9.4	103
13	Mutant EZH2 Induces a Pre-malignant Lymphoma Niche by Reprogramming the Immune Response. Cancer Cell, 2020, 37, 655-673.e11.	16.8	93
14	DNA Methylation Dynamics of Germinal Center B Cells Are Mediated by AID. Cell Reports, 2015, 12, 2086-2098.	6.4	87
15	The BCL6 RD2 Domain Governs Commitment of Activated B Cells to Form Germinal Centers. Cell Reports, 2014, 8, 1497-1508.	6.4	67
16	TBL1XR1 Mutations Drive Extranodal Lymphoma by Inducing a Pro-tumorigenic Memory Fate. Cell, 2020, 182, 297-316.e27.	28.9	63
17	Genetic and epigenetic inactivation of SESTRIN1 controls mTORC1 and response to EZH2 inhibition in follicular lymphoma. Science Translational Medicine, 2017, 9, .	12.4	52
18	AICDA drives epigenetic heterogeneity and accelerates germinal center-derived lymphomagenesis. Nature Communications, 2018, 9, 222.	12.8	51

#	Article	IF	Citations
19	Specific covalent inhibition of MALT1 paracaspase suppresses B cell lymphoma growth. Journal of Clinical Investigation, 2018, 128, 4397-4412.	8.2	51
20	Rational Targeting of Cooperating Layers of the Epigenome Yields Enhanced Therapeutic Efficacy against AML. Cancer Discovery, 2019, 9, 872-889.	9.4	36
21	The serine hydroxymethyltransferase-2 (SHMT2) initiates lymphoma development through epigenetic tumor suppressor silencing. Nature Cancer, 2020, 1, 653-664.	13.2	35
22	Combined EZH2 and Bcl-2 inhibitors as precision therapy for genetically defined DLBCL subtypes. Blood Advances, 2020, 4, 5226-5231.	5.2	28
23	Identification of MALT1 feedback mechanisms enables rational design of potent antilymphoma regimens for ABC-DLBCL. Blood, 2021, 137, 788-800.	1.4	22
24	Translational Activation of ATF4 through Mitochondrial Anaplerotic Metabolic Pathways Is Required for DLBCL Growth and Survival. Blood Cancer Discovery, 2022, 3, 50-65.	5.0	14
25	SETD2 Haploinsufficiency Enhances Germinal Centerâ€“Associated AICDA Somatic Hypermutation to Drive B-cell Lymphomagenesis. Cancer Discovery, 2022, 12, 1782-1803.	9.4	14
26	Reply to "Uveal melanoma cells are resistant to EZH2 inhibition regardless of BAP1 status". Nature Medicine, 2016, 22, 578-579.	30.7	7
27	BCL10 Mutations Define Distinct Dependencies Guiding Precision Therapy for DLBCL. Cancer Discovery, 0, , OF1-OF20.	9.4	2
28	Untangling the Web of Lymphoma Somatic Mutations. Cell, 2017, 171, 270-272.	28.9	1
29	Demethylase Activity of Aid during Germinal Center B Cell Maturation Could Contribute to Lymphomagenesis. Blood, 2014, 124, 59-59.	1.4	1
30	AICDA Introduces Epigenetic Plasticity in Germinal Center-Derived Lymphomas and Accelerates Lymphomagenesis. Blood, 2016, 128, 1045-1045.	1.4	1
31	Acute Myeloid Leukemia Cells Resist Chemotherapy through a Reversible Senescence-like State Maintaining Repopulation Potential. Blood, 2016, 128, 582-582.	1.4	1
32	A Chromatin Reader That Acts As a Key to Lock in and Coordinate Recruitment of Transcription Factors and a Novel Polycomb Complex to Bivalent Chromatin Thus Driving Formation of Germinal Centers and B-Cell Lymphomas. Blood, 2015, 126, 434-434.	1.4	0
33	BAP1 Loss Results in EZH2-Dependent Transformation in Myelodysplastic Syndromes. Blood, 2015, 126, 713-713.	1.4	0
34	Crebbp Mutations Disrupt Dynamic Enhancer Acetylation in B-Cells, Enabling HDAC3 to Drive Lymphomagenesis. Blood, 2016, 128, 735-735.	1.4	0
35	Cooperative Gene Repression By DNA Methylation and LSD1-Mediated Enhancer Inactivation in Acute Myeloid Leukemia. Blood, 2016, 128, 1048-1048.	1.4	0