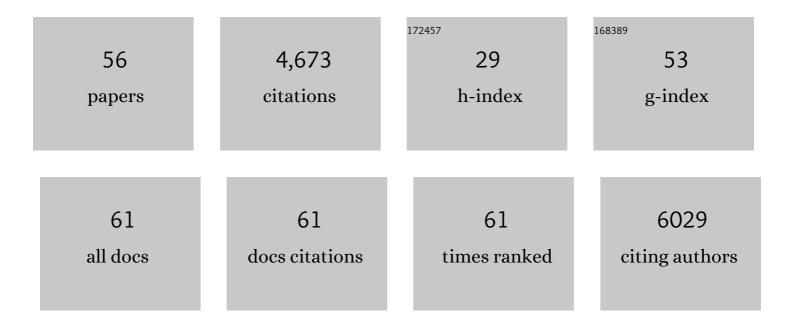
## Markus A Seeliger

List of Publications by Year in descending order

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#	Article	IF	CITATIONS
1	Resistance to kinase inhibition through shortened target engagement. Molecular and Cellular Oncology, 2022, 9, 2029999.	0.7	1
2	Protein Flexibility and Dissociation Pathway Differentiation Can Explain Onset of Resistance Mutations in Kinases**. Angewandte Chemie - International Edition, 2022, 61, e202200983.	13.8	21
3	Kinases on Double Duty: A Review of UniProtKB Annotated Bifunctionality within the Kinome. Biomolecules, 2022, 12, 685.	4.0	0
4	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
5	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
6	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
7	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
8	IRE1β negatively regulates IRE1α signaling in response to endoplasmic reticulum stress. Journal of Cell Biology, 2020, 219, .	5.2	31
9	Substrate-selective inhibitors that reprogram the activity of insulin-degrading enzyme. Nature Chemical Biology, 2019, 15, 565-574.	8.0	36
10	Prolyl isomerization of FAAP20 catalyzed by PIN1 regulates the Fanconi anemia pathway. PLoS Genetics, 2019, 15, e1007983.	3.5	9
11	What Makes a Kinase Promiscuous for Inhibitors?. Cell Chemical Biology, 2019, 26, 390-399.e5.	5.2	59
12	More Diversity Yields a Clearer Picture into the Architecture of the Protein Kinase Domain. Cell Systems, 2018, 7, 356-357.	6.2	0
13	KA1 Domains: Unity in Mechanistic Diversity. Structure, 2018, 26, 1045-1047.	3.3	2
14	An Open Library of Human Kinase Domain Constructs for Automated Bacterial Expression. Biochemistry, 2018, 57, 4675-4689.	2.5	37
15	Selective Targeting of SH2 Domain–Phosphotyrosine Interactions of Src Family Tyrosine Kinases with Monobodies. Journal of Molecular Biology, 2017, 429, 1364-1380.	4.2	25
16	Enzymatic Activity and Thermodynamic Stability of Biliverdin IXβ Reductase Are Maintained by an Active Site Serine. Chemistry - A European Journal, 2017, 23, 1891-1900.	3.3	12
17	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
18	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

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19	Structural and Biochemical Basis for Intracellular Kinase Inhibition by Src-specific Peptidic Macrocycles. Cell Chemical Biology, 2016, 23, 1103-1112.	5.2	12
20	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
21	Dynamically Coupled Residues within the SH2 Domain of FYN Are Key to Unlocking Its Activity. Structure, 2016, 24, 1947-1959.	3.3	8
22	Structural and Functional Analysis of the Allosteric Inhibition of IRE1α with ATP-Competitive Ligands. ACS Chemical Biology, 2016, 11, 2195-2205.	3.4	75
23	Conformation-Selective Analogues of Dasatinib Reveal Insight into Kinase Inhibitor Binding and Selectivity. ACS Chemical Biology, 2016, 11, 1296-1304.	3.4	58
24	A dynamically coupled allosteric network underlies binding cooperativity in Src kinase. Nature Communications, 2015, 6, 5939.	12.8	101
25	Targeting Conformational Plasticity of Protein Kinases. ACS Chemical Biology, 2015, 10, 190-200.	3.4	87
26	Anti-diabetic activity of insulin-degrading enzyme inhibitors mediated by multiple hormones. Nature, 2014, 511, 94-98.	27.8	207
27	An allosteric add-on. Nature Chemical Biology, 2014, 10, 796-797.	8.0	14
28	A novel activating, germline JAK2 mutation, JAK2R564Q, causes familial essential thrombocytosis. Blood, 2014, 123, 1059-1068.	1.4	62
29	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
30	Divergent allosteric control of the IRE1α endoribonuclease using kinase inhibitors. Nature Chemical Biology, 2012, 8, 982-989.	8.0	175
31	Analysis of DEAD-Box Proteins in mRNA Export. Methods in Enzymology, 2012, 511, 239-254.	1.0	17
32	Highly specific, bisubstrate-competitive Src inhibitors from DNA-templated macrocycles. Nature Chemical Biology, 2012, 8, 366-374.	8.0	61
33	Catalytic Control in the EGF Receptor and Its Connection to General Kinase Regulatory Mechanisms. Molecular Cell, 2011, 42, 9-22.	9.7	265
34	How Does a Drug Molecule Find Its Target Binding Site?. Journal of the American Chemical Society, 2011, 133, 9181-9183.	13.7	564
35	A conserved mechanism of DEAD-box ATPase activation by nucleoporins and InsP6 in mRNA export. Nature, 2011, 472, 238-242.	27.8	214
36	Discovery of a small-molecule type II inhibitor of wild-type and gatekeeper mutants of BCR-ABL, PDGFRα, Kit, and Src kinases: novel type II inhibitor of gatekeeper mutants. Blood, 2010, 115, 4206-4216.	1.4	61

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37	A conserved protonation-dependent switch controls drug binding in the Abl kinase. Proceedings of the United States of America, 2009, 106, 139-144.	7.1	240
38	Equally Potent Inhibition of c-Src and Abl by Compounds that Recognize Inactive Kinase Conformations. Cancer Research, 2009, 69, 2384-2392.	0.9	134
39	Comparative Analysis of Mutant Tyrosine Kinase Chemical Rescue. Biochemistry, 2009, 48, 3378-3386.	2.5	19
40	A MAPK Scaffold Lends a Helping Hand. Cell, 2009, 136, 994-996.	28.9	6
41	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
42	Activation of tyrosine kinases by mutation of the gatekeeper threonine. Nature Structural and Molecular Biology, 2008, 15, 1109-1118.	8.2	366
43	Tuning a Three-Component Reaction For Trapping Kinase Substrate Complexes. Journal of the American Chemical Society, 2008, 130, 17568-17574.	13.7	67
44	Structural Basis for the Recognition of c-Src by Its Inactivator Csk. Cell, 2008, 134, 124-134.	28.9	119
45	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
46	Activation of Ubiquitin Ligase SCFSkp2 by Cks1: Insights from Hydrogen Exchange Mass Spectrometry. Journal of Molecular Biology, 2006, 363, 673-686.	4.2	23
47	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
48	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
49	Folding and Fibril Formation of the Cell Cycle Protein Cks1. Journal of Biological Chemistry, 2006, 281, 18816-18824.	3.4	15
50	High yield bacterial expression of active c-Abl and c-Src tyrosine kinases. Protein Science, 2005, 14, 3135-3139.	7.6	206
51	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
52	Mechanism of CDK5/p25 Binding by CDK Inhibitors. Journal of Medicinal Chemistry, 2005, 48, 671-679.	6.4	173
53	Cooperative organization in a macromolecular complex. Nature Structural and Molecular Biology, 2003, 10, 718-724.	8.2	21
54	Three Different Binding Sites of Cks1 Are Required for p27-Ubiquitin Ligation. Journal of Biological Chemistry, 2002, 277, 42233-42240.	3.4	80

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55	Folding and Association of the Human Cell Cycle Regulatory Proteins ckshs1 and ckshs2. Biochemistry, 2002, 41, 1202-1210.	2.5	21
56	Protein Flexibility and Dissociation Pathway Differentiation Can Explain Onset Of Resistance Mutations in Kinases. Angewandte Chemie, 0, , .	2.0	0