

Christopher A Eide

List of Publications by Year in descending order

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85
papers

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109321
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times ranked

9871
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#	ARTICLE	IF	CITATIONS
1	AP24534, a Pan-BCR-ABL Inhibitor for Chronic Myeloid Leukemia, Potently Inhibits the T315I Mutant and Overcomes Mutation-Based Resistance. <i>Cancer Cell</i> , 2009, 16, 401-412.	16.8	1,050
2	Functional genomic landscape of acute myeloid leukaemia. <i>Nature</i> , 2018, 562, 526-531.	27.8	907
3	Bcr-Abl kinase domain mutations, drug resistance, and the road to a cure for chronic myeloid leukemia. <i>Blood</i> , 2007, 110, 2242-2249.	1.4	590
4	Oncogenic CSF3R Mutations in Chronic Neutrophilic Leukemia and Atypical CML. <i>New England Journal of Medicine</i> , 2013, 368, 1781-1790.	27.0	499
5	Comparison of imatinib mesylate, dasatinib (BMS-354825), and nilotinib (AMN107) in an N-ethyl-N-nitrosourea (ENU)-based mutagenesis screen: high efficacy of drug combinations. <i>Blood</i> , 2006, 108, 2332-2338.	1.4	368
6	Activating alleles of JAK3 in acute megakaryoblastic leukemia. <i>Cancer Cell</i> , 2006, 10, 65-75.	16.8	295
7	BCR-ABL1 Compound Mutations Combining Key Kinase Domain Positions Confer Clinical Resistance to Ponatinib in Ph Chromosome-Positive Leukemia. <i>Cancer Cell</i> , 2014, 26, 428-442.	16.8	292
8	Response and Resistance to BCR-ABL1-Targeted Therapies. <i>Cancer Cell</i> , 2020, 37, 530-542.	16.8	246
9	BCR-ABL1 compound mutations in tyrosine kinase inhibitor-resistant CML: frequency and clonal relationships. <i>Blood</i> , 2013, 121, 489-498.	1.4	187
10	Identification of Interleukin-1 by Functional Screening as a Key Mediator of Cellular Expansion and Disease Progression in Acute Myeloid Leukemia. <i>Cell Reports</i> , 2017, 18, 3204-3218.	6.4	187
11	Wnt/Ca2+/NFAT Signaling Maintains Survival of Ph+ Leukemia Cells upon Inhibition of Bcr-Abl. <i>Cancer Cell</i> , 2010, 18, 74-87.	16.8	164
12	A Novel Crizotinib-Resistant Solvent-Front Mutation Responsive to Cabozantinib Therapy in a Patient with ROS1-Rearranged Lung Cancer. <i>Clinical Cancer Research</i> , 2016, 22, 2351-2358.	7.0	141
13	Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. <i>Cancer Research</i> , 2019, 79, 4744-4753.	0.9	139
14	Combining the Allosteric Inhibitor Asciminib with Ponatinib Suppresses Emergence of and Restores Efficacy against Highly Resistant BCR-ABL1 Mutants. <i>Cancer Cell</i> , 2019, 36, 431-443.e5.	16.8	137
15	Targeting the BCR-ABL Signaling Pathway in Therapy-Resistant Philadelphia Chromosome-Positive Leukemia. <i>Clinical Cancer Research</i> , 2011, 17, 212-221.	7.0	127
16	High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. <i>Blood</i> , 2009, 113, 1749-1755.	1.4	119
17	Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. <i>Nature Communications</i> , 2019, 10, 244.	12.8	111
18	Antagonism of SET Using OP449 Enhances the Efficacy of Tyrosine Kinase Inhibitors and Overcomes Drug Resistance in Myeloid Leukemia. <i>Clinical Cancer Research</i> , 2014, 20, 2092-2103.	7.0	108

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19	Nilotinib and MEK Inhibitors Induce Synthetic Lethality through Paradoxical Activation of RAF in Drug-Resistant Chronic Myeloid Leukemia. <i>Cancer Cell</i> , 2011, 20, 715-727.	16.8	107
20	Foretinib is a potent inhibitor of oncogenic ROS1 fusion proteins. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2013, 110, 19519-19524.	7.1	106
21	A therapeutically targetable mechanism of BCR-ABL-independent imatinib resistance in chronic myeloid leukemia. <i>Science Translational Medicine</i> , 2014, 6, 252ra121.	12.4	105
22	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. <i>Leukemia</i> , 2019, 33, 1835-1850.	7.2	97
23	Structural insight into selectivity and resistance profiles of ROS1 tyrosine kinase inhibitors. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2015, 112, E5381-90.	7.1	93
24	The ABL Switch Control Inhibitor DCC-2036 Is Active against the Chronic Myeloid Leukemia Mutant BCR-ABL T315I and Exhibits a Narrow Resistance Profile. <i>Cancer Research</i> , 2011, 71, 3189-3195.	0.9	91
25	Molecularly targeted drug combinations demonstrate selective effectiveness for myeloid- and lymphoid-derived hematologic malignancies. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, E7554-E7563.	7.1	86
26	SGX393 inhibits the CML mutant Bcr-Abl T315I and preempts <i>in vitro</i> resistance when combined with nilotinib or dasatinib. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2008, 105, 5507-5512.	7.1	84
27	High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. <i>Blood</i> , 2008, 111, 4788-4796.	1.4	84
28	Targeting BCL-2 and ABL/LYN in Philadelphia chromosome-positive acute lymphoblastic leukemia. <i>Science Translational Medicine</i> , 2016, 8, 354ra114.	12.4	65
29	New Bcr-Abl inhibitors in chronic myeloid leukemia: keeping resistance in check. <i>Expert Opinion on Investigational Drugs</i> , 2008, 17, 865-878.	4.1	58
30	Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. <i>Blood</i> , 2019, 134, 867-879.	1.4	55
31	Acute dasatinib exposure commits Bcr-Abl-dependent cells to apoptosis. <i>Blood</i> , 2009, 114, 3459-3463.	1.4	54
32	CRISPR-Cas9-mediated saturated mutagenesis screen predicts clinical drug resistance with improved accuracy. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2017, 114, 11751-11756.	7.1	50
33	Cytokine-Mediated Inflammatory Pathways Promote Clonal Evolution and Disease Progression in Acute Myeloid Leukemia. <i>Blood</i> , 2016, 128, 1688-1688.	1.4	41
34	The Colony-Stimulating Factor 3 Receptor T640N Mutation Is Oncogenic, Sensitive to JAK Inhibition, and Mimics T618I. <i>Clinical Cancer Research</i> , 2016, 22, 757-764.	7.0	40
35	Chronic Myeloid Leukemia: Advances in Understanding Disease Biology and Mechanisms of Resistance to Tyrosine Kinase Inhibitors. <i>Current Hematologic Malignancy Reports</i> , 2015, 10, 158-166.	2.3	39
36	The BCR-ABL35INS insertion/truncation mutant is kinase-inactive and does not contribute to tyrosine kinase inhibitor resistance in chronic myeloid leukemia. <i>Blood</i> , 2011, 118, 5250-5254.	1.4	37

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37	Targeting of colony-stimulating factor 1 receptor (CSF1R) in the CLL microenvironment yields antineoplastic activity in primary patient samples. <i>Oncotarget</i> , 2018, 9, 24576-24589.	1.8	36
38	An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. <i>Leukemia</i> , 2008, 22, 2269-2272.	7.2	32
39	Src and STAT3 inhibitors synergize to promote tumor inhibition in renal cell carcinoma. <i>Oncotarget</i> , 2015, 6, 44675-44687.	1.8	27
40	Persistent LYN Signaling in Imatinib-Resistant, BCR-ABL ⁻ Independent Chronic Myelogenous Leukemia. <i>Journal of the National Cancer Institute</i> , 2008, 100, 908-909.	6.3	26
41	Threshold Levels of ABL Tyrosine Kinase Inhibitors Retained in Chronic Myeloid Leukemia Cells Determine Their Commitment to Apoptosis. <i>Cancer Research</i> , 2013, 73, 3356-3370.	0.9	26
42	NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2V617F-positive myeloproliferative neoplasm cells. <i>Signal Transduction and Targeted Therapy</i> , 2020, 5, 5.	17.1	26
43	GATA-2 functions downstream of BMPs and CaM KIV in ectodermal cells during primitive hematopoiesis. <i>Developmental Biology</i> , 2007, 310, 454-469.	2.0	23
44	Bcr-Abl Kinase Domain Mutations and the Unsettled Problem of Bcr-AblT315I: Looking into the Future of Controlling Drug Resistance in Chronic Myeloid Leukemia. <i>Clinical Lymphoma and Myeloma</i> , 2007, 7, S120-S130.	1.4	22
45	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. <i>Cancer Research</i> , 2013, 73, 5775-5786.	0.9	22
46	BCR-ABL1 promotes leukemia by converting p27 into a cytoplasmic oncoprotein. <i>Blood</i> , 2014, 124, 3260-3273.	1.4	20
47	Therapeutically Targetable ALK Mutations in Leukemia. <i>Cancer Research</i> , 2015, 75, 2146-2150.	0.9	20
48	Proteasome 26S subunit, non-ATPases 1 (PSMD1) and 3 (PSMD3), play an oncogenic role in chronic myeloid leukemia by stabilizing nuclear factor-kappa B. <i>Oncogene</i> , 2021, 40, 2697-2710.	5.9	20
49	IRS2 silencing increases apoptosis and potentiates the effects of ruxolitinib in JAK2V617F-positive myeloproliferative neoplasms. <i>Oncotarget</i> , 2016, 7, 6948-6959.	1.8	20
50	A genome-wide CRISPR screen identifies regulators of MAPK and MTOR pathways that mediate resistance to sorafenib in acute myeloid leukemia. <i>Haematologica</i> , 2022, 107, 77-85.	3.5	20
51	Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. <i>Leukemia</i> , 2020, 34, 2342-2353.	7.2	18
52	Biomarkers Predicting Venetoclax Sensitivity and Strategies for Venetoclax Combination Treatment. <i>Blood</i> , 2018, 132, 175-175.	1.4	18
53	Differentiation of leukemic blasts is not completely blocked in acute myeloid leukemia. <i>Proceedings of the National Academy of Sciences of the United States of America</i> , 2019, 116, 24593-24599.	7.1	17
54	Dual inhibition of JAK1/2 kinases and BCL2: a promising therapeutic strategy for acute myeloid leukemia. <i>Leukemia</i> , 2018, 32, 2025-2028.	7.2	16

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55	Synthetic lethality of TNK2 inhibition in PTPN11-mutant leukemia. <i>Science Signaling</i> , 2018, 11, .	3.6	16
56	ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. <i>Leukemia</i> , 2020, 34, 2798-2804.	7.2	16
57	High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. <i>Leukemia</i> , 2009, 23, 406-409.	7.2	15
58	Metformin exerts multitarget antileukemia activity in JAK2V617F-positive myeloproliferative neoplasms. <i>Cell Death and Disease</i> , 2018, 9, 311.	6.3	14
59	Differentiation status of primary chronic myeloid leukemia cells affects sensitivity to BCR-ABL1 inhibitors. <i>Oncotarget</i> , 2017, 8, 22606-22615.	1.8	13
60	The function of the pleckstrin homology domain in BCR-ABL-mediated leukemogenesis. <i>Leukemia</i> , 2010, 24, 226-229.	7.2	12
61	Understanding cancer from the stem cells up. <i>Nature Medicine</i> , 2017, 23, 656-657.	30.7	11
62	Integrating in vitro sensitivity and dose-response slope is predictive of clinical response to ABL kinase inhibitors in chronic myeloid leukemia. <i>Blood</i> , 2013, 122, 3331-3334.	1.4	10
63	Growth Arrest of BCR-ABL Positive Cells with a Sequence-Specific Polyamide-Chlorambucil Conjugate. <i>PLoS ONE</i> , 2008, 3, e3593.	2.5	9
64	HitWalker: variant prioritization for personalized functional cancer genomics. <i>Bioinformatics</i> , 2013, 29, 509-510.	4.1	9
65	Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. <i>Leukemia</i> , 2016, 30, 1418-1421.	7.2	9
66	A novel <i>AGGF1-PDGFRb</i> fusion in pediatric T-cell acute lymphoblastic leukemia. <i>Haematologica</i> , 2018, 103, e87-e91.	3.5	8
67	NT157, an IGF1R-IRS1/2 inhibitor, exhibits antineoplastic effects in pre-clinical models of chronic myeloid leukemia. <i>Investigational New Drugs</i> , 2021, 39, 736-746.	2.6	7
68	Aurora A kinase as a target for therapy in <i>TCF3-HLF</i> rearranged acute lymphoblastic leukemia. <i>Haematologica</i> , 2021, 106, 2990-2994.	3.5	6
69	Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. <i>Cancer Research</i> , 2021, 81, 4685-4695.	0.9	6
70	Associating drug sensitivity with differentiation status identifies effective combinations for acute myeloid leukemia. <i>Blood Advances</i> , 2022, 6, 3062-3067.	5.2	6
71	New Strategies for the First-Line Treatment of Chronic Myeloid Leukemia: Can Resistance Be Avoided?. <i>Clinical Lymphoma and Myeloma</i> , 2008, 8, S107-S117.	1.4	5
72	Clonal hematopoiesis as determined by the HUMARA assay is a marker for acquired mutations in epigenetic regulators in older women. <i>Experimental Hematology</i> , 2016, 44, 857-865.e5.	0.4	5

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73	Novel Combination Therapy of Venetoclax and Ruxolitinib in the Treatment of Patients with Relapsed/Refractory Acute Myeloid Leukemia. <i>Blood</i> , 2021, 138, 2333-2333.	1.4	5
74	Inhibition of T315I Bcr-Abl and Other Imatinib-Resistant Bcr-Abl Mutants by the Selective Abl Kinase Inhibitor SGX70393.. <i>Blood</i> , 2006, 108, 1373-1373.	1.4	3
75	Functional validation of the oncogenic cooperativity and targeting potential of tuberous sclerosis mutation in medulloblastoma using a MYC-amplified model cell line. <i>Pediatric Blood and Cancer</i> , 2017, 64, e26553.	1.5	2
76	Combining the Allosteric ABL1 Tyrosine Kinase Inhibitor ABL001 with ATP-Competitive Inhibitors to Suppress Resistance in Chronic Myeloid Leukemia. <i>Blood</i> , 2016, 128, 2747-2747.	1.4	2
77	Characterizing Population Heterogeneity and Signaling Changes in Chronic Myeloid Leukemia Stem and Progenitor Cells upon Combined Treatment with Imatinib and MEK Inhibitors Using Quantitative Single Cell Phospho-Imaging. <i>Blood</i> , 2018, 132, 4248-4248.	1.4	2
78	Kinase Inhibitor Therapy in CML: It's What's Inside That Counts. <i>Oncotarget</i> , 2013, 4, 1332-1333.	1.8	1
79	A New Generation of Drugs in Cancer Treatment: Molecularly Targeted Therapies. , 0, , 193-221.		0
80	Identification of Tyrosine Kinase Mutations by Large-Scale DNA Sequencing in Patients with Chronic Myelomonocytic Leukemia/Atypical Chronic Myeloid Leukemia.. <i>Blood</i> , 2006, 108, 3606-3606.	1.4	0
81	High-Throughput Validation of Mutations Identified in Primary Leukemia Cells. <i>Blood</i> , 2016, 128, 4725-4725.	1.4	0
82	Combining p38MAPK Inhibitors with a Second Targeted Agent Enhances Blockade of Inflammatory Signaling-Mediated Survival in Acute Myeloid Leukemia Cells. <i>Blood</i> , 2018, 132, 2726-2726.	1.4	0
83	Mathematical and Experimental Evidence That Differentiation of Leukemic Blasts in Acute Myeloid Leukemia Is Not Completely Blocked. <i>Blood</i> , 2019, 134, 1435-1435.	1.4	0
84	A Role for Lipid Metabolism in Tyrosine Kinase Inhibitor (TKI) Resistance of Chronic Myeloid Leukemia (CML). <i>Blood</i> , 2021, 138, 2542-2542.	1.4	0
85	Patterns of Venetoclax Sensitivity in Chronic Lymphocytic Leukemia. <i>Blood</i> , 2020, 136, 12-14.	1.4	0