Markus A Seeliger

List of Publications by Year
in descending order

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56 papers	4,673 citations	172457 29 h-index	168389 53 g-index
61 all docs	61 docs citations	61 times ranked	6029 citing authors

#	Article	IF	Citations
1	How Does a Drug Molecule Find Its Target Binding Site?. Journal of the American Chemical Society, 2011, 133, 9181-9183.	13.7	564
2	Activation of tyrosine kinases by mutation of the gatekeeper threonine. Nature Structural and Molecular Biology, 2008, 15, 1109-1118.	8.2	366
3	Structure of the Kinase Domain of an Imatinib-Resistant Abl Mutant in Complex with the Aurora Kinase Inhibitor VX-680. Cancer Research, 2006, 66, 1007-1014.	0.9	282
4	Catalytic Control in the EGF Receptor and Its Connection to General Kinase Regulatory Mechanisms. Molecular Cell, 2011, 42, 9-22.	9.7	265
5	A conserved protonation-dependent switch controls drug binding in the Abl kinase. Proceedings of the National Academy of Sciences of the United States of America, 2009, 106, 139-144.	7.1	240
6	A conserved mechanism of DEAD-box ATPase activation by nucleoporins and InsP6 in mRNA export. Nature, 2011, 472, 238-242.	27.8	214
7	Anti-diabetic activity of insulin-degrading enzyme inhibitors mediated by multiple hormones. Nature, 2014, 511, 94-98.	27.8	207
8	High yield bacterial expression of active c-Abl and c-Src tyrosine kinases. Protein Science, 2005, 14, 3135-3139.	7.6	206
9	c-Src Binds to the Cancer Drug Imatinib with an Inactive Abl/c-Kit Conformation and a Distributed Thermodynamic Penalty. Structure, 2007, 15, 299-311.	3.3	203
10	Organization of the SH3-SH2 Unit in Active and Inactive Forms of the c-Abl Tyrosine Kinase. Molecular Cell, 2006, 21, 787-798.	9.7	192
11	Divergent allosteric control of the IRE1Î± endoribonuclease using kinase inhibitors. Nature Chemical Biology, 2012, 8, 982-989.	8.0	175
12	Mechanism of CDK5/p25 Binding by CDK Inhibitors. Journal of Medicinal Chemistry, 2005, 48, 671-679.	6.4	173
13	Equally Potent Inhibition of c-Src and Abl by Compounds that Recognize Inactive Kinase Conformations. Cancer Research, 2009, 69, 2384-2392.	0.9	134
14	Structural Basis for the Recognition of c-Src by Its Inactivator Csk. Cell, 2008, 134, 124-134.	28.9	119
15	A dynamically coupled allosteric network underlies binding cooperativity in Src kinase. Nature Communications, 2015, 6, 5939.	12.8	101
16	Targeting Conformational Plasticity of Protein Kinases. ACS Chemical Biology, 2015, 10, 190-200.	3.4	87
17	Three Different Binding Sites of Cks1 Are Required for p27-Ubiquitin Ligation. Journal of Biological Chemistry, 2002, 277, 42233-42240.	3.4	80
18	Structural and Functional Analysis of the Allosteric Inhibition of IRE1Î± with ATP-Competitive Ligands. ACS Chemical Biology, 2016, 11, 2195-2205.	3.4	75

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19	Tuning a Three-Component Reaction For Trapping Kinase Substrate Complexes. Journal of the American Chemical Society, 2008, 130, 17568-17574.	13.7	67
20	A novel activating, germline JAK2 mutation, JAK2R564Q, causes familial essential thrombocytosis. Blood, 2014, 123, 1059-1068.	1.4	62
21	Discovery of a small-molecule type II inhibitor of wild-type and gatekeeper mutants of BCR-ABL, PDGFRÎ±, Kit, and Src kinases: novel type II inhibitor of gatekeeper mutants. Blood, 2010, 115, 4206-4216.	1.4	61
22	Highly specific, bisubstrate-competitive Src inhibitors from DNA-templated macrocycles. Nature Chemical Biology, 2012, 8, 366-374.	8.0	61
23	What Makes a Kinase Promiscuous for Inhibitors?. Cell Chemical Biology, 2019, 26, 390-399.e5.	5.2	59
24	Conformation-Selective Analogues of Dasatinib Reveal Insight into Kinase Inhibitor Binding and Selectivity. ACS Chemical Biology, 2016, 11, 1296-1304.	3.4	58
25	N-Myristoylated c-Abl Tyrosine Kinase Localizes to the Endoplasmic Reticulum upon Binding to an Allosteric Inhibitor. Journal of Biological Chemistry, 2009, 284, 29005-29014.	3.4	52
26	A Novel In Vitro CypD-Mediated p53 Aggregation Assay Suggests a Model for Mitochondrial Permeability Transition by Chaperone Systems. Journal of Molecular Biology, 2016, 428, 4154-4167.	4.2	45
27	Survey of solution dynamics in Src kinase reveals allosteric cross talk between the ligand binding and regulatory sites. Nature Communications, 2017, 8, 2160.	12.8	38
28	An Open Library of Human Kinase Domain Constructs for Automated Bacterial Expression. Biochemistry, 2018, 57, 4675-4689.	2.5	37
29	Substrate-selective inhibitors that reprogram the activity of insulin-degrading enzyme. Nature Chemical Biology, 2019, 15, 565-574.	8.0	36
30	Targeting the Hemopexin-like Domain of Latent Matrix Metalloproteinase-9 (proMMP-9) with a Small Molecule Inhibitor Prevents the Formation of Focal Adhesion Junctions. ACS Chemical Biology, 2017, 12, 2788-2803.	3.4	32
31	IRE1Î² negatively regulates IRE1Î± signaling in response to endoplasmic reticulum stress. Journal of Cell Biology, 2020, 219, .	5.2	31
32	Conformation-Selective Inhibitors Reveal Differences in the Activation and Phosphate-Binding Loops of the Tyrosine Kinases Abl and Src. ACS Chemical Biology, 2013, 8, 2734-2743.	3.4	30
33	Mutation in Abl kinase with altered drug-binding kinetics indicates a novel mechanism of imatinib resistance. Proceedings of the National Academy of Sciences of the United States of America, 2021, 118, .	7.1	30
34	Selective Targeting of SH2 Domainâ€“Phosphotyrosine Interactions of Src Family Tyrosine Kinases with Monobodies. Journal of Molecular Biology, 2017, 429, 1364-1380.	4.2	25
35	Role of Conformational Heterogeneity in Domain Swapping and Adapter Function of the Cks Proteins. Journal of Biological Chemistry, 2005, 280, 30448-30459.	3.4	23
36	Activation of Ubiquitin Ligase SCFSkp2 by Cks1: Insights from Hydrogen Exchange Mass Spectrometry. Journal of Molecular Biology, 2006, 363, 673-686.	4.2	23

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37	Folding and Association of the Human Cell Cycle Regulatory Proteins ckshs1 and ckshs2. Biochemistry, 2002, 41, 1202-1210.	2.5	21
38	Cooperative organization in a macromolecular complex. Nature Structural and Molecular Biology, 2003, 10, 718-724.	8.2	21
39	Protein Flexibility and Dissociation Pathway Differentiation Can Explain Onset of Resistance Mutations in Kinases**. Angewandte Chemie - International Edition, 2022, 61, e202200983.	13.8	21
40	Comparative Analysis of Mutant Tyrosine Kinase Chemical Rescue. Biochemistry, 2009, 48, 3378-3386.	2.5	19
41	Analysis of DEAD-Box Proteins in mRNA Export. Methods in Enzymology, 2012, 511, 239-254.	1.0	17
42	Folding and Fibril Formation of the Cell Cycle Protein Cks1. Journal of Biological Chemistry, 2006, 281, 18816-18824.	3.4	15
43	How ATP-Competitive Inhibitors Allosterically Modulate Tyrosine Kinases That Contain a Src-like Regulatory Architecture. ACS Chemical Biology, 2020, 15, 2005-2016.	3.4	15
44	An allosteric add-on. Nature Chemical Biology, 2014, 10, 796-797.	8.0	14
45	Structural and Biochemical Basis for Intracellular Kinase Inhibition by Src-specific Peptidic Macrocycles. Cell Chemical Biology, 2016, 23, 1103-1112.	5.2	12
46	Enzymatic Activity and Thermodynamic Stability of Biliverdin IXÎ² Reductase Are Maintained by an Active Site Serine. Chemistry - A European Journal, 2017, 23, 1891-1900.	3.3	12
47	Prolyl isomerization of FAAP20 catalyzed by PIN1 regulates the Fanconi anemia pathway. PLoS Genetics, 2019, 15, e1007983.	3.5	9
48	Dynamically Coupled Residues within the SH2 Domain of FYN Are Key to Unlocking Its Activity. Structure, 2016, 24, 1947-1959.	3.3	8
49	Extended DNA-binding interfaces beyond the canonical SAP domain contribute to the function of replication stress regulator SDE2 at DNA replication forks. Journal of Biological Chemistry, 2022, 298, 102268.	3.4	8
50	A MAPK Scaffold Lends a Helping Hand. Cell, 2009, 136, 994-996.	28.9	6
51	Validation of an Allosteric Binding Site of Src Kinase Identified by Unbiased Ligand Binding Simulations. Journal of Molecular Biology, 2022, 434, 167628.	4.2	6
52	KA1 Domains: Unity in Mechanistic Diversity. Structure, 2018, 26, 1045-1047.	3.3	2
53	Resistance to kinase inhibition through shortened target engagement. Molecular and Cellular Oncology, 2022, 9, 2029999.	0.7	1
54	More Diversity Yields a Clearer Picture into the Architecture of the Protein Kinase Domain. Cell Systems, 2018, 7, 356-357.	6.2	0

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55	Protein Flexibility and Dissociation Pathway Differentiation Can Explain Onset Of Resistance Mutations in Kinases. Angewandte Chemie, 0, , .	2.0	0
56	Kinases on Double Duty: A Review of UniProtKB Annotated Bifunctionality within the Kinome. Biomolecules, 2022, 12, 685.	4.0	0