Oliver D Hantschel

List of Publications by Year
in descending order

87 papers	6,507 citations	126708 33 h-index	69108 77 g-index
93 all docs	93 docs citations	93 times ranked	8284 citing authors

#	Article	IF	Citations
1	Synthesis of the <scp>l</scp>- and <scp>d</scp>-SH2 domain of the leukaemia oncogene Bcr-Abl. RSC Chemical Biology, 2022, 3, 1008-1012.	2.0	1
2	Precision Medicine in Hematology 2021: Definitions, Tools, Perspectives, and Open Questions. HemaSphere, 2021, 5, e536.	1.2	11
3	Tuning SAS-6 architecture with monobodies impairs distinct steps of centriole assembly. Nature Communications, 2021, 12, 3805.	5.8	3
4	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-ABL1T315I and BCR-ABL1T315I-E255K. Annals of Hematology, 2021, 100, 2023-2029.	0.8	6
5	BTK operates a phospho-tyrosine switch to regulate NLRP3 inflammasome activity. Journal of Experimental Medicine, 2021, 218, .	4.2	33
6	Selective inhibition of STAT3 signaling using monobodies targeting the coiled-coil and N-terminal domains. Nature Communications, 2020, 11, 4115.	5.8	36
7	CDK6 degradation hits Ph+ ALL hard. Blood, 2020, 135, 1512-1514.	0.6	0
8	Btk SH2-kinase interface is critical for allosteric kinase activation and its targeting inhibits B-cell neoplasms. Nature Communications, 2020, 11, 2319.	5.8	23
9	Monobodies as enabling tools for structural and mechanistic biology. Current Opinion in Structural Biology, 2020, 60, 167-174.	2.6	31
10	Rapid Screen for Tyrosine Kinase Inhibitor Resistance Mutations and Substrate Specificity. ACS Chemical Biology, 2019, 14, 1888-1895.	1.6	8
11	Î³-Catenin-Dependent Signals Maintain BCR-ABL1+ B Cell Acute Lymphoblastic Leukemia. Cancer Cell, 2019, 35, 649-663.e10.	7.7	20
12	Targeted Protein Degradation through Cytosolic Delivery of Monobody Binders Using Bacterial Toxins. ACS Chemical Biology, 2019, 14, 916-924.	1.6	29
13	The phosphatase UBASH3B/Sts-1 is a negative regulator of Bcr-Abl kinase activity and leukemogenesis. Leukemia, 2019, 33, 2319-2323.	3.3	10
14	Chronic myeloid leukemia. HemaSphere, 2019, 3, 47.	1.2	2
15	BioSITe: A Method for Direct Detection and Quantitation of Site-Specific Biotinylation. Journal of Proteome Research, 2018, 17, 759-769.	1.8	70
16	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.	6.6	28
17	BCR-ABL1 compound mutants display differential and dose-dependent responses to ponatinib. Haematologica, 2018, 103, e10-e12.	1.7	26
18	Differential signaling networks of Bcrâ€“Abl p210 and p190 kinases in leukemia cells defined by functional proteomics. Leukemia, 2017, 31, 1502-1512.	3.3	84

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19	NDEL1-PDGFRB fusion gene in a myeloid malignancy with eosinophilia associated with resistance to tyrosine kinase inhibitors. Leukemia, 2017, 31, 237-240.	3.3	11
20	Kinase-templated abiotic reaction. Chemical Science, 2017, 8, 5119-5125.	3.7	13
21	Molecular Simulations to Unravel the Allosteric Interplay between the SH2 Domain and A-loop Plasticity in Protein Kinases. Biophysical Journal, 2017, 112, 32a.	0.2	0
22	Selective Targeting of SH2 Domainâ€“Phosphotyrosine Interactions of Src Family Tyrosine Kinases with Monobodies. Journal of Molecular Biology, 2017, 429, 1364-1380.	2.0	25
23	Alkaline phosphatase-fused repebody as a new format of immuno-reagent for an immunoassay. Analytica Chimica Acta, 2017, 950, 184-191.	2.6	13
24	Unpaired Extracellular Cysteine Mutations of CSF3R Mediate Gain or Loss of Function. Cancer Research, 2017, 77, 4258-4267.	0.4	10
25	Structural and functional dissection of the DH and PH domains of oncogenic Bcr-Abl tyrosine kinase. Nature Communications, 2017, 8, 2101.	5.8	33
26	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.	6.5	49
27	Bcr-Abl: one kinase, two isoforms, two diseases. Oncotarget, 2017, 8, 78257-78258.	0.8	7
28	Monobodies as possible next-generation protein therapeutics â€“ a perspective. Swiss Medical Weekly, 2017, 147, w14545.	0.8	11
29	Abstract 532: Identification of unpaired cysteine-mediated gain and loss of function CSF3R extracellular mutations. , 2017, , .		0
30	2016 International Symposium on Chemical Biology of the NCCR Chemical Biology Campus Biotech, Geneva 13â€“15.1.2016. Chimia, 2016, 70, 215-219.	0.3	0
31	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, 8836-8847.	1.6	33
32	Normal ABL1 is a tumor suppressor and therapeutic target in human and mouse leukemias expressing oncogenic ABL1 kinases. Blood, 2016, 127, 2131-2143.	0.6	32
33	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.	2.3	22
34	Identification and Characterization of Tyrosine Kinase Nonreceptor 2 Mutations in Leukemia through Integration of Kinase Inhibitor Screening and Genomic Analysis. Cancer Research, 2016, 76, 127-138.	0.4	31
35	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.	0.6	0
36	Targeting BCR-ABL and JAK2 in Ph+ ALL. Blood, 2015, 125, 1362-1363.	0.6	4

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37	Kinase Regulation in Mycobacterium tuberculosis: Variations on a Theme. Structure, 2015, 23, 975-976.	1.6	4
38	Unexpected Off-Targets and Paradoxical Pathway Activation by Kinase Inhibitors. ACS Chemical Biology, 2015, 10, 234-245.	1.6	52
39	Crystal structure of an SH2â€“kinase construct of c-Abl and effect of the SH2 domain on kinase activity. Biochemical Journal, 2015, 468, 283-291.	1.7	27
40	Specificity and mechanism-of-action of the JAK2 tyrosine kinase inhibitors ruxolitinib and SAR302503 (TG101348). Leukemia, 2014, 28, 404-407.	3.3	98
41	c-Abl phosphorylates Î±-synuclein and regulates its degradation: implication for Î±-synuclein clearance and contribution to the pathogenesis of Parkinson's disease. Human Molecular Genetics, 2014, 23, 2858-2879.	1.4	176
42	The SH2 domain of Abl kinases regulates kinase autophosphorylation by controlling activation loop accessibility. Nature Communications, 2014, 5, 5470.	5.8	36
43	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
44	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.	1.7	38
45	A Novel Fusion Gene NDEL1-Pdgfrb in a Patient with JMML with a New Variant of TKI-Resistant Mutation in the Kinase Domain of PDGFRI². Blood, 2014, 124, 613-613.	0.6	5
46	The SH2 Domain of BCR-ABL1 Regulates Kinase Autophosphorylation By Controlling Activation Loop Accessibility. Blood, 2014, 124, 2209-2209.	0.6	0
47	Mechanisms of resistance to BCR-ABL and other kinase inhibitors. Biochimica Et Biophysica Acta - Proteins and Proteomics, 2013, 1834, 1449-1459.	1.1	51
48	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, 14924-14929.	3.3	85
49	Structure, Regulation, Signaling, and Targeting of Abl Kinases in Cancer. Genes and Cancer, 2012, 3, 436-446.	0.6	108
50	Cell biology: A key driver of therapeutic innovation. Journal of Cell Biology, 2012, 199, 571-575.	2.3	2
51	Allosteric BCR-ABL inhibitors in Philadelphia chromosome-positive acute lymphoblastic leukemia: novel opportunities for drug combinations to overcome resistance. Haematologica, 2012, 97, 157-159.	1.7	14
52	BCR-ABL uncouples canonical JAK2-STAT5 signaling in chronic myeloid leukemia. Nature Chemical Biology, 2012, 8, 285-293.	3.9	158
53	The Growing Arsenal of ATP-Competitive and Allosteric Inhibitors of BCRâ€“ABL. Cancer Research, 2012, 72, 4890-4895.	0.4	73
54	Mig6 Is a Sensor of EGF Receptor Inactivation that Directly Activates c-Abl to Induce Apoptosis during Epithelial Homeostasis. Developmental Cell, 2012, 23, 547-559.	3.1	47

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55	Nilotinib as frontline and second-line therapy in chronic myeloid leukemia: Open questions. Critical Reviews in Oncology/Hematology, 2012, 82, 370-377.	2.0	8
56	Targeting the SH2-Kinase Interface in Bcr-Abl Inhibits Leukemogenesis. Cell, 2011, 147, 306-319.	13.5	122
57	Targeting allosteric regulatory modules in oncoproteins: â€œDrugging the Undruggableâ€. Oncotarget, 2011, 2, 828-829.	0.8	7
58	BCR-ABL SH3-SH2 domain mutations in chronic myeloid leukemia patients on imatinib. Blood, 2010, 116, 3278-3285.	0.6	69
59	A potent and highly specific FN3 monobody inhibitor of the Abl SH2 domain. Nature Structural and Molecular Biology, 2010, 17, 519-527.	3.6	138
60	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.	3.3	67
61	Bcr-Abl Directly Activates Stat5 Independent of Jak2. Blood, 2010, 116, 511-511.	0.6	0
62	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, 7414-7419.	3.3	146
63	The structure of the leukemia drug imatinib bound to human quinone reductase 2 (NQO2). BMC Structural Biology, 2009, 9, 7.	2.3	83
64	Global target profile of the kinase inhibitor bosutinib in primary chronic myeloid leukemia cells. Leukemia, 2009, 23, 477-485.	3.3	254
65	The Bcr-Abl SH2-Kinase Domain Interface Is Critical for Leukemogenesis and An Additional Therapeutic Target in CML.. Blood, 2009, 114, 37-37.	0.6	1
66	The DEAD-box helicase DDX3X is a critical component of the TANK-binding kinase 1-dependent innate immune response. EMBO Journal, 2008, 27, 2135-2146.	3.5	276
67	Intrinsic differences between the catalytic properties of the oncogenic NUP214-ABL1 and BCR-ABL1 fusion protein kinases. Leukemia, 2008, 22, 2208-2216.	3.3	42
68	Characterization of BCR-ABL deletion mutants from patients with chronic myeloid leukemia. Leukemia, 2008, 22, 1184-1190.	3.3	38
69	The chemokine interleukinâ€8 and the surface activation protein CD69 are markers for Bcrâ€“Abl activity in chronic myeloid leukemia. Molecular Oncology, 2008, 2, 272-281.	2.1	27
70	Target spectrum of the BCR-ABL inhibitors imatinib, nilotinib and dasatinib. Leukemia and Lymphoma, 2008, 49, 615-619.	0.6	233
71	Structural Coupling of SH2-Kinase Domains Links Fes and Abl Substrate Recognition and Kinase Activation. Cell, 2008, 134, 793-803.	13.5	190
72	Structural Positioning of the SH2 Domain Is Critical for Bcr-Abl Kinase Activity, Signal Transduction and Oncogenic Transformation. Blood, 2008, 112, 569-569.	0.6	0

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73	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, 13283-13288.	3.3	274
74	Chemical proteomic profiles of the BCR-ABL inhibitors imatinib, nilotinib, and dasatinib reveal novel kinase and nonkinase targets. Blood, 2007, 110, 4055-4063.	0.6	600
75	Chronic myeloid leukemia â€“ some topical issues. Leukemia, 2007, 21, 1347-1352.	3.3	12
76	Systematic Profiling and Novel Targets of the Bcr-Abl Kinase Inhibitors Imatinib, Nilotinib and Dasatinib.. Blood, 2007, 110, 4542-4542.	0.6	1
77	Characterization of BCR-ABL Deletion Mutants from Patients with Chronic Myeloid Leukemia.. Blood, 2007, 110, 2936-2936.	0.6	0
78	Mechanisms of Activation of Abl Family Kinases. , 2006, , 1-10.		0
79	Organization of the SH3-SH2 Unit in Active and Inactive Forms of the c-Abl Tyrosine Kinase. Molecular Cell, 2006, 21, 787-798.	4.5	192
80	An efficient tandem affinity purification procedure for interaction proteomics in mammalian cells. Nature Methods, 2006, 3, 1013-1019.	9.0	366
81	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.	0.6	0
82	NMR Assignment Reveals an Î±-Helical Fold for the F-Actin Binding Domain of Human Bcr-Abl/c-Abl. Journal of Biomolecular NMR, 2005, 32, 335-335.	1.6	3
83	Structural Basis for the Cytoskeletal Association of Bcr-Abl/c-Abl. Molecular Cell, 2005, 19, 461-473.	4.5	63
84	The central domain of the matrix protein of HIV-1: influence on protein structure and virus infectivity. Biological Chemistry, 2004, 385, 303-13.	1.2	3
85	Regulation of the c-Abl and Bcrâ€“Abl tyrosine kinases. Nature Reviews Molecular Cell Biology, 2004, 5, 33-44.	16.1	429
86	A Myristoyl/Phosphotyrosine Switch Regulates c-Abl. Cell, 2003, 112, 845-857.	13.5	404
87	Structural Basis for the Autoinhibition of c-Abl Tyrosine Kinase. Cell, 2003, 112, 859-871.	13.5	762