Patrick ChÃ¨ne

List of Publications by Year
in descending order

53	2,737	346980 22	232693 48
papers	citations	h-index	g-index

59	59	59	3565
all docs	docs citations	times ranked	citing authors

#	Article	IF	Citations
1	The role of lysine palmitoylation/myristoylation in the function of the TEAD transcription factors. Scientific Reports, 2022, 12, 4984.	1.6	8
2	Long-range structural preformation in yes-associated protein precedes encounter complex formation with TEAD. IScience, 2022, 25, 104099.	1.9	5
3	Study of the TEAD â€binding domain of the YAP protein from animal species. Protein Science, 2021, 30, 339-349.	3.1	4
4	Biochemical properties of VGLL4 from Homo sapiens and Tgi from Drosophila melanogaster and possible biological implications. Protein Science, 2021, 30, 1871-1881.	3.1	4
5	Identification of FAM181A and FAM181B as new interactors with the TEAD transcription factors. Protein Science, 2020, 29, 509-520.	3.1	24
6	A new perspective on the interaction between the Vg/VGLL1-3 proteins and the TEAD transcription factors. Scientific Reports, 2020, 10, 17442.	1.6	15
7	An Early Association between the Î±-Helix of the TEAD Binding Domain of YAP and TEAD Drives the Formation of the YAP:TEAD Complex. Biochemistry, 2020, 59, 1804-1812.	1.2	16
8	Structure-based design of potent linear peptide inhibitors of the YAP-TEAD protein-protein interaction derived from the YAP omega-loop sequence. Bioorganic and Medicinal Chemistry Letters, 2019, 29, 2316-2319.	1.0	32
9	Structural States of Hdm2 and HdmX: Xâ€ray Elucidation of Adaptations and Binding Interactions for Different Chemical Compound Classes. ChemMedChem, 2019, 14, 1305-1314.	1.6	17
10	Molecular and structural characterization of a <scp>TEAD</scp> mutation at the origin of Sveinsson's chorioretinal atrophy. FEBS Journal, 2019, 286, 2381-2398.	2.2	23
11	Discovery and Structural Characterization of ATP-Site Ligands for the Wild-Type and V617F Mutant JAK2 Pseudokinase Domain. ACS Chemical Biology, 2019, 14, 587-593.	1.6	19
12	1H, 13C, 15N resonance assignment of human YAP 50â€“171 fragment. Biomolecular NMR Assignments, 2018, 12, 179-182.	0.4	8
13	Adaptation of the bound intrinsically disordered protein YAP to mutations at the YAP:TEAD interface. Protein Science, 2018, 27, 1810-1820.	3.1	25
14	Effect of the acylation of TEAD4 on its interaction with coâ€activators YAP and TAZ. Protein Science, 2017, 26, 2399-2409.	3.1	52
15	Dissection of the interaction between the intrinsically disordered YAP protein and the transcription factor TEAD. ELife, 2017, 6, .	2.8	45
16	Different Recognition of TEAD Transcription Factor by the Conserved B-strand:loop:a-helix Motif of the TEAD Binding Site of YAP and VGLL1. ChemistrySelect, 2016, 1, 2993-2997.	0.7	9
17	The Surprising Features of the TEAD4â€Vgll1 Proteinâ€“Protein Interaction. ChemBioChem, 2014, 15, 537-542.	1.3	27
18	Study of the Selectivity of Insulin-Like Growth Factor-1 Receptor (IGF1R) Inhibitors. The Open Enzyme Inhibition Journal, 2014, 3, 27-37.	2.0	4

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19	The TEAD4â€“YAP/TAZ Proteinâ€“Protein Interaction: Expected Similarities and Unexpected Differences. ChemBioChem, 2013, 14, 1218-1225.	1.3	61
20	Factors influencing the inhibition of protein kinases. Journal of Enzyme Inhibition and Medicinal Chemistry, 2012, 27, 194-200.	2.5	2
21	Can biochemistry drive drug discovery beyond simple potency measurements?. Drug Discovery Today, 2012, 17, 388-395.	3.2	5
22	The central valine concept provides an entry in a new class of non peptide inhibitors of the p53â€“MDM2 interaction. Bioorganic and Medicinal Chemistry Letters, 2012, 22, 3498-3502.	1.0	66
23	Simultaneous protein expression and modification: an efficient approach for production of unphosphorylated and biotinylated receptor tyrosine kinases by triple infection in the baculovirus expression system. Journal of Biomolecular Techniques, 2010, 21, 9-17.	0.8	7
24	Catalytic inhibition of topoisomerase II by a novel rationally designed ATP-competitive purine analogue. BMC Chemical Biology, 2009, 9, 1.	1.6	65
25	Discovery of a new class of catalytic topoisomerase II inhibitors targeting the ATP-binding site by structure based design. Part I. Bioorganic and Medicinal Chemistry Letters, 2009, 19, 4014-4017.	1.0	42
26	NS3 Helicases as Drug Targets. Current Chemical Biology, 2009, 3, 334-342.	0.2	0
27	Novel, Potent and Selective JAK2 Inhibitors.. Blood, 2009, 114, 3777-3777.	0.6	0
28	PcrA/UvrD/Rep DNA helicases in bacterial genomes. Biochemical Systematics and Ecology, 2008, 36, 101-109.	0.6	7
29	Challenges in design of biochemical assays for the identification of small molecules to target multiple conformations of protein kinases. Drug Discovery Today, 2008, 13, 522-529.	3.2	31
30	Biochemical Study of Recombinant PcrA from Staphylococcus aureus for the Development of Screening Assays. BMB Reports, 2007, 40, 7-14.	1.1	2
31	Study of the ATP-binding site of helicase IV from Escherichia coli. Biochemical and Biophysical Research Communications, 2006, 341, 828-836.	1.0	2
32	Inhibition of DNA helicases with DNA-competitive inhibitors. Bioorganic and Medicinal Chemistry Letters, 2006, 16, 923-927.	1.0	16
33	Drugs Targeting Proteinâ€“Protein Interactions. ChemMedChem, 2006, 1, 400-411.	1.6	122
34	Inhibition of the p53-hdm2 Interaction with Low Molecular Weight Compounds. Cell Cycle, 2004, 3, 458-459.	1.3	16
35	The Novel Yeast PAS Kinase Rim15 Orchestrates G0-Associated Antioxidant Defense Mechanisms. Cell Cycle, 2004, 3, 460-466.	1.3	154
36	Inhibition of the p53-MDM2 interaction: targeting a protein-protein interface. Molecular Cancer Research, 2004, 2, 20-8.	1.5	60

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37	Inhibiting the p53â€“MDM2 interaction: an important target for cancer therapy. Nature Reviews Cancer, 2003, 3, 102-109.	12.8	645
38	The ATPases: a new family for a family-based drug design approach. Expert Opinion on Therapeutic Targets, 2003, 7, 453-461.	1.5	12
39	Study of the cytotoxic effect of a peptidic inhibitor of the p53-hdm2 interaction in tumor cells. FEBS Letters, 2002, 529, 293-297.	1.3	34
40	The gain of function of the p53 mutant Asp281Gly is dependent on its ability to form tetramers. Cancer Letters, 2002, 185, 103-109.	3.2	18
41	Aib-based peptide backbone as scaffolds for helical peptide mimics. Chemical Biology and Drug Design, 2002, 60, 88-94.	1.2	70
42	ATPases as drug targets: learning from their structure. Nature Reviews Drug Discovery, 2002, 1, 665-673.	21.5	141
43	p53 as a drug target in cancer therapy. Expert Opinion on Therapeutic Patents, 2001, 11, 923-935.	2.4	16
44	Coupling of the antennapedia third helix to a potent antagonist of the p53/hdm2 proteinâ€“protein interaction. Bioorganic and Medicinal Chemistry Letters, 2001, 11, 2161-2164.	1.0	17
45	Targeting p53 in Cancer. Anti-Cancer Agents in Medicinal Chemistry, 2001, 1, 151-161.	7.0	26
46	A small synthetic peptide, which inhibits the p53-hdm2 interaction, stimulates the p53 pathway in tumour cell lines 1 1Edited by A. R. Fersht. Journal of Molecular Biology, 2000, 299, 245-253.	2.0	149
47	Discovery of Potent Antagonists of the Interaction between Human Double Minute 2 and Tumor Suppressor p53. Journal of Medicinal Chemistry, 2000, 43, 3205-3208.	2.9	250
48	Functional analyses of a uniquep53 germline mutant (y236?) associated with a familial brain tumor syndrome. , 1999, 82, 17-22.		5
49	P53 mutants without a functional tetramerisation domain are not oncogenic 1 1Edited by A. R. Fersht. Journal of Molecular Biology, 1999, 286, 1269-1274.	2.0	31
50	Cellular characterisation of p53 mutants with a single missense mutation in the Î²-strand 326â€“333 and correlation of their cellular activities with in vitro properties. Journal of Molecular Biology, 1999, 288, 891-897.	2.0	16
51	Characterization ofp53 mutants identified in human tumors with a missense mutation in the tetramerization domain. , 1998, 78, 372-376.		24
52	Molecular characterization of the hdm2-p53 interaction 1 1Edited by J. Karn. Journal of Molecular Biology, 1997, 269, 744-756.	2.0	255
53	In vitro structure-function analysis of the Î²-strand 326-333 of human p53 1 1Edited by A. Fersht. Journal of Molecular Biology, 1997, 273, 873-881.	2.0	33