Christopher A Eide

List of Publications by Year in descending order

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85 papers 7,560 citations

35 h-index 78 g-index

85 all docs

85 docs citations

85 times ranked 9871 citing authors

#	Article	IF	CITATIONS
1	A genome-wide CRISPR screen identifies regulators of MAPK and MTOR pathways that mediate resistance to sorafenib in acute myeloid leukemia. Haematologica, 2022, 107, 77-85.	3.5	20
2	Associating drug sensitivity with differentiation status identifies effective combinations for acute myeloid leukemia. Blood Advances, 2022, 6, 3062-3067.	5.2	6
3	NT157, an IGF1R-IRS1/2 inhibitor, exhibits antineoplastic effects in pre-clinical models of chronic myeloid leukemia. Investigational New Drugs, 2021, 39, 736-746.	2.6	7
4	Proteasome 26S subunit, non-ATPases 1 (PSMD1) and 3 (PSMD3), play an oncogenic role in chronic myeloid leukemia by stabilizing nuclear factor-kappa B. Oncogene, 2021, 40, 2697-2710.	5.9	20
5	Aurora A kinase as a target for therapy in <i>TCF3-HLF</i> rearranged acute lymphoblastic leukemia. Haematologica, 2021, 106, 2990-2994.	3 . 5	6
6	Lentiviral-Driven Discovery of Cancer Drug Resistance Mutations. Cancer Research, 2021, 81, 4685-4695.	0.9	6
7	A Role for Lipid Metabolism in Tyrosine Kinase Inhibitor (TKI) Resistance of Chronic Myeloid Leukemia (CML). Blood, 2021, 138, 2542-2542.	1.4	0
8	Novel Combination Therapy of Venetoclax and Ruxolitinib in the Treatment of Patients with Relapsed/Refractory Acute Myeloid Leukemia. Blood, 2021, 138, 2333-2333.	1.4	5
9	ERBB2/HER2 mutations are transforming and therapeutically targetable in leukemia. Leukemia, 2020, 34, 2798-2804.	7.2	16
10	Simultaneous kinase inhibition with ibrutinib and BCL2 inhibition with venetoclax offers a therapeutic strategy for acute myeloid leukemia. Leukemia, 2020, 34, 2342-2353.	7.2	18
11	NT157 has antineoplastic effects and inhibits IRS1/2 and STAT3/5 in JAK2V617F-positive myeloproliferative neoplasm cells. Signal Transduction and Targeted Therapy, 2020, 5, 5.	17.1	26
12	Response and Resistance to BCR-ABL1-Targeted Therapies. Cancer Cell, 2020, 37, 530-542.	16.8	246
13	Patterns of Venetoclax Sensitivity in Chronic Lymphocytic Leukemia. Blood, 2020, 136, 12-14.	1.4	O
14	Genomic landscape of neutrophilic leukemias of ambiguous diagnosis. Blood, 2019, 134, 867-879.	1.4	55
15	Targeting BCR-ABL1 in Chronic Myeloid Leukemia by PROTAC-Mediated Targeted Protein Degradation. Cancer Research, 2019, 79, 4744-4753.	0.9	139
16	Combining the Allosteric Inhibitor Asciminib with Ponatinib Suppresses Emergence of and Restores Efficacy against Highly Resistant BCR-ABL1 Mutants. Cancer Cell, 2019, 36, 431-443.e5.	16.8	137
17	Laying the foundation for genomically-based risk assessment in chronic myeloid leukemia. Leukemia, 2019, 33, 1835-1850.	7.2	97
18	Differentiation of leukemic blasts is not completely blocked in acute myeloid leukemia. Proceedings of the National Academy of Sciences of the United States of America, 2019, 116, 24593-24599.	7.1	17

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19	Clinical resistance to crenolanib in acute myeloid leukemia due to diverse molecular mechanisms. Nature Communications, 2019, 10, 244.	12.8	111
20	Mathematical and Experimental Evidence That Differentiation of Leukemic Blasts in Acute Myeloid Leukemia Is Not Completely Blocked. Blood, 2019, 134, 1435-1435.	1.4	0
21	Metformin exerts multitarget antileukemia activity in JAK2V617F-positive myeloproliferative neoplasms. Cell Death and Disease, 2018, 9, 311.	6.3	14
22	A novel <i>AGGF1-PDGFRb</i> fusion in pediatric T-cell acute lymphoblastic leukemia. Haematologica, 2018, 103, e87-e91.	3.5	8
23	Targeting of colony-stimulating factor 1 receptor (CSF1R) in the CLL microenvironment yields antineoplastic activity in primary patient samples. Oncotarget, 2018, 9, 24576-24589.	1.8	36
24	Functional genomic landscape of acute myeloid leukaemia. Nature, 2018, 562, 526-531.	27.8	907
25	Dual inhibition of JAK1/2 kinases and BCL2: a promising therapeutic strategy for acute myeloid leukemia. Leukemia, 2018, 32, 2025-2028.	7.2	16
26	Synthetic lethality of TNK2 inhibition in PTPN11-mutant leukemia. Science Signaling, 2018, 11, .	3.6	16
27	Biomarkers Predicting Venetoclax Sensitivity and Strategies for Venetoclax Combination Treatment. Blood, 2018, 132, 175-175.	1.4	18
28	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. Blood, 2018, 132, 4248-4248.	1.4	2
29	Combining p38MAPK Inhibitors with a Second Targeted Agent Enhances Blockade of Inflammatory Signaling-Mediated Survival in Acute Myeloid Leukemia Cells. Blood, 2018, 132, 2726-2726.	1.4	0
30	Functional validation of the oncogenic cooperativity and targeting potential of tuberous sclerosis mutation in medulloblastoma using a MYCâ€amplified model cell line. Pediatric Blood and Cancer, 2017, 64, e26553.	1.5	2
31	Identification of Interleukin-1 by Functional Screening as a Key Mediator of Cellular Expansion and Disease Progression in Acute Myeloid Leukemia. Cell Reports, 2017, 18, 3204-3218.	6.4	187
32	CRISPR-Cas9–mediated saturated mutagenesis screen predicts clinical drug resistance with improved accuracy. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, 11751-11756.	7.1	50
33	Molecularly targeted drug combinations demonstrate selective effectiveness for myeloid- and lymphoid-derived hematologic malignancies. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E7554-E7563.	7.1	86
34	Understanding cancer from the stem cells up. Nature Medicine, 2017, 23, 656-657.	30.7	11
35	Differentiation status of primary chronic myeloid leukemia cells affects sensitivity to BCR-ABL1 inhibitors. Oncotarget, 2017, 8, 22606-22615.	1.8	13
36	Targeting BCL-2 and ABL/LYN in Philadelphia chromosome–positive acute lymphoblastic leukemia. Science Translational Medicine, 2016, 8, 354ra114.	12.4	65

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37	Clonal hematopoiesis as determined by the HUMARA assay is a marker for acquired mutations in epigenetic regulators in older women. Experimental Hematology, 2016, 44, 857-865.e5.	0.4	5
38	A Novel Crizotinib-Resistant Solvent-Front Mutation Responsive to Cabozantinib Therapy in a Patient with <i>ROS1</i> -Rearranged Lung Cancer. Clinical Cancer Research, 2016, 22, 2351-2358.	7.0	141
39	Extreme mutational selectivity of axitinib limits its potential use as a targeted therapeutic for BCR-ABL1-positive leukemia. Leukemia, 2016, 30, 1418-1421.	7.2	9
40	The Colony-Stimulating Factor 3 Receptor T640N Mutation Is Oncogenic, Sensitive to JAK Inhibition, and Mimics T618I. Clinical Cancer Research, 2016, 22, 757-764.	7.0	40
41	Cytokine-Mediated Inflammatory Pathways Promote Clonal Evolution and Disease Progression in Acute Myeloid Leukemia. Blood, 2016, 128, 1688-1688.	1.4	41
42	Combining the Allosteric ABL1 Tyrosine Kinase Inhibitor ABL001 with ATP-Competitive Inhibitors to Suppress Resistance in Chronic Myeloid Leukemia. Blood, 2016, 128, 2747-2747.	1.4	2
43	IRS2 silencing increases apoptosis and potentiates the effects of ruxolitinib in JAK2V617F-positive myeloproliferative neoplasms. Oncotarget, 2016, 7, 6948-6959.	1.8	20
44	High-Throughput Validation of Mutations Identified in Primary Leukemia Cells. Blood, 2016, 128, 4725-4725.	1.4	0
45	Therapeutically Targetable ALK Mutations in Leukemia. Cancer Research, 2015, 75, 2146-2150.	0.9	20
46	Chronic Myeloid Leukemia: Advances in Understanding Disease Biology and Mechanisms of Resistance to Tyrosine Kinase Inhibitors. Current Hematologic Malignancy Reports, 2015, 10, 158-166.	2.3	39
47	Structural insight into selectivity and resistance profiles of ROS1 tyrosine kinase inhibitors. Proceedings of the National Academy of Sciences of the United States of America, 2015, 112, E5381-90.	7.1	93
48	Src and STAT3 inhibitors synergize to promote tumor inhibition in renal cell carcinoma. Oncotarget, 2015, 6, 44675-44687.	1.8	27
49	A therapeutically targetable mechanism of BCR-ABL–independent imatinib resistance in chronic myeloid leukemia. Science Translational Medicine, 2014, 6, 252ra121.	12.4	105
50	BCR-ABL1 Compound Mutations Combining Key Kinase Domain Positions Confer Clinical Resistance to Ponatinib in Ph Chromosome-Positive Leukemia. Cancer Cell, 2014, 26, 428-442.	16.8	292
51	Antagonism of SET Using OP449 Enhances the Efficacy of Tyrosine Kinase Inhibitors and Overcomes Drug Resistance in Myeloid Leukemia. Clinical Cancer Research, 2014, 20, 2092-2103.	7.0	108
52	BCR-ABL1 promotes leukemia by converting p27 into a cytoplasmic oncoprotein. Blood, 2014, 124, 3260-3273.	1.4	20
53	BCR-ABL1 compound mutations in tyrosine kinase inhibitor–resistant CML: frequency and clonal relationships. Blood, 2013, 121, 489-498.	1.4	187
54	Threshold Levels of ABL Tyrosine Kinase Inhibitors Retained in Chronic Myeloid Leukemia Cells Determine Their Commitment to Apoptosis. Cancer Research, 2013, 73, 3356-3370.	0.9	26

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55	Oncogenic (i) CSF3R (i) Mutations in Chronic Neutrophilic Leukemia and Atypical CML. New England Journal of Medicine, 2013, 368, 1781-1790.	27.0	499
56	HitWalker: variant prioritization for personalized functional cancer genomics. Bioinformatics, 2013, 29, 509-510.	4.1	9
57	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. Cancer Research, 2013, 73, 5775-5786.	0.9	22
58	Foretinib is a potent inhibitor of oncogenic ROS1 fusion proteins. Proceedings of the National Academy of Sciences of the United States of America, 2013, 110, 19519-19524.	7.1	106
59	Integrating in vitro sensitivity and dose-response slope is predictive of clinical response to ABL kinase inhibitors in chronic myeloid leukemia. Blood, 2013, 122, 3331-3334.	1.4	10
60	Kinase Inhibitor Therapy in CML: It's What's Inside That Counts. Oncotarget, 2013, 4, 1332-1333.	1.8	1
61	The BCR-ABL35INS insertion/truncation mutant is kinase-inactive and does not contribute to tyrosine kinase inhibitor resistance in chronic myeloid leukemia. Blood, 2011, 118, 5250-5254.	1.4	37
62	Nilotinib and MEK Inhibitors Induce Synthetic Lethality through Paradoxical Activation of RAF in Drug-Resistant Chronic Myeloid Leukemia. Cancer Cell, 2011, 20, 715-727.	16.8	107
63	The ABL Switch Control Inhibitor DCC-2036 Is Active against the Chronic Myeloid Leukemia Mutant BCR-ABLT315I and Exhibits a Narrow Resistance Profile. Cancer Research, 2011, 71, 3189-3195.	0.9	91
64	Targeting the BCR-ABL Signaling Pathway in Therapy-Resistant Philadelphia Chromosome-Positive Leukemia. Clinical Cancer Research, 2011, 17, 212-221.	7.0	127
65	Wnt/Ca2+/NFAT Signaling Maintains Survival of Ph+ Leukemia Cells upon Inhibition of Bcr-Abl. Cancer Cell, 2010, 18, 74-87.	16.8	164
66	The function of the pleckstrin homology domain in BCR–ABL-mediated leukemogenesis. Leukemia, 2010, 24, 226-229.	7.2	12
67	AP24534, a Pan-BCR-ABL Inhibitor for Chronic Myeloid Leukemia, Potently Inhibits the T315I Mutant and Overcomes Mutation-Based Resistance. Cancer Cell, 2009, 16, 401-412.	16.8	1,050
68	High-throughput mutational screen of the tyrosine kinome in chronic myelomonocytic leukemia. Leukemia, 2009, 23, 406-409.	7.2	15
69	Acute dasatinib exposure commits Bcr-Abl–dependent cells to apoptosis. Blood, 2009, 114, 3459-3463.	1.4	54
70	High-throughput sequencing screen reveals novel, transforming RAS mutations in myeloid leukemia patients. Blood, 2009, 113, 1749-1755.	1.4	119
71	An activating KRAS mutation in imatinib-resistant chronic myeloid leukemia. Leukemia, 2008, 22, 2269-2272.	7.2	32
72	New Strategies for the First-Line Treatment of Chronic Myeloid Leukemia: Can Resistance Be Avoided?. Clinical Lymphoma and Myeloma, 2008, 8, S107-S117.	1.4	5

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73	SGX393 inhibits the CML mutant Bcr-Abl ^{T315I} and preempts <i>in vitro</i> resistance when combined with nilotinib or dasatinib. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 5507-5512.	7.1	84
74	Persistent LYN Signaling in Imatinib-Resistant, BCR-ABL–Independent Chronic Myelogenous Leukemia. Journal of the National Cancer Institute, 2008, 100, 908-909.	6.3	26
75	New Bcr-Abl inhibitors in chronic myeloid leukemia: keeping resistance in check. Expert Opinion on Investigational Drugs, 2008, 17, 865-878.	4.1	58
76	High-throughput sequence analysis of the tyrosine kinome in acute myeloid leukemia. Blood, 2008, 111, 4788-4796.	1.4	84
77	Growth Arrest of BCR-ABL Positive Cells with a Sequence-Specific Polyamide-Chlorambucil Conjugate. PLoS ONE, 2008, 3, e3593.	2.5	9
78	Bcr-Abl kinase domain mutations, drug resistance, and the road to a cure for chronic myeloid leukemia. Blood, 2007, 110, 2242-2249.	1.4	590
79	GATA-2 functions downstream of BMPs and CaM KIV in ectodermal cells during primitive hematopoiesis. Developmental Biology, 2007, 310, 454-469.	2.0	23
80	Bcr-Abl Kinase Domain Mutations and the Unsettled Problem of Bcr-AblT315I: Looking into the Future of Controlling Drug Resistance in Chronic Myeloid Leukemia. Clinical Lymphoma and Myeloma, 2007, 7, S120-S130.	1.4	22
81	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. Blood, 2006, 108, 2332-2338.	1.4	368
82	Activating alleles of JAK3 in acute megakaryoblastic leukemia. Cancer Cell, 2006, 10, 65-75.	16.8	295
83	Inhibition of T315I Bcr-Abl and Other Imatinib-Resistant Bcr-Abl Mutants by the Selective Abl Kinase Inhibitor SGX70393 Blood, 2006, 108, 1373-1373.	1.4	3
84	Identification of Tyrosine Kinase Mutations by Large-Scale DNA Sequencing in Patients with Chronic Myelomonocytic Leukemia/Atypical Chronic Myeloid Leukemia Blood, 2006, 108, 3606-3606.	1.4	0
85	A New Generation of Drugs in Cancer Treatment: Molecularly Targeted Therapies. , 0, , 193-221.		O