Oliver D Hantschel

List of Publications by Citations

80 papers	5,378 citations	32 h-index	73 g-index
93 ext. papers	6,059 ext. citations	10.2 avg, IF	5.38 L-index

#	Paper	IF	Citations
80	Structural basis for the autoinhibition of c-Abl tyrosine kinase. *Cell*, **2003**, 112, 859-71	56.2	661
79	Chemical proteomic profiles of the BCR-ABL inhibitors imatinib, nilotinib, and dasatinib reveal novel kinase and nonkinase targets. *Blood*, **2007**, 110, 4055-63	2.2	538
78	Regulation of the c-Abl and Bcr-Abl tyrosine kinases. *Nature Reviews Molecular Cell Biology*, **2004**, 5, 33-44	28.7	380
77	A myristoyl/phosphotyrosine switch regulates c-Abl. *Cell*, **2003**, 112, 845-57	56.2	332
76	An efficient tandem affinity purification procedure for interaction proteomics in mammalian cells. *Nature Methods*, **2006**, 3, 1013-9	21.6	326
75	The Btk tyrosine kinase is a major target of the Bcr-Abl inhibitor dasatinib. *Proceedings of the National Academy of Sciences of the United States of America*, **2007**, 104, 13283-8	11.5	242
74	Global target profile of the kinase inhibitor bosutinib in primary chronic myeloid leukemia cells. *Leukemia*, **2009**, 23, 477-85	10.7	216
73	The DEAD-box helicase DDX3X is a critical component of the TANK-binding kinase 1-dependent innate immune response. *EMBO Journal*, **2008**, 27, 2135-46	13	210
72	Target spectrum of the BCR-ABL inhibitors imatinib, nilotinib and dasatinib. *Leukemia and Lymphoma*, **2008**, 49, 615-9	1.9	199
71	Organization of the SH3-SH2 unit in active and inactive forms of the c-Abl tyrosine kinase. *Molecular Cell*, **2006**, 21, 787-98	17.6	174
70	Structural coupling of SH2-kinase domains links Fes and Abl substrate recognition and kinase activation. *Cell*, **2008**, 134, 793-803	56.2	171
69	BCR-ABL uncouples canonical JAK2-STAT5 signaling in chronic myeloid leukemia. *Nature Chemical Biology*, **2012**, 8, 285-93	11.7	135
68	Charting the molecular network of the drug target Bcr-Abl. *Proceedings of the National Academy of Sciences of the United States of America*, **2009**, 106, 7414-9	11.5	130
67	c-Abl phosphorylates α-synuclein and regulates its degradation: implication for α-synuclein clearance and contribution to the pathogenesis of Parkinson's disease. *Human Molecular Genetics*, **2014**, 23, 2858-79	5.6	126
66	A potent and highly specific FN3 monobody inhibitor of the Abl SH2 domain. *Nature Structural and Molecular Biology*, **2010**, 17, 519-27	17.6	120
65	Targeting the SH2-kinase interface in Bcr-Abl inhibits leukemogenesis. *Cell*, **2011**, 147, 306-19	56.2	102
64	Structure, regulation, signaling, and targeting of abl kinases in cancer. *Genes and Cancer*, **2012**, 3, 436-46	2.9	86

63	Specificity and mechanism-of-action of the JAK2 tyrosine kinase inhibitors ruxolitinib and SAR302503 (TG101348). *Leukemia*, **2014**, 28, 404-7	10.7	80
62	The structure of the leukemia drug imatinib bound to human quinone reductase 2 (NQO2). *BMC Structural Biology*, **2009**, 9, 7	2.7	67
61	BCR-ABL SH3-SH2 domain mutations in chronic myeloid leukemia patients on imatinib. *Blood*, **2010**, 116, 3278-85	2.2	65
60	The growing arsenal of ATP-competitive and allosteric inhibitors of BCR-ABL. *Cancer Research*, **2012** , 72, 4890-5	10.1	62
59	Dissection of the BCR-ABL signaling network using highly specific monobody inhibitors to the SHP2 SH2 domains. *Proceedings of the National Academy of Sciences of the United States of America*, **2013**, 110, 14924-9	11.5	62
58	A comprehensive target selectivity survey of the BCR-ABL kinase inhibitor INNO-406 by kinase profiling and chemical proteomics in chronic myeloid leukemia cells. *Leukemia*, **2010**, 24, 44-50	10.7	58
57	Structural basis for the cytoskeletal association of Bcr-Abl/c-Abl. *Molecular Cell*, **2005**, 19, 461-73	17.6	57
56	Differential signaling networks of Bcr-Abl p210 and p190 kinases in leukemia cells defined by functional proteomics. *Leukemia*, **2017**, 31, 1502-1512	10.7	53
55	BioSITe: A Method for Direct Detection and Quantitation of Site-Specific Biotinylation. *Journal of Proteome Research*, **2018**, 17, 759-769	5.6	46
54	Mechanisms of resistance to BCR-ABL and other kinase inhibitors. *Biochimica Et Biophysica Acta - Proteins and Proteomics*, **2013**, 1834, 1449-59	4	45
53	Intrinsic differences between the catalytic properties of the oncogenic NUP214-ABL1 and BCR-ABL1 fusion protein kinases. *Leukemia*, **2008**, 22, 2208-16	10.7	39
52	Mig6 is a sensor of EGF receptor inactivation that directly activates c-Abl to induce apoptosis during epithelial homeostasis. *Developmental Cell*, **2012**, 23, 547-59	10.2	38
51	Single-molecule kinetic analysis of HP1-chromatin binding reveals a dynamic network of histone modification and DNA interactions. *Nucleic Acids Research*, **2017**, 45, 10504-10517	20.1	34
50	NUP214-ABL1-mediated cell proliferation in T-cell acute lymphoblastic leukemia is dependent on the LCK kinase and various interacting proteins. *Haematologica*, **2014**, 99, 85-93	6.6	34
49	Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. *Leukemia*, **2008**, 22, 1184-90	10.7	33
48	The SH2 domain of Abl kinases regulates kinase autophosphorylation by controlling activation loop accessibility. *Nature Communications*, **2014**, 5, 5470	17.4	28
47	Unexpected off-targets and paradoxical pathway activation by kinase inhibitors. *ACS Chemical Biology*, **2015**, 10, 234-45	4.9	27
46	Identification and Characterization of Tyrosine Kinase Nonreceptor 2 Mutations in Leukemia through Integration of Kinase Inhibitor Screening and Genomic Analysis. *Cancer Research*, **2016**, 76, 127-38	10.1	25

45	The chemokine interleukin-8 and the surface activation protein CD69 are markers for Bcr-Abl activity in chronic myeloid leukemia. *Molecular Oncology*, **2008**, 2, 272-81	7.9	25
44	Allosteric Inhibition of Bcr-Abl Kinase by High Affinity Monobody Inhibitors Directed to the Src Homology 2 (SH2)-Kinase Interface. *Journal of Biological Chemistry*, **2016**, 291, 8836-47	5.4	23
43	Structural and functional dissection of the DH and PH domains of oncogenic Bcr-Abl tyrosine kinase. *Nature Communications*, **2017**, 8, 2101	17.4	21
42	Crystal structure of an SH2-kinase construct of c-Abl and effect of the SH2 domain on kinase activity. *Biochemical Journal*, **2015**, 468, 283-91	3.8	19
41	Selective Targeting of SH2 Domain-Phosphotyrosine Interactions of Src Family Tyrosine Kinases with Monobodies. *Journal of Molecular Biology*, **2017**, 429, 1364-1380	6.5	18
40	Normal ABL1 is a tumor suppressor and therapeutic target in human and mouse leukemias expressing oncogenic ABL1 kinases. *Blood*, **2016**, 127, 2131-43	2.2	18
39	Targeted Protein Degradation through Cytosolic Delivery of Monobody Binders Using Bacterial Toxins. *ACS Chemical Biology*, **2019**, 14, 916-924	4.9	16
38	ATP Site Ligands Determine the Assembly State of the Abelson Kinase Regulatory Core via the Activation Loop Conformation. *Journal of the American Chemical Society*, **2018**, 140, 1863-1869	16.4	16
37	Selective inhibition of STAT3 signaling using monobodies targeting the coiled-coil and N-terminal domains. *Nature Communications*, **2020**, 11, 4115	17.4	16
36	HRD Motif as the Central Hub of the Signaling Network for Activation Loop Autophosphorylation in Abl Kinase. *Journal of Chemical Theory and Computation*, **2016**, 12, 5563-5574	6.4	16
35	Monobodies as enabling tools for structural and mechanistic biology. *Current Opinion in Structural Biology*, **2020**, 60, 167-174	8.1	15
34	Catenin-Dependent Signals Maintain BCR-ABL1 B Cell Acute Lymphoblastic Leukemia. *Cancer Cell*, **2019**, 35, 649-663.e10	24.3	14
33	BCR-ABL1 compound mutants display differential and dose-dependent responses to ponatinib. *Haematologica*, **2018**, 103, e10-e12	6.6	14
32	Kinase-templated abiotic reaction. *Chemical Science*, **2017**, 8, 5119-5125	9.4	12
31	Btk SH2-kinase interface is critical for allosteric kinase activation and its targeting inhibits B-cell neoplasms. *Nature Communications*, **2020**, 11, 2319	17.4	12
30	Alkaline phosphatase-fused repebody as a new format of immuno-reagent for an immunoassay. *Analytica Chimica Acta*, **2017**, 950, 184-191	6.6	11
29	NDEL1-PDGFRB fusion gene in a myeloid malignancy with eosinophilia associated with resistance to tyrosine kinase inhibitors. *Leukemia*, **2017**, 31, 237-240	10.7	10
28	BTK operates a phospho-tyrosine switch to regulate NLRP3 inflammasome activity. *Journal of Experimental Medicine*, **2021**, 218,	16.6	9

27	The phosphatase UBASH3B/Sts-1 is a negative regulator of Bcr-Abl kinase activity and leukemogenesis. *Leukemia*, **2019**, 33, 2319-2323	10.7	8
26	Unpaired Extracellular Cysteine Mutations of CSF3R Mediate Gain or Loss of Function. *Cancer Research*, **2017**, 77, 4258-4267	10.1	8
25	Monobodies as possible next-generation protein therapeutics - a perspective. *Swiss Medical Weekly*, **2017**, 147, w14545	3.1	6
24	Nilotinib as frontline and second-line therapy in chronic myeloid leukemia: open questions. *Critical Reviews in Oncology/Hematology*, **2012**, 82, 370-7	7	5
23	Precision Medicine in Hematology 2021: Definitions, Tools, Perspectives, and Open Questions. *HemaSphere*, **2021**, 5, e536	0.3	5
22	A Novel Fusion Gene NDEL1-Pdgfrb in a Patient with JMML with a New Variant of TKI-Resistant Mutation in the Kinase Domain of PDGFR *Blood*, **2014**, 124, 613-613	2.2	4
21	Rapid Screen for Tyrosine Kinase Inhibitor Resistance Mutations and Substrate Specificity. *ACS Chemical Biology*, **2019**, 14, 1888-1895	4.9	3
20	The central domain of the matrix protein of HIV-1: influence on protein structure and virus infectivity. *Biological Chemistry*, **2004**, 385, 303-13	4.5	3
19	NMR Assignment Reveals an alpha-Helical Fold for the F-Actin Binding Domain of Human Bcr-Abl/c-Abl. *Journal of Biomolecular NMR*, **2005**, 32, 335	3	3
18	C-Abl Phosphorylates Alpha-synuclein And Regulates Its Degradation, Implication For Alpha-synuclein Clearance And Contribution To The Pathogenesis Of Parkinson's Disease **2014**,		2
17	Kinase Regulation in Mycobacterium tuberculosis: Variations on a Theme. *Structure*, **2015**, 23, 975-6	5.2	2
16	Tuning SAS-6 architecture with monobodies impairs distinct steps of centriole assembly. *Nature Communications*, **2021**, 12, 3805	17.4	2
15	Targeting BCR-ABL and JAK2 in Ph+ ALL. *Blood*, **2015**, 125, 1362-3	2.2	1
14	Cell biology: a key driver of therapeutic innovation. *Journal of Cell Biology*, **2012**, 199, 571-5	7.3	1
13	Crizotinib acts as ABL1 inhibitor combining ATP-binding with allosteric inhibition and is active against native BCR-ABL1 and its resistance and compound mutants BCR-ABL1 and BCR-ABL1. *Annals of Hematology*, **2021**, 100, 2023-2029	3	1
12	Chronic myeloid leukemia.. *HemaSphere*, **2019**, 3, 47	0.3	1
11	The Bcr-Abl SH2-Kinase Domain Interface Is Critical for Leukemogenesis and An Additional Therapeutic Target in CML.. *Blood*, **2009**, 114, 37-37	2.2	0
10	CDK6 degradation hits Ph+ ALL hard. *Blood*, **2020**, 135, 1512-1514	2.2	

9	Allosterische Kinaseinhibitoren. *Onkologe*, **2017**, 23, 626-631	0.1
8	Mechanisms of Activation of Abl Family Kinases **2006**, 1-10	
7	A Subset of Chronic Myeloid Leukemia (CML) Patients on ABL Kinase Inhibitor Therapy Develop Point Mutations outside the BCR-ABL Kinase Domain That Decrease Drug Sensitivity and May Have a Role in Disease Progression.. *Blood*, **2006**, 108, 2188-2188	2.2
6	Characterization of BCR-ABL Deletion Mutants from Patients with Chronic Myeloid Leukemia.. *Blood*, **2007**, 110, 2936-2936	2.2
5	The SH2 Domain of BCR-ABL1 Regulates Kinase Autophosphorylation By Controlling Activation Loop Accessibility. *Blood*, **2014**, 124, 2209-2209	2.2
4	Comprehensive Analysis of the Structural, Biochemical and Signaling Differences of the p210 and p185 Isoforms of Bcr-Abl in CML and B-ALL. *Blood*, **2016**, 128, 4238-4238	2.2
3	Structural Positioning of the SH2 Domain Is Critical for Bcr-Abl Kinase Activity, Signal Transduction and Oncogenic Transformation. *Blood*, **2008**, 112, 569-569	2.2
2	Bcr-Abl Directly Activates Stat5 Independent of Jak2. *Blood*, **2010**, 116, 511-511	2.2
1	2016 International Symposium on Chemical Biology of the NCCR Chemical Biology Campus Biotech, Geneva 13-15.1.2016. *Chimia*, **2016**, 70, 215-9	1.3