Paul W Manley

List of Publications by Year
in descending order

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41 papers	7,973 citations	212478 28 h-index	325983 40 g-index
43 all docs	43 docs citations	43 times ranked	8816 citing authors

#	Article	IF	Citations
1	A kinase inhibitor which specifically targets the ABL myristate pocket (STAMP), but unlike asciminib crosses the bloodâ€“brain barrier. Bioorganic and Medicinal Chemistry Letters, 2022, 59, 128577.	1.0	2
2	In Vivo Evaluation of Fibroblast Growth Factor Receptor Inhibition in Mouse Xenograft Models of Gastrointestinal Stromal Tumor. Biomedicines, 2022, 10, 1135.	1.4	4
3	Evidence supporting that the attribution of first success in use of arsenic for the treatment of leukaemia should be to David Lissauer (1836â€“1892). Leukemia, 2022, , .	3.3	0
4	The specificity of asciminib, a potential treatment for chronic myeloid leukemia, as a myristate-pocket binding ABL inhibitor and analysis of its interactions with mutant forms of BCR-ABL1 kinase. Leukemia Research, 2020, 98, 106458.	0.4	68
5	Investigations into the Potential Role of Metabolites on the Anti-Leukemic Activity of Imatinib, Nilotinib and Midostaurin. Chimia, 2019, 73, 561.	0.3	5
6	Tyrosine kinase inhibitors relax pulmonary arteries in human and murine precision-cut lung slices. Respiratory Research, 2019, 20, 111.	1.4	19
7	Spotlight on midostaurin in the treatment of FLT3-mutated acute myeloid leukemia and systemic mastocytosis: design, development, and potential place in therapy. OncoTargets and Therapy, 2018, Volume 11, 175-182.	1.0	15
8	Midostaurin: its odyssey from discovery to approval for treating acute myeloid leukemia and advanced systemic mastocytosis. Blood Advances, 2018, 2, 444-453.	2.5	115
9	Discovery of Asciminib (ABL001), an Allosteric Inhibitor of the Tyrosine Kinase Activity of BCR-ABL1. Journal of Medicinal Chemistry, 2018, 61, 8120-8135.	2.9	275
10	Comparison of the Kinase Profile of Midostaurin (Rydapt) with That of Its Predominant Metabolites and the Potential Relevance of Some Newly Identified Targets to Leukemia Therapy. Biochemistry, 2018, 57, 5576-5590.	1.2	21
11	Progress in the Discovery of BCR-ABL Kinase Inhibitors for the Treatment of Leukemia. Topics in Medicinal Chemistry, 2017, , 1-37.	0.4	5
12	Imatinib relaxes the pulmonary venous bed of guinea pigs. Respiratory Research, 2017, 18, 32.	1.4	17
13	A Novel Potent Oral Series of VEGFR2 Inhibitors Abrogate Tumor Growth by Inhibiting Angiogenesis. Journal of Medicinal Chemistry, 2016, 59, 132-146.	2.9	35
14	Phosphoinositide 3-Kinase Inhibitors Combined with Imatinib in Patient-Derived Xenograft Models of Gastrointestinal Stromal Tumors: Rationale and Efficacy. Clinical Cancer Research, 2014, 20, 6071-6082.	3.2	45
15	Efficacy and selectivity of nilotinib on NF1-associated tumors in vitro. Journal of Neuro-Oncology, 2014, 116, 231-236.	1.4	10
16	Imaging the intracellular distribution of tyrosine kinase inhibitors in living cells with quantitative hyperspectral stimulated Raman scattering. Nature Chemistry, 2014, 6, 614-622.	6.6	243
17	The kinetic deuterium isotope effect as applied to metabolic deactivation of imatinib to the des-methyl metabolite, CGP74588. Bioorganic and Medicinal Chemistry, 2013, 21, 3231-3239.	1.4	42
18	KIT Signaling Governs Differential Sensitivity of Mature and Primitive CML Progenitors to Tyrosine Kinase Inhibitors. Cancer Research, 2013, 73, 5775-5786.	0.4	22

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19	Modulation of Activation-Loop Phosphorylation by JAK Inhibitors Is Binding Mode Dependent. Cancer Discovery, 2012, 2, 512-523.	7.7	106
20	What do kinase inhibition profiles tell us about tyrosine kinase inhibitors used for the treatment of CML?. Leukemia Research, 2012, 36, 253-261.	0.4	13
21	Structural resemblances and comparisons of the relative pharmacological properties of imatinib and nilotinib. Bioorganic and Medicinal Chemistry, 2010, 18, 6977-6986.	1.4	141
22	Extended kinase profile and properties of the protein kinase inhibitor nilotinib. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2010, 1804, 445-453.	1.1	199
23	Targeting Bcrâ€“Abl by combining allosteric with ATP-binding-site inhibitors. Nature, 2010, 463, 501-506.	13.7	525
24	Functional Activity of the OCT-1 Protein Is Predictive of Long-Term Outcome in Patients With Chronic-Phase Chronic Myeloid Leukemia Treated With Imatinib. Journal of Clinical Oncology, 2010, 28, 2761-2767.	0.8	167
25	Comparative In Vitro Cellular Data Alone Are Insufficient to Predict Clinical Responses and Guide the Choice of BCR-ABL Inhibitor for Treating Imatinib-Resistant Chronic Myeloid Leukemia. Journal of Clinical Oncology, 2010, 28, e169-e171.	0.8	48
26	Inhibition of Chronic Myeloid Leukemia Stem Cells by the Combination of the Hedgehog Pathway Inhibitor LDE225 with Nilotinib. Blood, 2010, 116, 514-514.	0.6	8
27	The interplay of structural information and functional studies in kinase drug design: insights from BCR-Abl. Current Opinion in Cell Biology, 2009, 21, 288-295.	2.6	54
28	Inhibition of collagen-induced discoidin domain receptor 1 and 2 activation by imatinib, nilotinib and dasatinib. European Journal of Pharmacology, 2008, 599, 44-53.	1.7	237
29	Solution Conformations and Dynamics of ABL Kinase-Inhibitor Complexes Determined by NMR Substantiate the Different Binding Modes of Imatinib/Nilotinib and Dasatinib. Journal of Biological Chemistry, 2008, 283, 18292-18302.	1.6	183
30	Structural biology contributions to the discovery of drugs to treat chronic myelogenous leukaemia. Acta Crystallographica Section D: Biological Crystallography, 2007, 63, 80-93.	2.5	215
31	OCT-1â€“mediated influx is a key determinant of the intracellular uptake of imatinib but not nilotinib (AMN107): reduced OCT-1 activity is the cause of low in vitro sensitivity to imatinib. Blood, 2006, 108, 697-704.	0.6	413
32	Allosteric inhibitors of Bcr-ablâ€“dependent cell proliferation. Nature Chemical Biology, 2006, 2, 95-102.	3.9	349
33	Nilotinib in Imatinib-Resistant CML and Philadelphia Chromosomeâ€“Positive ALL. New England Journal of Medicine, 2006, 354, 2542-2551.	13.9	1,253
34	Characterization of AMN107, a selective inhibitor of native and mutant Bcr-Abl. Cancer Cell, 2005, 7, 129-141.	7.7	1,387
35	The Crystal Structure of a c-Src Complex in an Active Conformation Suggests Possible Steps in c-Src Activation. Structure, 2005, 13, 861-871.	1.6	304
36	Imatinib (STI571) Resistance in Chronic Myelogenous Leukemia: Molecular Basis of the Underlying Mechanisms and Potential Strategies for Treatment. Mini-Reviews in Medicinal Chemistry, 2004, 4, 285-299.	1.1	152

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37	Urea derivatives of STI571 as inhibitors of Bcr-Abl and PDGFR kinases. Bioorganic and Medicinal Chemistry Letters, 2004, 14, 5793-5797.	1.0	64
38	Anthranilic Acid Amides:Â A Novel Class of Antiangiogenic VEGF Receptor Kinase Inhibitors. Journal of Medicinal Chemistry, 2002, 45, 5687-5693.	2.9	101
39	Inhibition of mutant FLT3 receptors in leukemia cells by the small molecule tyrosine kinase inhibitor PKC412. Cancer Cell, 2002, 1, 433-443.	7.7	574
40	Tyrosine kinase inhibitors: From rational design to clinical trials. Medicinal Research Reviews, 2001, 21, 499-512.	5.0	307
41	New Anilinophthalazines as Potent and Orally Well Absorbed Inhibitors of the VEGF Receptor Tyrosine Kinases Useful as Antagonists of Tumor-Driven Angiogenesis. Journal of Medicinal Chemistry, 2000, 43, 2310-2323.	2.9	224